Human Paleopathology

Current Syntheses and Future Options

Edited by Donald J. Ortner
and Arthur C. Aufderheide

Human Paleopathology

Current Syntheses
and
Future Options

ANNA GIFT TO S/C

Human Paleopathology

Current Syntheses
and
Future Options

Edited by
Donald J. Ortner
and
Arthur C. Aufderheide

A Symposium held at the International Congress of
Anthropological and Ethnological Sciences
Zagreb, Yugoslavia, 24—31 July 1988

Smithsonian Institution Press
Washington and London

R
134.8
H86
1991
A11T4

Copyright © 1991 by the Smithsonian Institution.
All rights are reserved.

This book was edited and designed by Nancy P. Dutro.

Library of Congress Cataloging-in-Publication Data
Human paleopathology : current syntheses and future options /
edited by Donald J. Ortner and Arthur C. Aufderheide.
p. cm.
Based on a symposium held during the International
Congress of Anthropological and Ethnological Sciences,
Zagreb, Yugoslavia, July 24–31, 1988.
Includes bibliographical references and index.
ISBN 1-56098-039-7
1. Paleopathology—Congresses. I. Ortner, Donald J.
II. Aufderheide, Arthur C. III. International Congress of
Anthropological and Ethnological Sciences (12th : 1988 :
Zagreb, Croatia)
[DNLM: 1. Paleopathology—congresses. QZ 11.5 H918
1988]
R134.8.H86 1991
616.07—dc20
DNLM/DLC
for Library of Congress 90-10348

British Library Cataloguing-in-Publication Data is available.

∞The paper used in this publication meets the minimum
requirements of the American National Standard for Permanence
of Paper for Printed Library Materials Z39.48-1984.

Printed in the United States of America.
5 4 3 2 1
94 93 92 91 90

For permission to reproduce individual illustrations appearing in
this book, please correspond directly with the owners of the
images, as stated in the picture captions. The Smithsonian
Institution Press does not retain reproduction rights for these
illustrations individually or maintain a file of addresses for photo
sources.

SMITHSONIAN
NOV 19 1
LIBRARIES

Preface

In 1985 Dr. Pavao Rudan asked one of us (DJO) to organize a symposium on paleopathology for the International Congress of Anthropological and Ethnological Sciences, planned for July 1988 in Zagreb, Yugoslavia. This invitation was intriguing for many reasons but principally because we felt that research in paleopathology had reached a plateau. In our opinion the time had arrived to evaluate research conducted thus far as a basis for suggesting what needed to be done to continue the development of research in the discipline.

In 1985 initial invitations for papers for the symposium went out to a large and diverse group of leading scholars in paleopathology. The response was remarkably supportive and definite plans were made.

A commensal scholarly/scientific relationship in paleopathology between anthropologically and medically trained researchers has existed for many decades. It clearly was in the interest of good science to have both these disciplines involved in planning the symposium. Arthur C. Aufderheide, M.D., a medically trained pathologist, became coorganizer of the symposium and coeditor of the proceedings in the early stages of planning. Donald J. Ortner, Ph.D., a physical anthropologist, provided the anthropological perspective in the organization and editorial process.

Objectives for the symposium included: (1) review of the current status of research in paleopathology, (2) an effort to explore what can and cannot presently be said about paleopathology, (3) the contributions of paleopathology to our understanding of the history and evolution of disease, (4) an effort to explore the possibility of paleoepidemiology, (5) an attempt to establish criteria that would permit comparative research in paleopathology, (6) an effort to establish the antiquity of modern diseases, (7) an exploration of what paleopathology could contribute in an ideal research context, (8) ways to achieve the above objective, and (9) the directions research in paleopathology could take in the future.

There are four major subject areas in paleopathology: (1) soft tissue research generally conducted by medically trained scientists, (2) skeletal tissue research more often conducted by anthropologists, (3) analysis of historical and archeological materials in which medical historians are the major scholars, and (4) biochemical research on trace elements and more recently on DNA and immunoglobulins generally conducted by biochemists or medically trained scientists.

The organizers attempted to have a significant representation of papers in each of these general research areas. We achieved only partial success. We did get a good group of papers on theory and methodology, another group on skeletal paleopathology, and a fairly substantial cluster of papers on soft-tissue and biochemical research. We were less successful in stimulating papers on the use of archeological and historical materials in research on paleopathology. This partially reflects the inadequate efforts by paleopathologists to create scholarly networks and linkages with the historians of disease and medicine. This clearly is a problem that needs to be corrected.

Given the international nature of the Congress we also stressed the need to have scholars from many countries. Clearly the research interests and focus vary somewhat in different nations. It is equally true that writing a scientific paper in a language other than one's native tongue may place colleagues from other than English-speaking countries in a difficult position. We felt, however, that the rewards of international scholarship outweighed the problems inherent in writing in another language.

The coeditors have rewritten portions of some of the manuscripts in an attempt to convey more effectively our understanding of what the author was attempting to communicate. The pressures of deadlines and our wish to expedite publication made author review of our revisions impossible. We take full responsibility for any failures to accurately reflect the original meaning of the author.

Not all participants were able to complete manuscripts for the proceedings. Svante Paabo told us in advance that because of other commitments he would be unable to prepare a formal manuscript. He gave a verbal presentation during the symposium and participated actively in the discussions. Sara Bisel was unable to complete the revision of her manuscript due to a very serious illness. Because of this the coeditors felt that it would be inappropriate to publish her paper.

The coeditors would like to acknowledge the assistance of Agnes I. Stix and Janet T. Beck, Department of Anthropology, National Museum of Natural History, Smithsonian Institution, who have provided editorial, administrative, and logistical support throughout the planning and editorial phases of this scholarly endeavor. Marcia Bakry, also of the Smithsonian Department of Anthropology, improved many of the illustrations. Sara Hammer, Paleobiology Laboratory, University of Minnesota-Duluth School of Medicine, also contributed significantly to the completion of the editorial process.

Contents

1 Donald J. Ortner and Arthur C. Aufderheide: Introduction

THEORY

5 Donald J. Ortner: Theoretical and methodological issues in paleopathology

12 Susan Pfeiffer: Is paleopathology a relevant predictor of contemporary health patterns?

18 Don R. Brothwell: On zoonoses and their relevance to paleopathology

23 Keith Manchester: Tuberculosis and leprosy: Evidence for interaction of disease

36 Patty Stuart-Macadam: Porotic hyperostosis: Changing interpretations

40 Ann Stirland: Diagnosis of occupationally related paleopathology: Can it be done?

METHODS

51 Noreen Tuross: Recovery of bone and serum proteins from human skeletal tissue: IgG, osteonectin, and albumin

55 Debra L. Martin: Bone histology and paleopathology: Methodological considerations

60 William W. Hauswirth, Cynthia D. Dickel, Glen H. Doran, Philip J. Laipis and David N. Dickel: 8000-year-old brain tissue from the Windover site: Anatomical, cellular, and molecular analysis

73 Antonio Ascenzi, A. Bellelli, M. Brunori, G. Citro, R. Ippoliti, E. Lendaro and R. Zito: Diagnosis of thalassemia in ancient bones: Problems and prospects in pathology

76 U.E.C. Confalonieri, L.F. Ferreira, A.J.G. Araujo, M. Chame, and B.M. Ribeiro Filho: Trends and perspectives in paleoparasitological research

79 Arthur C. Aufderheide and Mary L. Aufderheide: Taphonomy of spontaneous ("natural") mummification with applications to the mummies of Venzone, Italy

87 C.-A. Baud and Christiane Kramar: Soft tissue calcifications in paleopathology

90 Peter K. Lewin: Technological innovations and discoveries in the investigation of ancient preserved man

92 Lubos Vyhnanek and Milan Stloukal: Harris' lines in adults: An open problem

95 Oscar Urteaga-Ballon: Medical ceramic representation of nasal leishmaniasis and surgical amputation in ancient Peruvian civilization

POPULATION STUDIES

105 Douglas W. Owsley: Temporal variation in femoral cortical thickness of North American Plains Indians

111 Marc A. Kelley: Ethnohistorical accounts as a method of assessing health, disease, and population decline among Native Americans

119 Jerome C. Rose and Philip Hartnady: Interpretation of infectious skeletal lesions from a historic Afro-American cemetery

128 Takao Suzuki: Paleopathological study on infectious diseases in Japan

140 Pia Bennike: Epidemiological aspects of paleopathology in Denmark: Past, present, and future studies

145 Juan R. Munizaga: Human skeletal pathology in pre-Columbian populations of northern Chile

151 Miroslav Prokopec and Graeme L. Pretty: Observations on health, genetics, and culture from analysis of skeletal remains from Roonka, South Australia

TUBERCULOSIS

161 Jane E. Buikstra and Sloan Williams: Tuberculosis in the Americas: Current perspectives

173 Mary Lucas Powell: Endemic treponematosis and tuberculosis in the prehistoric southeastern United States: Biological costs of chronic endemic disease

181 Eugen Strouhal: Vertebral tuberculosis in ancient Egypt and Nubia

LEPROSY

197 Joseph Zias: Leprosy and tuberculosis in the Byzantine monasteries of the Judean Desert

200 Debra A. Chase: Evidence of disease in ancient Near Eastern texts: Leprosy in the Epilogue to the Code of Hammurapi?

205 Johs G. Andersen: The medieval diagnosis of leprosy

ARTHRITIS

211 James C.C. Leisen, Howard Duncan, and J.M. Riddle: Rheumatoid erosive arthropathy as seen in macerated (dry) bone specimens

216 Jan Dequeker: Paleopathology of rheumatism in paintings

TRAUMA

225 Charlotte Roberts: Trauma and treatment in the British Isles in the Historic Period: A design for multidisciplinary research

241 Robert D. Jurmain: Paleoepidemiology of trauma in a prehistoric central California population

TUMORS

251 Judyta Gladykowska-Rzeczycka: Tumors in antiquity in East and Middle Europe

257 Enrique Gerszten and Marvin J. Allison: Human soft tissue tumors in paleopathology

261 James M. Tenney: Identification and study of carcinoma in paleopathological material: Present status and future directions

DENTAL DISEASE

269 Albert A. Dahlberg: Interpretations of general problems in amelogenesis

273 Gabor Kocsis and Antonia Marcsik: Two developmental anomalies of the teeth and resulting secondary pathosis

280 Alan H. Goodman: Stress, adaptation, and enamel developmental defects

MISCELLANEOUS CONDITIONS

291 James Blackman, Marvin J. Allison, Arthur C. Aufderheide, Norman Oldroyd, and R. Ted Steinbock: Secondary hyperparathyroidism in an Andean mummy

297 Wolfgang M. Pahl and W. Undeutsch: Noma—cancer aquaticus: First indication of the skin involving disease in ancient Egypt?

305 Arthur C. Aufderheide and Donald J. Ortner: Synthesis and conclusions

309 Participants

Introduction

Donald J. Ortner and Arthur C. Aufderheide

What scientific generalizations about paleopathology can now be made? What can paleopathology tell us about biological processes in the past? To what extent does current medical knowledge relate to the interpretation of paleopathological specimens? These and many more questions are of critical importance to the status of current and future research in paleopathology. We need to give careful thought to more general and longer-range goals for paleopathology and the need to integrate that research into the broader context of biomedical theory. These considerations will be among the major factors that will determine the nature and quality of research in the future.

Clearly our descriptive methodology and classificatory system are currently major barriers to comparative research. In many published reports it is virtually impossible to evaluate the evidence presented because the descriptions are vague and imprecise. Worse still, some authors provide a medically based diagnostic opinion with insufficient data to permit independent evaluation.

The coeditors of this volume suggest that at least some aspects of paleopathological research have reached a plateau beyond which significant further progress cannot be made without major changes in the type of research we do and the methods we use to do it. Any effort, for example, to find general trends in the history and evolution of disease on the basis of existing published reports on paleopathology immediately confronts serious problems in the lack of comparability of source materials. We must develop at least general guidelines for the basic data needed and the methodology necessary to build a base of data that will permit research on some of the important questions we need to address.

In our opinion paleopathology has reached the point where we can at least begin to evaluate the potential of various avenues of research and begin to suggest methodological options for achieving these objectives Rapidly developing biomedical technology is beginning to offer some potentially powerful research tools for paleopathology. Trace element and isotopic analysis have already clarified important dietary and nutritional factors in human archeological populations. The recovery of DNA from archeological human tissues may provide important data on diseases that have a genetic basis. The recovery of human IgG from archeological bone tissue, reported in this volume by Noreen Tuross, offers the potential of identifying infectious diseases that were present in a population. This is possible even if individuals in the living population were only exposed to the disease organisms and did not have the disease itself.

The application of high-tech biochemical methods to problems in paleopathology involves many methodological hazards that are poorly understood. Postmortem diagenic change is a major barrier in such research. Little is known about the potential of false positives or negatives resulting from diagenic processes or contamination of biological tissues by natural products in the soil environment. We also need to know more about the biology of the tissues we use in this research. For example, are there age-related differences in the biochemistry and histology of normal bone tissue? These problems clearly deserve more attention than is apparent in some of the current publications. The well-known problems that have emerged with trace element research in archeological bone tissue provide a good case study regarding the hazards associated with the simplistic application of biochemical methods to such materials.

Another issue in paleopathology is that inadequate attention has been given to theory. There have been some important attempts to explore theoretical aspects of both the time and space dimensions of disease in antiquity (e.g., Cockburn 1963; McNeill 1976; Grmek 1989). However, much of this emphasis has been in the scholarly context of medical history and very little attempt has been made to interpret paleopathological data in the general context of biological and medical theory. An exception to this is the recent book by Grmek (1989). There are interesting and important theoretical questions that must be explored as we begin to integrate research in paleopathology with the body of theory and data in other disciplines.

Perhaps the most urgent need in paleopathology is a careful review of the methods we are using. Methodology helps us to respond to the question of "what is it?" when we see a pathological condition. Certainly describing and, if possible, classifying what we observe are important. However, this objective must be achieved within the framework of principles of descriptive and classificatory rigor that are well known and established in other biological and medical disciplines.

We must also move beyond description and classification and ask "what does it mean?" In the context of this question, theory, including both medical and biological, is critical. Theoretical questions in paleopathology abound and are largely unanswered. For example, is evidence of disease in archeological bone tissue indicative of the fact that the individual had poor health during life? This question must be evaluated in comparison with an individual that has no skeletal indicators of disease. Does dental hypoplasia reflect similar disease conditions and processes as those that produce lesions in bone tissue?

We also need a much stronger theoretical base on the evolution of disease and the dynamics of host/vector interaction through time. Brothwell's report on animal/human interaction in the transmission of disease in this volume offers insight and raises important questions on this issue. Clearly there is a dynamic relationship between the evolution of infectious agents and the immune response of individuals in the host population through time (e.g., Cockburn 1963; Fiennes 1978). Infectious agents tend to become less virulent and the immune response of the host-population improves with selective evolutionary processes affecting both the agent and the host. However, what are the evolutionary mechanisms when the interaction between the infectious agent and the host is indirect. This appears to be the case in some of the erosive joint diseases where an infectious triggering agent initiates a genetically defective immune response that results in disease.

Other theoretical issues include a careful understanding of what constitutes disease versus what constitutes a dysfunctional biomedical response. For example, we have known for some time that, in some environmental situations, disease may have a net benefit. Sicklemia in Africa and thalassemia in the Mediterranean region represent a biological adaptation to endemic malaria that has a net functional value despite the serious, generally fatal anemia that is associated with the homozygous expression of the disease. Stuart-Macadam suggests, in her report in this volume, that iron deficiency anemia may reflect a functional response to other infectious diseases. As we develop theory in paleopathology that integrates information from several disciplines, we need to insure that our terminology does not limit our interpretative options; that is, evidence of disease may, in fact, be indicative of adaptive biological responses to problems.

The role of population density in disease is another important theoretical question. We know very little about the relationship of population density and disease in antiquity. For example, at what point in human history did viruses become a significant source of disease? Some viral infectious diseases require substantial population sizes and densities for maintenance (Fiennes 1978:20). McGrath's (1986) computer simulations suggest that some infectious diseases (e.g., human tuberculosis) could not have been maintained in prehistoric Native American populations. This observation is in apparent opposition to paleopathological evidence that tuberculosis was in fact present well before the 15th century. Are theoretical models based on modern medical experience applicable to conditions extant in living archeological human populations?

Related to this question is the important issue of whether or not paleoepidemiological research is possible with archeological materials. Certainly one of the goals for paleopathology should be the establishment of epidemiological trends in antiquity. There clearly are theoretical and methodological limitations in doing this. We need to establish the potential as well as the limitations of paleoepidemiology.

We know very little about the impact of culture change on human health. For example, what effect did rapid and traumatic social change have on historic Native Americans? Is the apparent increase in infectious disease in these people due to exposure to new diseases for which they had minimal immunological experience and response or are social-psychological and other factors contributing as well? In trying to organize the topics and themes for the symposium, we have encouraged the authors of papers to review the status of research in their areas of special research interest. This review raises many questions about the nature and value of research conducted thus far. Asking good questions is the first step in getting good answers and making informed decisions about future research. Both reviewing the current status of research in paleopathology and exploring future options were objectives for the symposium and the published papers. A careful look at where we now are in paleopathology provides the basis for a more informed process of choosing among the options for future research. We hope that we have been at least partially successful in achieving this objective.

Literature cited

Cockburn, A. 1963. *The Evolution and Eradication of Infectious Diseases*. Baltimore: Johns Hopkins University Press.

Fiennes, R.N. 1978. *Zoonoses and the Origins and Ecology of Human Disease*. New York: Academic Press.

Grmek, M.D. 1989. *Diseases in the Ancient Greek World*. Baltimore: Johns Hopkins University Press.

McGrath, J.W. 1986. A Computer Simulation of the Spread of Tuberculosis in Prehistoric Populations of the Lower Illinois River Valley. Ph.D. dissertation, Northwestern University, Evanston, Ill.

McNeill, W.H. 1976. *Plagues and Peoples*. Garden City, N.Y.: Anchor Press/Doubleday.

Theory

Theoretical and methodological issues in paleopathology

Donald J. Ortner

The progress of paleopathology, as a specific subject of research, parallels the development of many scientific and scholarly disciplines. This process includes an overlapping sequence of phases, each of which contributes to the objective of improved understanding regarding the scientific significance of the discipline. Stages in development include: (1) definition of a well-defined subject area of scientific interest and significance, (2) creation of a methodology for conducting research on the subject, (3) accumulation of a body of descriptive data related to the subject, (4) development of a classification system for what is observed, (5) generation of hypotheses regarding the scientific/scholarly significance of observed phenomena, and (6) relating data and hypotheses to similar research and theory in cognate fields.

The progress made through each of these stages in paleopathology varies, but there is an emerging awareness of critical problems that need to be resolved before major further development can take place. The objectives of this paper are to explore the current status of research in paleopathology, highlight areas where problems and opportunities exist, and offer some suggestions on strategies for future research. I hope that most readers will understand that my emphasis on research in skeletal paleopathology reflects my own research bias and is not due to a failure to recognize the importance of other avenues of research. The principles expressed in this essay should, in fact, apply broadly to most research in paleopathology.

Interest in pathological specimens from paleontological and archeological sites has existed for more than 150 years (Ortner and Putschar 1981:5). The establishment of paleopathology as a distinct focus of scientific research goes back at least to the early part of this century with the remarkable research and publications of Ruffer, Elliot-Smith, Wood-Jones, Moodie, and others. This early research was largely descriptive and classificatory in nature, asking the question "what is it?" when confronted with a paleopathological specimen. This emphasis on description continues to predominate in publications on paleopathology today. However, another question, "what does it mean?" is being asked with increasing frequency and is forcing us to look more carefully at our descriptive methodology, classificatory system, and theoretical assumptions. This process reflects the recognition that paleopathology must, increasingly, address broader issues in the biomedical and anthropological sciences in addition to its well-established contributions to the history of human disease.

Answers to the fundamental questions of "what is it?" and "what does it mean?" remain problematic in many cases, although we are much further along with answers to the former question than the latter. Problems in responding to the question "what is it?" involve issues of description and classification. The current reference point for such responses is in medicine and primarily with its subdisciplines of pathology and radiology. In this context, two issues are relevant to this essay. First, even in a modern clinical setting, it may be difficult or impossible to arrive at an accurate diagnosis (classification) of a pathological condition affecting a living patient. Second, even when diagnosis is possible in a modern medical patient, the necessary criteria may not apply or be available for classification of similar diseases in paleopathological specimens. A response to the question "what does it mean?" requires an adequate base of data in which the question "what is it?" has been answered with reasonable scientific precision on a sufficient number of specimens. A response also requires a theoretical context in which issues of biological mechanisms, cultural influences, and strategies of human adaptation can be considered and evaluated.

Significant progress in achieving creative responses to both these questions will depend on much greater attention to the methodological problems which now limit the utility of past and current research in paleopathology. For example, we need a method for describing pathological cases that provides information which can be evaluated independently by other observers. We also need to develop a classificatory system for paleopathology which will take into consideration the type and detail of information that is available and can be evaluated in a paleopathological specimen. This will almost certainly mean that we will be including some features that are not presently part of the classificatory system associated with orthopedic radiology and pathology. This cannot, of

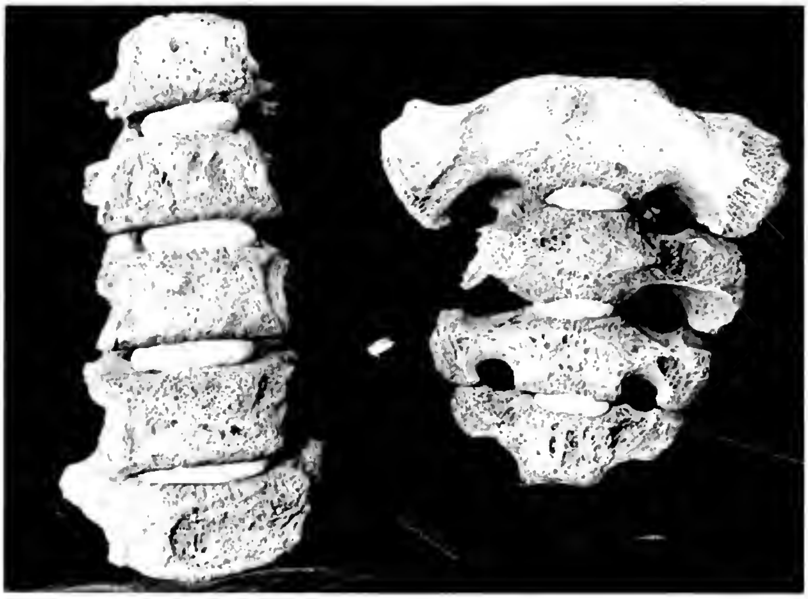


FIGURE 1. Destructive lesions of anterior, lumbar vertebral bodies and sacrum. (Child about 12 years from medieval cemetery associated with hospital of St. James and St. Mary Magdalene, Chichester, England.)

course, be done in isolation from modern medicine, but will stimulate questions for medical colleagues which address classificatory problems of paleopathology in terms of the data and observations unique to that discipline.

We also need to achieve an improved understanding of the theoretical implications of our findings in paleopathology. Does, for example, an increased prevalence of skeletal infectious disease in a skeletal sample mean that the living group of people represented by this sample was less healthy than a skeletal sample which has fewer cases of infectious disease? There are complex immunological, pathological, and demographic issues in such a situation that must be understood before reasonable conclusions can be reached. Angel's (e.g., 1966) studies of skeletal changes in thalassemia and the significance of evidence for this disease in archeological skeletal samples from the Eastern Mediterranean provide a seminal example of this kind of research.

Methodological issues in paleopathology

One of the problems in paleopathology may be an overreliance on clinical diagnostic criteria. Paleopathologists try to fit their observations into a descriptive and classificatory system that has been developed for different objectives, namely the treatment of living patients. There are many features apparent in a paleopathological skeleton that have no direct correlate in clinical medicine. For example, the observation in the medical literature that spinal tuberculosis usually predelicts the vertebral bodies (e.g., Schinz et al. 1951–1952:571; Resnick and Niwayama 1981:217; Ortner and Putschar 1981:145) may mean only that radiological evidence for involvement of the posterior elements is rarely observed.

A case of spinal lesions in the skeleton of a 12-year-old child (Burial no. S–211) from the cemetery site associated with the medieval hospital of St. James and St. Mary Magdalene in Chichester, England, illustrates the problem. Destructive lesions are apparent on the anterior surface of the vertebral bodies in the lower spine and sacrum (Figure 1). Periosteal reactive bone is seen on some of the vertebral arches (Figure 2). Other bones in the skeleton are not affected.

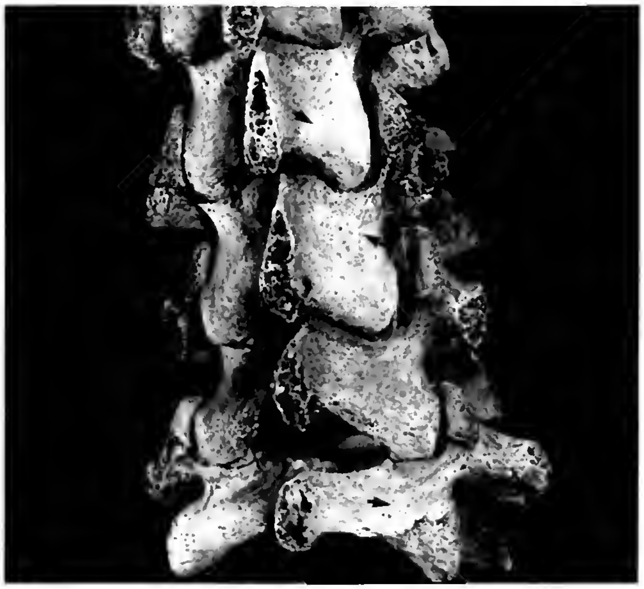


FIGURE 2. Proliferative, reactive bone on lumbar vertebral arches. (Child about 12 years from medieval cemetery associated with hospital of St. James and St. Mary Magdalene, Chichester, England.)

Both bacterial (e.g., *Mycobacterium tuberculosis* and *Staphylococcus aureus*) and mycotic (fungal) infection should be considered in differential diagnosis. Almost certainly the proliferative lesions apparent on the vertebral arches would not be seen in an x-ray film of a living patient. They thus might not be part of the clinical understanding of skeletal involvement in any of the diseases that could have produced the lesions seen in this case. In view of this possibility one needs to exercise caution in rejecting tuberculosis as a diagnostic option simply because of atypical, reactive bone formation on some of the arches. This is particularly true in view of diagnostic probabilities suggested by the high prevalence of tuberculosis in England from the latter part of the medieval period until the dramatic decline in this century.

It is possible, of course, that the lesions were caused by another bacterium such as *Staphylococcus* or by fungi, although the latter disease is rare in recent medical history. The crucial point is that the skeletal paleopathologist often sees details of bone involvement in paleopathological cases that include features not normally associated with modern clinical cases. The existence of such lesions requires careful thought and evaluation with the limitations of clinical experience being given appropriate consideration.

The high prevalence of periosteal lesions on the tibia is another example of a common observation in North American archeological skeletons that is rarely seen or noted in a clinical context. Paleopathologists have difficulty in interpreting the significance of this condition, particularly in cases where there are no additional lesions in other parts of the skeleton. Trauma and infection are likely to be the most common causes of such lesions. We are, however, unlikely to differentiate a specific cause for many, if not most, cases of this condition without further anatomical/histological studies in modern clinical cases.

Close cooperation between paleopathology and clinical medicine is an obvious and critical need in paleopathological research. Research in skeletal paleopathology is now exploring the diagnostic boundaries of clinical orthopedic disease. We are starting to ask questions for which there is no obvious clinical knowledge. Many of these questions are significant for both research in paleopathology and an improved understanding of orthopedic pathology. Collaboration between the paleopathologist and various medical specialists is likely to provide a more complete picture of skeletal responses to disease.

Dr. Bruce Ragsdale, a pathologist with a specialty in orthopedic diseases, and I are collaborating in studies of joint disease, in an attempt to answer some of the questions raised in paleopathological research (Figure 3). One important question is, what are the soft tissues associated with some of the lesions seen in joint diseases? Such research can be conducted in some situations once the problem is defined, but may be very difficult to do in conditions that rarely, if ever, are brought to the attention of the clinician.

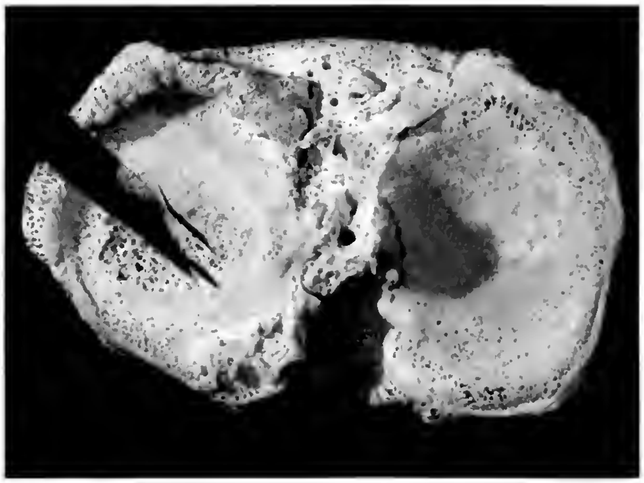


FIGURE 3. Macerated proximal tibia from a modern case of joint disease with a section through a lesion of the joint surface removed before maceration. Clinical history, antemortem and specimen x-ray films, and histological preparations of the section are being studied.

Another illustration of the potentially productive relationship that can exist between paleopathology and clinical medicine is seen in the skeletal changes associated with the various syndromes of inflammatory erosive joint disease. Ortner and Utermohle (1981) published a case of polyarticular erosive joint disease in a pre-Columbian female skeleton from Kodiak Island, Alaska. The authors suggested that the most likely syndrome for this case was rheumatoid arthritis. The patterns of most lesions, as well as the distribution of osteoporosis, were major features supporting this opinion. The problem with this diagnostic option is the extensive and severe involvement of sacroiliac and spinal joints in the disease process.

Clinically, spinal and sacroiliac joint destruction is thought to be rare in rheumatoid arthritis. The questions posed by this case include: (1) how often is the spine and sacroiliac joint involved in clinical cases of rheumatoid arthritis and (2) to what extent is the failure to observe spinal and sacroiliac joint involvement in rheumatoid arthritis an artifact of the limitations of radiology? The answer to both of these questions is important to both clinical medicine and paleopathology. Since raising these questions, colleagues in rheumatology have showed me two cases of rheumatoid arthritis that have clear evidence of erosive changes in the sacroiliac joint. I suspect that other parts of the spine are involved as well, but such changes are not well known clinically because of the limitations of radiological imaging. The significance is that a paleopathological case raised diagnostic questions which required a second and more careful look at clinical cases. This process has been a valuable experience for both the paleopathologist and the clinician.

Clinical diagnosis of orthopedic diseases relies heavily on observations derived from radiology. Several conditions limit the value of this perspective for research in skeletal paleopathology. First, radiologists rarely take an x-ray film unless a patient has a complaint of some type. Clinical orthopedic radiology is thus based on a human sample that has a medical problem to begin with. In addition, the radiologist takes only the x-ray films needed to evaluate the complaint of the patient. The implication of this is that the total pattern of skeletal involvement in orthopedic diseases may not be well known.

Furthermore, most radiologists concede that a change in bone density on the order of magnitude of forty percent is necessary before a pathological change can be seen on a clinical x-ray film. This means that many of the more subtle changes apparent on a dry-bone specimen will not be part of the experience of the radiologist and will thus not be part of the radiological descriptive and classificatory system. The overall pattern of various types of lesions in a skeleton is an observation that is accessible to the paleopathologist (when skeletal preservation is good) and is certainly of critical importance in evaluating paleopathological specimens. To utilize fully this type of observation will require thoughtful feedback from clinical experience and additional research on total patterns of skeletal disease in known cases.

Another major problem in paleopathology is the lack of comparability between reports on abnormal specimens, which precludes meaningful comparison of data. There are two major reasons for this: (1) the knowledge about skeletal disease varies greatly between observers, and (2) there is no consistent protocol for the types of data included in various reports, so that different scholars observe different conditions. If paleopathology is to address important questions regarding, for example, the evolutionary significance of disease, we must improve the comparability for both content and quality of our observations.

Recently I was coauthor of an invited manuscript on human health and disease in the Mesolithic and Neolithic ages (Ortner and Theobald 1987). The research for this manuscript included a survey of published observations and data on disease in archeological skeletons from sites dated to the Mesolithic and Neolithic ages in the Near East, Eastern Mediterranean, Europe, and the USSR. In these geographical areas, over 1200 human skeletons dated to the Mesolithic and more than 12,000 skeletons dated to the Neolithic Age have been reported in the literature.

One of the major problems in these published sources is that most authors offer an opinion on diagnosis of paleopathological specimens without carefully describing the type and location of lesions. This effectively prohibits an independent evaluation of these cases. This limits any statements regarding variation in disease prevalence in antiquity to highly speculative observations. The obvious strategy is to develop and apply a descriptive methodology to the analysis and publication of pathological specimens that does not necessarily preclude classification or diagnosis but, at the very least, permits the reader to reach his or her own conclusion regarding the nature of the pathology, without having to accept the diagnostic opinion of the author.

To achieve this objective, a much greater emphasis is needed in paleopathology on describing the abnormal conditions seen in archeological human remains. There are two dimensions of such a descriptive methodology. First, we need a widely accepted method to describe the types of abnormal conditions that exist, using criteria that reflect the underlying pathological processes. Second, we need to show, in detail, the location of all abnormal conditions within a paleopathological case. Application of a good descriptive methodology, including the type and location of lesions, to the analysis of archeological skeletal samples would be a major step in providing descriptive data that would allow independent evaluation of pathological conditions.

Description of the type of lesion should be based on the activity of the cells that produce the lesion. Three basic conditions exist: (1) formation of abnormal mineralized tissue (osteoblasts), (2) destruction of existing mineralized tissue (osteoclasts), and (3) a combination of both processes either in different parts of a lesion or different areas of the skeleton. Any descriptive system should include, at least, this information about skeletal lesions. There is, in addition, considerable variation in the amount and shape of the abnormal mineralized tissue that is formed, which adds to our understanding of the pathological process. The rapidity of bone loss in destructive lesions is indicated by the morphology of lesion margins. This is a diagnostic feature that is apparent in paleopathological specimens and can be linked with radiological diagnostic criteria. There are also abnormalities in the size and shape of bones as seen, for example, in the dysplasias and rickets.

Describing the type and location of abnormal conditions is a far less complex problem than arriving at an accurate diagnosis. Furthermore, it is the type of data that can be transferred across disciplinary lines with relative ease, and provides an important basic step in the diagnostic process. It also permits reevaluation by other scholars who can arrive at a different opinion on the diagnosis if their evaluation of the data so warrants.

Computer-aided design (CAD) software can be helpful in such research. In my laboratory we have been exploring the application of AutoCAD, a CAD software package developed by Autodesk, Inc., U.S.A., to research in skeletal paleopathology (Figure 4). This software has many powerful features. One is the ability to enlarge or reduce images. Another feature is the potential of putting different types of lesions on different layers of the CAD record for a paleopathological specimen. One or more of these layers can be turned on or off to clarify patterns and relationships between different kinds of lesions.

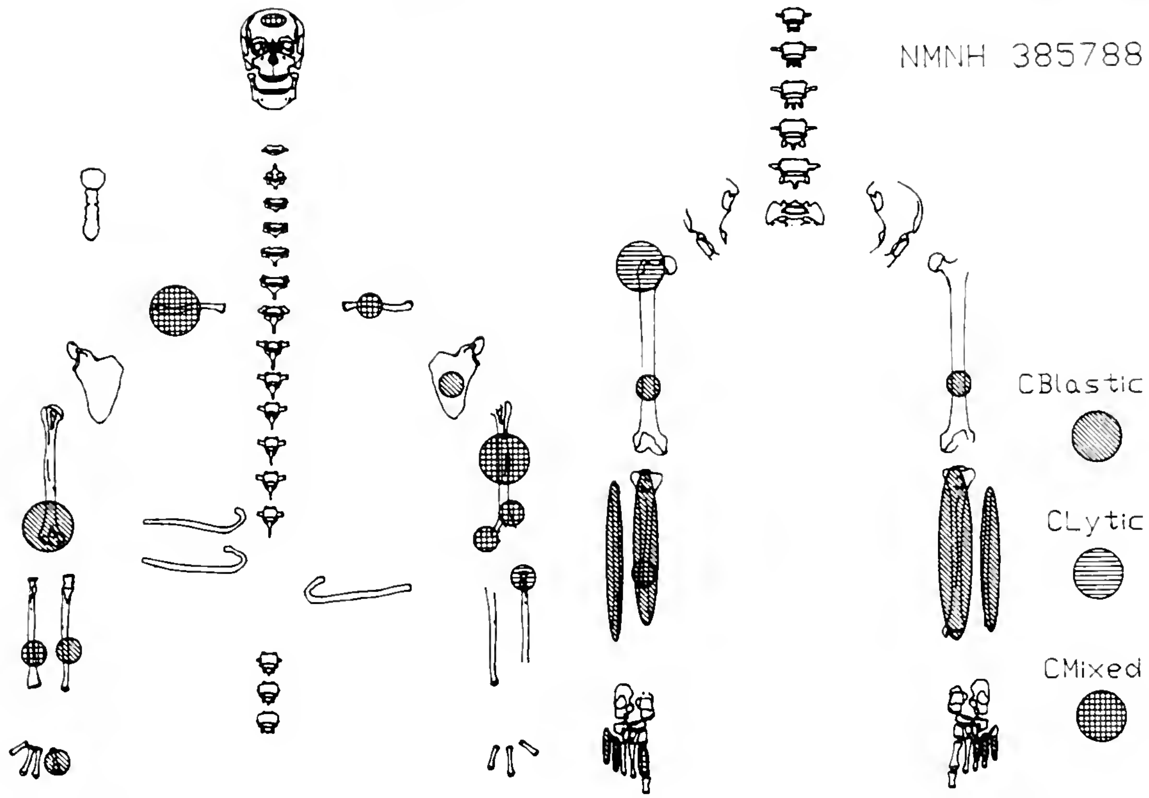


FIGURE 4. Computer-aided design drawing of the pattern of various types of lesions in a probable case of treponematosis from a pre-Columbian archeological site in Virginia, U.S.A. Note the pattern of proliferative bone lesions *(Blastic)* versus those that are largely destructive *(Lytic)* in nature. (Drawing based on an adult male skeleton from the Fisher Site, Virginia. U.S. National Museum of Natural History Catalog No. 385788.)

At the moment, however, the process of generating a graphic image showing the distribution of lesion types in a skeletal specimen is tedious and time consuming. In cases where there are several different types of lesions and a complex distribution pattern, CAD may offer important insight regarding the pathological process and is well worth the time investment. Eventually, when the system is developed further, it will be much easier to use. We also anticipate developing a data base management system to store the data which will allow statistical analysis of types of lesions.

Theoretical issues in paleopathology

Theory in paleopathology is poorly developed at present. This, in part, reflects the problems in the data base discussed above. There are, however, some tentative scholarly probes in paleopathological research that are starting to raise important theoretical issues. Research on skeletal disease in hunter-gatherer, as compared with agricultural skeletal populations, is one example (Cohen and Armelagos 1984). Clearly an improved theoretical context will become increasingly important as we continue to ask questions about the broader meaning of our data and observations.

One of the fundamental questions within the context of paleopathological evidence of infectious diseases now being asked is "what does the presence of infectious lesions of the skeleton mean, for both the health of the individual and the population from which such an individual comes?" There is, of course, a fundamental question about accuracy in diagnosis of infectious lesions. Assuming, for the moment, that such a diagnosis is possible and is accurate, we can begin to interpret the significance of such findings. Is, for example, an increased prevalence of skeletons with infectious lesions in a population an indicator of poor health for that population?

The easy assumption is that a relatively high prevalence of cases of infectious skeletal disease in a skeletal sample is, indeed, indicative of decreased population health. This may, in fact, be true, but additional evidence is likely to be needed to support this conclusion. The reason this assumption might not be true lies in the nature of the bone tissue response to infectious disease. In some cases of skeletal infection, the primary site is bone (e.g., septic arthritis). More commonly, however, involvement of the skeleton in infectious disease occurs late in the disease process (e.g., treponematosis). This means that the individual with the disease must survive the initial, often acute, phase of infection before the skeletal manifestations occur (Ortner and Hunter 1981). This implies a good immune response and a relatively healthy individual, in contrast with a person having the same disease who dies before the bone tissue is affected by the disease process.

It is at least theoretically possible that evidence of skeletal infectious disease may really be evidence of a good immune response to disease, and thus evidence of relatively good health. Certainly such an immune response is not as effective as one that successfully rids the body of the infectious organisms during the early stages of the disease. Nevertheless, it is clear that many of the infectious conditions encountered in archeological skeletons represent a long-term chronic response to the infectious agent. Resolution of the implications of this observation requires careful attention to demographic data, among other things, for the skeletal sample in question. I would want to know, for example, that individuals with evidence of skeletal disease were dying at a younger average age than individuals without such evidence. It also requires an understanding of the immunological responses to disease and their relationship to skeletal involvement. The point to be made is that, as paleopathologists begin to ask questions concerning the meaning of our observations, we must be very careful that our assumptions take into consideration all of the possible skeletal responses to disease.

Resolution of many of these questions regarding the meaning of our data and observations involves complex issues and knowledge inherent in several disciplines. Nevertheless, it is essential that paleopathology make a significant effort to move beyond the diagnostic phase of research, and ask questions about the biological and evolutionary significance of our findings. At the very least, we need to clarify the role disease has played in the complex process of adaptation between human groups and their environment.

In the last ten thousand years there have been major changes in mankind's relationship with the environment. The predominant economic subsistence pattern has shifted from hunting-gathering to agriculture. There have been major changes in settlement patterns with relatively dispersed, mobile human groups giving way to sedentary, major concentrations in small geographical areas as in the cities of today. Both of these epochal changes must have had a major impact on the prevalence of many diseases, but particularly on infectious conditions, thus representing a major challenge for research in paleopathology.

Conclusions

The flood of books and papers on paleopathology in the past twenty years is beginning to reveal some interesting scientific opportunities as well as some fundamental theoretical and methodological problems. These must be addressed if paleopathology is to make further significant contributions to both anthropological and medical theory and knowledge.

Perhaps the most serious problem is the current inability to use most of the data on paleopathology found in published sources for comparative research. There is considerable variability, both in the quality of paleopathological observations and in the types of disease conditions included in the data protocols of the source materials. A partial solution to this problem would be to develop and disseminate descriptive terminology and methodology that are less dependent on a sophisticated knowledge of pathology and radiology. The emphasis would be on careful description of abnormal conditions rather than reaching a diagnostic conclusion. Such a procedure will permit independent review of differential diagnosis in publications on paleopathology. We also need to reach a consensus on the minimal types of diseases that should be included in all paleopathological studies.

Such descriptive methodology would permit a more meaningful way of integrating data and observations from multiple research sources. It is now very difficult for one person to collect all the data needed for a regional study of paleopathology. The relatively few cases (ca. fifteen percent) affected by disease in a skeletal sample requires screening large numbers of skeletons in order to obtain meaningful data. Problems in funding, access, travel, and the sheer amount of material available for study mean that we must develop a methodology that will permit independent analysis by scholars who may not be able to study the original specimens. It is likely that there will always be problems in doing this, but the development and widespread use of a rigorous descriptive methodology with a precise terminology for skeletal lesions and their location in the skeleton would bring this goal much closer to reality. If carefully done, such data can be reinterpreted by other observers and integrated with data from other similar studies. Computer-aided design software offers an important potential research tool in recording distribution patterns of skeletal disease.

Emerging technology, particularly in the areas of chemistry and microscopy (both light and scanning electron microscopes), may offer important insight for paleopathological research. Bone is a tissue, as well as an organ, and responds to a variety of systemic conditions at the molecular, microscopic as well as at the gross level of biological organization. Immunoproteins can now be detected at the parts per billion level. If antibodies, or other proteins linked to the disease response in the body, survive in archeological skeletal tissue they can now almost certainly be detected and analyzed. Currently, I am collaborating in preliminary research on immunoproteins in archeological bone tissue that is promising. Other chemical studies of abnormal bone tissue can also assist in understanding the pathological process involved (Von Endt and Ortner 1982). Such research may open up new and important sources of data in paleopathology.

Paleopathology has made minimal effort to use microscopic data in archeological specimens, because the biological significance of many microscopic features apparent in bone is poorly understood (Richman, Ortner, and Schulter-Ellis 1979). Use of this potential source of data will require basic research on the biological significance of these features and on standards for distinguishing normal histological patterns from abnormal.

There are, of course, practical limits on what we can say about major processes, including diseases that affect human populations. Almost certainly there are new diseases today that were not present in antiquity and vice versa. It is also likely that the skeletal response in some disease processes has changed. The evolutionary tendency for infectious agents to become less virulent with time (Cockburn 1963) will increase the probability that older diseases will be more chronic in their relationship to the host. Infectious diseases that progress to a chronic phase are more likely to affect the skeleton. This means that some skeletal disease processes apparent in archeological specimens will have minimal impact on overall biological function or longevity, and may not be the primary cause of death. A response to the question regarding the broad meaning of our descriptive data will require careful thought about the implications of this and other similar facts.

Paleopathology has at least the potential to contribute to our understanding of several important processes including: (1) the biological and evolutionary role of disease in human societies, particularly as reliable data on disease prevalence accumulates, (2) the complex relationships between disease and the epochal social changes that took place in human history, such as sedentism and urbanism, and (3) the biomedical response of the skeleton to disease.

To achieve this added insight, however, we must get our methodological and theoretical house in order. We will need to avoid easy assumptions based on inadequate knowledge of pathological processes in bone tissue. As we develop a greater research emphasis on hypothesis testing and theory development in paleopathology, we must deal more effectively with the broad theoretical implications of our research. We must give more careful thought to the biological significance of evidence for infectious and other diseases in the skeleton and their significance for population morbidity. While enlightened speculation may be helpful, it is very easy to careen down scientifically blind alleys because of ignorance or because we have overextended our data. If we hope to achieve the full potential of research in paleopathology we must avoid doing this.

Literature cited

Angel, J.L. 1966. Porotic Hyperostosis, Anemias, Malarias and Marshes in the Prehistoric Eastern Mediterranean. *Science*, 153:760–763.

Cockburn, A. 1963. *The Evolution and Eradication of Infectious Diseases*. Baltimore: Johns Hopkins University Press.

Cohen, M.N., and G.J. Armelagos, eds. 1984. *Paleopathology at the Origins of Agriculture*. New York: Academic Press.

Ortner, D.J., and S. Hunter. 1981. Hematogenous Osteomyelitis in a Pre-Columbian Child's Skeleton from Maryland. *MASCA Journal*, 1:236–238.

Ortner, D.J., and W.G.J. Putschar. 1981. Identification of Pathological Conditions in Human Skeletal Remains. *Smithsonian Contributions to Anthropology*, 28. Washington, D.C.: Smithsonian Institution Press.

Ortner, D.J., and G. Theobald. 1987. Human Health and Disease in the Mesolithic and Neolithic Ages. Manuscript.

Ortner, D.J., and C.J. Utermohle. 1981. Polyarticular Inflammatory Arthritis in a Pre-Columbian Skeleton from Kodiak Island, Alaska. *American Journal of Physical Anthropology*, 56:23–31.

Resnick, D., and G. Niwayama. 1981. *Diagnosis of Bone and Joint Disorders*. Philadelphia: W.B. Saunders.

Richman, E.A., D.J. Ortner, and F.P. Schulter-Ellis. 1979. Differences in Intracortical Bone Remodeling in Three Aboriginal American Populations: Possible Dietary Factors. *Calcified Tissue International*, 28:209–214.

Schinz, H.R., W.E. Baensch, E. Friedl, and E. Uehlinger. 1951–1952. *Roentgen Diagnostics: Skeleton*. 2 volumes. English translation by J.T. Case. New York: Grune and Stratton.

Von Endt, D.W., and D.J. Ortner. 1982. Amino Acid Analysis of Bone from a Possible Case of Prehistoric Iron Deficiency Anemia. *American Journal of Physical Anthropology*, 59:377–385.

SUMMARY OF AUDIENCE DISCUSSION: Granted the need for a fundamental change in both the content and methods of paleopathological research, stimulating the necessary changes is a challenging task. Part of the problem is that paleopathologists include anthropologically and medically trained specialists and each type brings a different knowledge, experience, training, jargon, and methodology to the subject. Both the anthropological and medical disciplines are necessary for paleopathological research, and a good paleopathologist must become as knowledgable as possible about both disciplines. Equally important is that each type of specialist should cultivate collaborative relationships with specialists in the other discipline.

Basic to both, however, is a methodology that places a strong emphasis on careful description of the abnormal conditions we see. There will be problems in language but these will be minor if the basic description is done carefully. The paleopathologist also needs to develop a classificatory system that takes full advantage of the data available in the material being studied. We need ongoing dialogue between the paleopathologists and medical colleagues to insure as much overlap as possible with extant medical terminology and classificatory (diagnostic) categories.

If significant progress is to be made in paleoepidemiological research much more comprehensive skeletal samples will be needed to provide the data for both synchronic and diachronic research. One person is unlikely to be able to study all the necessary specimens, so a carefully considered and generally accepted descriptive and classificatory methodology is a critical need.

Is paleopathology a relevant predictor of contemporary health patterns?

Susan Pfeiffer

Current actions in many parts of the world toward deaccession or reburial of skeletal material have stimulated more public discussion of paleopathologists' goals and achievements. The motto of the Paleopathology Association, *Mortui viventes docent* (the dead are our teachers), suggests that we are learning things that are relevant to our contemporary context. As Kerley and Bass noted in 1967, "having some knowledge of past history, one is better able to predict the course of future events." But how specific can these predictions be? Our past research has often demonstrated the effects of ecological change on morbidity profiles (cf. Saul 1972). Our lobbying material contains references to the potential for insights into disease processes and analogies between past and current ailments (cf. Neiburger 1987; Pfeiffer 1980). However, information about past populations' mortality and morbidity is only scientifically useful if it is somehow predictive: if it can help us predict for a specific population their susceptibility to a pathogen, or the incidence of a congenital anomaly, or the effect of an environmental stressor on their growth/aging. Hence, while accepting as given the very valuable historical function performed by paleopathology, I wish to explore its ability to help us anticipate and solve contemporary health problems. I will argue that methodological limitations constrain the role that paleopathology can play. Nevertheless, it can help us predict modern disease susceptibility. Its value may sometimes lie in simple continuities or analogies. In other instances it may help us understand the commonalty of biological response which in ancient times led to one outcome, but in modern times may lead to quite another. I intend to search for such links in populations where paleopathological study has been relatively intensive, and with which I am familiar, namely the Indian and Inuit populations of Canada.

What makes the past relevant?

The dead can teach us about the living only if there is commonalty between the two. This commonalty may be genetic, behavioral, and/or environmental. Genetic continuity from past to contemporary populations is never complete, and quantitative estimates of genetic heritage are rarely available. Perhaps wisely, literature on population differences in disease pattern usually refers to "ethnicity" (a sociological term) rather than "race" (a biological term) (Cooper 1986). Association with an ethnic group may indicate only a general hint about biological heritage. For example, through Canada's Indian Act of 1876 one was defined as a status Indian only if one's male line was Indian in 1874. If an Indian woman married a non-Indian, all subsequent progeny were disenfranchised, while the progeny of an Indian man and a non-Indian were all status Indians (Price 1979). Federal legislation changed this operational definition in 1987. Such arrangements illustrate how tentative the genetic links between past racial groups and current ethnic groups may be.

Partial behavioral continuity may occasionally occur between past and present populations, in that contemporary populations may eat the same diet, build the same dwellings, or perform the same subsistence activities as their predecessors. However, such situations will be rare, such as purposeful attempts by northern Canadian natives to "go back to their roots" or "live off the land." Ostensibly, paleopathological evidence could tell such groups what health risks they might face. In practice, however, no large groups have fully adopted prehistoric native technology and divorced themselves from outside institutional support (Tungavik Federation of Nunavut 1987).

Environmental continuity of past and present populations is certainly possible. Although the mix of large plants and animals may change, as well as the influences of human population density, there may also be continuity in some of the pathogens endemic to a region. And, obviously, the arctic stays cold, the tropics stay hot. Paleopathology could in some special cases offer information about the climatic or pathogenic hazards of an environment. However, such information is likely to supplement other sources, and is very unlikely to be superior to those other sources.

Hence, there appears to be genetic/behavioral/environmental continuity between past and present populations only if these categories are sketched very broadly. In certain cases, any of these three overlapping categories may offer a strong case for prediction through homology, but a more general analogous argument will more likely be appropriate in most cases.

Nature of paleopathological evidence

The physical remains that form the basis for most paleopathological study have both attractive and unattractive features. Their attractive characteristics include their potential time depth. It is possible to examine the physical remains of a cultural lineage ranging over thousands of years, and hence ascertain the antiquity of certain maladies, as well as how they vary with behavioral or environmental shifts. Similarly, paleopathological evidence often allows observations of great geographic breadth. A particular condition can be traced throughout an environmental zone, or between one cultural lineage and its neighbors. Nonmummified remains offer a view of skeletal and dental changes that is unobscured by overlying soft tissue: there is no need to find noninvasive modes of investigation or to weigh the value of information gathered against the danger of radiating the patient. In most dry and mummified remains, evidence is of unmodified, untreated disease processes. Hence, there is no need to factor out iatrogenic effects.

On the other hand, paleopathological evidence is seriously limited. When we approach the remains clinically, the absence of soft tissue severely limits the accuracy of a differential diagnosis. There is no opportunity to establish symptomatology, no disease progress to follow. Hence our confidence in our diagnosis is often limited. Further limitations on diagnosis are imposed by the relatively nonspecific reactions of bone tissue to extrinsic stressors. Paleopathologists must be particularly cautious diagnosticians.

Perhaps the remains' most serious fault for purposes of health status prediction is their serendipitous nature. Patterns of human burial and mummification are very diverse, and retrieval of remains is relatively rare. Phillip Tobias noted in 1982 that South African hominids were represented by 511 individuals spanning over 3 million years; that is, one individual for every 5871 years (Tobias 1983). So too, the evidence of more recent human populations is sporadically distributed and not much more complete. Because of this lack of control over sampling, paleopathological evidence tells of a disease's existence but not of its prevalence nor its incidence (Moore et al. 1980). This seriously limits our ability to predict the probability of a condition's modern occurrence.

Nevertheless, progress has been made in establishing an epidemiological approach to paleopathology, in which features due to abnormal bone remodeling dynamics (or enamel formation) are observed in all elements from all skeletons of an archeological skeletal series, and patterns of incidence are tested against specific models (Buikstra and Cook 1980). Further, there are certain special ethnographic settings in which the skeletal remains recovered may approximate an unbiased sample of a Mendelian population. The practice of ossuary burial among Iroquoian people of southern Ontario is one example of such a setting (Trigger 1969,1976).

Given this list of strengths and weaknesses, I will identify a number of conditions which particularly jeopardize northern Indian and Inuit health, and attempt to see what insights or predictions the paleopathology of these groups may offer. The discussion of current native health problems is not complete, but rather emphasizes skeletal traits, or conditions which may be linked in some way to skeletal metabolism.

Health problems of Native Americans

The conditions of greatest public health interest among Native Americans are those of the so-called "New World Syndrome": obesity, adult-onset diabetes mellitus, gallstones, and gallbladder cancer (Weiss et al. 1984). Other conditions to which Native Americans appear particularly susceptible include the infectious diseases tuberculosis and otitis media, plus a number of hereditary or partially hereditary conditions: hereditary polymorphic light eruption, rheumatoid arthritis, oral clefts, hyperbilirubinemia, polydactyly, and congenital dislocation of the hip (Sievers and Fisher 1981; Criss 1985). Not all these conditions show a high relative incidence in all native groups. Furthermore, other conditions not listed here may constitute major health problems for some Native American groups. Among many groups, alcoholism and cirrhosis are common causes of morbidity and mortality. Accidents account for more fatalities among natives than among non-natives for a variety of reasons.

Note that neoplasms and coronary heart disease, which are the most common causes of mortality among middle class North Americans, are not major causes of mortality among Native Americans. This is at least partially explained by the relative youth of the native population. In Canada, only 3% of the native population is over age 65 years compared to 9% over age 65 among non-native Canadians (Statistics Canada 1984). In 1981, Indian males could expect to live an average of 9.5 years fewer than the Canadian national male population, while Indian women could expect to live an average of 10.0 years fewer than the national female population (Mao et al. 1986). In the United States, too, the median age of natives is low: 20.5 years, as compared to 28.9 years for White Americans (Criss 1985).

Relevance of paleopathology to congenital conditions

Several of the hereditary conditions listed above are manifest in the skeleton, and could be traced through paleopathological cases. Polydactyly is the most prevalent major birth defect in natives at 2.4 per 1000 live births (Niswander et al. 1975). Its antiquity has been argued from prehistoric rock art depictions (Wellmann 1972). However we cannot ascertain the incidence of the trait from such evidence. Hence, paleopathology can show us that this trait, which is a common developmental anomaly in many species, is ancient in humans; it can even suggest that ancient humans thought it noteworthy. It is unlikely to tell us anything new about polydactyly.

Congenital dislocation of the hip, which is especially common among certain Canadian native groups (6% among Cree-Ojibway; Corrigan and Segal 1950; Walker 1975), has also been documented in prehistoric remains (Clabeaux 1977). Several authors have postulated that hip dislocations, sometimes incorrectly considered a correlate of generalized joint laxity (Walker 1975), represent a genetic tendency exacerbated by cradle-boarding. Contemporary cross-cultural studies have been inconclusive (see Sievers and Fisher 1981 for discussion). A comparison of incidence between prehistoric Cree-Ojibway remains and contemporary populations would offer valuable evidence regarding the effect of cradle-board binding on hip development. However, no substantial Cree-Ojibway samples have been excavated to date. A number of small samples would need to be combined and compared carefully to gather accurate incidence data.

Rheumatoid arthritis is another example of a congenital tendency which may be exacerbated by environmental conditions. Its contemporary incidence is particularly high among Haida Indians of the Northwest Coast (Gofton et al. 1964; Lawrence et al. 1966). The absence of reported prehistoric cases has led some authors to suggest a recent origin for the condition, perhaps triggered by some environmental change (Short 1974). However, it is more likely that the absence of paleopathological cases is due to uncertainty of the diagnosis of rheumatoid versus traumatic arthritis or degenerative joint disease (DJD). Progress is now being made in differentiating the two (cf. Leisen 1986). Since relatively large isolated prehistoric skeletal samples are accessible from the geographic areas in question, it may now be possible to use paleopathological evidence (or lack of evidence) to elucidate which environmental factors are relevant to the development of rheumatoid arthritis in these native groups (cf. Cybulski 1978).

Relevance of paleopathology to infectious diseases

Otitis media is a common affliction among the children of many Indian groups, and as many as one-third of the children in some Inuit villages have chronic infection of the middle ear (Brody et al. 1965). The condition, which can cause permanent hearing impairment, appears to be tied to bottle-feeding in infancy (Schaefer 1971), possibly exacerbated by a particularly short, straight eustachian tube (Timmermans and Gerson 1980). It seems improbable that paleopathological evidence could be gathered relevant to its prehistoric incidence, which should, indeed, be nil. However, study of the normal auditory bulla of native crania in comparison to those of other racial groups might help to explain why native groups are especially susceptible to the condition when bottle-feeding is practiced. The potential relevance of paleopathology to the problem of otitis media has recently been argued by Daniel et al. (1988).

Tuberculosis has been a great source of native mortality in historic times, and it continues to be found more commonly among natives than among non-natives (Enarson and Grzybowski 1986). In 1975, native deaths from tuberculosis were 8.3 times higher than the U.S. national average (Sievers and Fisher 1981). This increased susceptibility was first thought to reflect the natives' virgin immunological status upon European contact (cf. Dubos 1965). However, the discovery of acid-fast bacilli in indisputably pre-Columbian mummified tissue (Allison et al. 1973) disproved this idea. Evidence of tuberculous-type lesions in various pre- and protohistoric native samples is accumulating (cf. Pfeiffer 1984; Hartney 1981; Perzigian and Widmer 1978; Clark et al. 1987). Paleopathological studies indicate a lack of probable tuberculosis among highly mobile, broadly dispersed (low-density) populations, but a wide range of disease incidence among more sedentary, village-living (high-density) populations.

The variability in disease incidence among the latter groups approximates that of an epidemic wave in some areas, such as southern Ontario (Pfeiffer 1984). Thus, paleopathology has demonstrated the error of the earlier "virgin immune status" assumption, and has demonstrated that tuberculosis occurred prehistorically under the same kinds of environmental stresses that cause problems today: poor hygiene, crowding, and poor nutrition. This information is very relevant to the current debate regarding whether certain ethnic groups with high rates of infection are genotypically unique, with respect to HLA haplotypes, for example (Ottenhoff et al. 1986). This would appear to be a clear case of predictive relevance for paleopathology. However, in fairness one must add that the paleopathological evidence is not well known by nonanthropological researchers.

Relevance of paleopathology to other metabolic conditions

The discussion thus far has not addressed the central group of contemporary maladies, the "New World Syndrome." Nor has it addressed certain ubiquitous skeletal conditions of prehistoric natives which seem not to pertain to contemporary health problems.

Studies of cortical bone quality of prehistoric natives have frequently demonstrated low adult bone mass (Pfeiffer and King 1983; Mazess 1966; Thompson and Guinness-Hey 1981) and relatively rapid rates of adult cortical bone loss (Ruff and Hayes 1983).

Ericksen has summarized the results of several studies of North American Indian femora, all of which indicate an adult rate of loss greater than that seen in modern black or white groups. The calculation of a bone loss rate is dependent on accurate ages at death, and there is a tendency for most skeletal estimators of age at death to underestimate the age of old adults. Hence, there is reason to question the common proposition that the amount of bone lost by modern groups from ages 20 to 90+ years was lost over only three or four decades in archeological native samples (Ericksen 1982). Nevertheless, measures of cortical thickness from such samples show lower values than either archeological black samples or modern cadaver collections. Pfeiffer and King (1983) studied femora, metacarpals, and lumbar vertebrae of prehistoric American Indians using a cross-sectional approach, and argued that mean adult values for all measures were below expected normal values.

High incidences of vertebral compression fractures have been demonstrated among prehistoric Inuit (Merbs 1983) and Iroquoians (Pfeiffer 1984). Indeed, such compression fractures appear to be relatively common among all northern native skeletal samples in which they have been studied. Note that such samples are believed to represent primarily young adults, with very few survivors over age 55.

This phenomenon of low cortical mass has been explored in modern Inuit populations. While initial study of Alaskan Eskimos suggested that these lower bone densities were tied to a high-protein, high-phosphate, low-calcium, sea mammal diet (Mazess and Mather 1974), subsequent studies of Canadian Inuit (Mazess and Mather 1975) and southern Alaskan Eskimos (Harper et al. 1984) show that the bone loss is not associated with a very specific dietary regime. Among these groups there is some variability in young adult peak bone mass, but all show an accelerated rate of bone loss with aging. Harper et al. note that, despite the potential, these Eskimo groups do not display a high incidence of osteoporotic fractures. They postulate that this might be due to short life expectancy.

Among Indians, a small cross-sectional study of southern Ontario natives demonstrated significantly lower cortical bone density in postmenopausal Indian women (N = 34) than in postmenopausal white women (N = 43) (Evers et al. 1985). However, no osteoporotic fractures had occurred among the Indian women (Evers, pers. comm.). There was no attempt to identify compressed vertebrae, which can function as an early, asymptomatic indicator of osteoporosis.

If low adult bone mass is a predictable characteristic of native populations, it may be relevant to predicting an increase in the incidence of senile osteoporosis as native life expectancy increases. Low peak adult bone mass has been observed for other Asiatic-origin populations, through both growth studies (Garn et al. 1964; Eveleth 1979) and adult radiographic screening (Nordin 1966; Yano et al. 1984). Depending on geographic location, Asiatics may show osteoporotic fracture rates that are extremely low, or higher than those of whites (Wong 1966; Chalmers and Ho 1970). Certainly diet, intensity of physical work, and the presence of confounding diseases such as tuberculosis will influence the probability of fracture in these groups. Obesity has been demonstrated to have a protective effect, helping to maintain postmenopausal bone mass (Ribot et al. 1988). Many native populations currently show high weights for height (cf. Nutrition Canada 1980), and so may not experience the expected magnitude of postmenopausal bone loss. However, future public health measures are likely to emphasize weight reduction as a generally desirable goal. Should populations achieve that goal, the paleopathological evidence of Native Americans is consistent with a prediction of a relatively high incidence of osteoporotic fractures as the contemporary population ages.

There is thus far no metabolic mechanism known to link low cortical bone mass to diabetes mellitus or any other component of the "New World Syndrome." Women with non-insulin-dependent diabetes may be at risk for bone loss, possibly because of the adverse effect of insulin deficiency on protein synthesis (Nordin 1983). Diabetics may also show high cortical bone mass (Meema and Meema 1967). Obesity, diabetes, and gallstones may all be directly associated with abnormalities in protein metabolism. Insofar as protein and calcium metabolism are interactive, consideration of a link with bone mass maintenance is not unreasonable.

Population differences in endocrinological activity have not been oriented toward explaining differences in adult peak bone mass or rates of bone loss. However, apparent population differences in steroid control are consistent with observed patterns of bone mass acquisition and loss (Purifoy 1981). Blacks, who appear to tend toward a set point favoring an increased ratio of androgens to glucocorticoids, exhibit increased peak bone density, and frequently a slower rate of bone loss. Asiatics, conversely, show decreased androgens (both groups described relative to whites) and increased glucocorticoids. Such fundamental differences could explain the observed tendency toward lower peak bone mass in Asiatics.

Conclusions

The contribution that paleopathology can make directly to the health of Native Americans is limited by the perspective of modern health sciences. When population differences in disease prevalence are associated solely with ethnic affiliation, there is a tendency for researchers to look for behavioral or environmental causation, ignoring the more difficult matter of genetic predisposition. The research necessary to identify a racial or genetic component is extremely difficult to design, owing to the difficulty of identifying Mendelian populations in the past or present and the lack of information on the genetic basis of most diseases. Nevertheless, the acknowledged need for race-specific standards of child development plus acknowledged racial differences in morphology, physiology, and biochemistry will continue to push population health studies in this direction (Watts 1981). The quantification of contemporary native groups' genetic link with pre-European native populations will allow a clearer weighing to be placed on paleopathological evidence.

This evidence has been used to some good effect to predict environmental conditions under which tuberculosis is likely to be most virulent. It could be used in a similar fashion to elucidate the etiology of other conditions such as otitis media, congenital hip dysplasia, and low peak bone mass. Paleopathological evidence is consistent with preliminary observations of low bone density in modern natives and is predictive of future osteoporosis. Finally, a retrospective study of bone metabolism through paleopathological evidence could be very helpful in understanding the metabolic pathway underlying the New World Syndrome.

Thus, paleopathology can be of some predictive utility, and can help us improve the health of contemporary Indians and Inuit. It can be more useful in the future if: (a) a racial (biological) component is acknowledged where present and quantified in ethnic disease differences, (b) paleopathological studies are designed with specific contemporary health problems in mind, (c) such studies are pursued using an epidemiological approach, and (d) paleopathologists convey their relevant knowledge directly to practitioners and health professionals.

Acknowledgment

This work benefited from comments offered by A. Herring and M. Mahaney, who read an earlier version of the manuscript.

Literature cited

Allison, M.J., J. Mendoza, and A. Pezzi. 1973. Documentation of a Case of Tuberculosis in Pre-Columbian America. *American Review of Respiratory Disease*, 107:985–991.

Brody, J.A., T. Overfield, and R. McAlister. 1965. Draining Ears and Deafness among Alaskan Eskimos. *Archives of Otolaryngology*, 81:29–33.

Buikstra, J.E., and D.C. Cook. 1980. Paleopathology: An American Account. *Annual Review of Anthropology*, 9:433–470.

Chalmers, J., and K.C. Ho. 1970. Geographical Variations in Senile Osteoporosis. *Journal of Bone and Joint Surgery*, 52B:667–675.

Clabeaux, M.S. 1977. Congenital Dislocation of the Hip in the Prehistoric Northeast. *Bulletin of the New York Academy of Medicine*, 53:339–346.

Clark, G.A., M.A. Kelley, J.M. Grange, and M.C. Hill. 1987. The Evolution of Mycobacterial Disease in Human Populations. *Current Anthropology*, 28:45–51.

Cooper, R. 1986. Race, Disease and Health. In T. Rathwell and D. Phillips, eds., *Health, Race and Ethnicity*, 21–79. London: Croon Helm.

Corrigan, C.E., and S. Segal. 1950. Incidence of Congenital Dislocation of the Hip at Island Lake, Manitoba. *Canadian Medical Association Journal*, 62:535–540.

Criss, B. 1985. Biologic Variation in Sioux Indians. In T. Overfield, ed., *Biologic Variation in Health and Illness*, 145–165. Menlo Park, Calif.: Addison-Wesley.

Cybulski, J.S. 1978. An Earlier Population of Hesquiat Harbor, British Columbia. *Cultural Recovery Paper*, 1. British Columbia: British Columbia Provincial Museum.

Daniel, H.J. III., R.T. Schmidt, R.S. Fulghum, and L. Ruckriegal. 1988. Otitis Media: A Problem for the Physical Anthropologist. *Yearbook of Physical Anthropology*, 31:143–167.

Dubos, R. 1965. *Man Adapting*. New Haven, Conn.: Yale University Press.

Enarson, D.A., and S. Grzybowski. 1986. Incidence of Active Tuberculosis in the Native Population of Canada. *Canadian Medical Association Journal*, 134:1149–1152.

Ericksen, M.F. 1982. Aging Changes in Thickness of the Proximal Femoral Cortex. *American Journal of Physical Anthropology*, 59:121–130.

Eveleth, P.B. 1979. Population Differences in Growth: Environmental and Genetic Factors. In F. Falkner and J.M. Tanner, eds., *Human Growth*, vol. 3, 373–394. New York: Plenum Press.

Evers, S.E., J.W. Orchard, and R.G. Haddad. 1985. Bone Density in Postmenopausal North American Indian and Caucasian Females. *Human Biology*, 57:719–726.

Garn, S.M., E.M. Pao, and M.E. Rihl. 1964. Compact Bone in Chinese and Japanese. *Science*, 143:1439–1440.

Gofton, J.P., H.S. Robinson, and G.E. Price. 1964. A Study of Rheumatic Disease in a Canadian Indian Population. II: Rheumatoid Arthritis in the Haida Indians. *Annals of the Rheumatic Diseases*, 23:364.

Harper, A.B., W.S. Laughlin, and R.B. Mazess. 1984. Bone Mineral Content in St. Lawrence Island Eskimos. *Human Biology*, 56:63–77.

Hartney, P.G. 1981. Tuberculous Lesions in a Prehistoric Population Sample from Southern Ontario. In J. Buikstra, ed., Prehistoric Tuberculosis in the Americas. *Northwestern University Archeological Program Scientific Papers*, 5:141–160. Evanston, Ill.: Northwestern University.

Kerley, E.R., and W.M. Bass. 1967. Paleopathology: Meeting Ground for Many Disciplines. *Science*, 157:638–644.

Lawrence, J.S., T. Behrend, P.H. Bennett, J.M. Bremner, T.A. Burch, J.P. Gofton, L.W. O'Brian, and H. Robinson. 1966. Geographical Studies on Rheumatoid Arthritis. *Annals of Rheumatic Diseases*, 25:425–431.

Leisen, J.C. 1986. Morphology of the Rheumatoid Erosion in the Dry Bone of the Metacarpal Head. *Paleopathology Newsletter*, 54:13.

Mao, Y., H. Morrison, and R. Semenceew. 1986. Mortality on Canadian Indian Reserves, 1977–82. *Canadian Journal of Public Health*, 77.

Mazess, R.B. 1966. Bone Density in Sadlermiut Eskimos. *Human Biology*, 38:42–49.

Mazess, R.B., and W. Mather. 1974. Bone Mineral Content of North Alaskan Eskimos. *American Journal of Clinical Nutrition*, 27:916–925.

———. 1975. Bone Mineral Content in Canadian Eskimos. *Human Biology*, 47:45–63.

Meema, H.E., and S. Meema. 1967. The Relationship of Diabetes Mellitus and Body Weight to Osteoporosis in Elderly Females. *Canadian Medical Association Journal*, 96:132–139.

Merbs, C.F. 1983. Patterns of Activity-Induced Pathology in a Canadian Inuit Population. *National Museum of Man Mercury Series*, 119. Ottawa: National Museums of Canada.

Moore, L.G., P.W. Van Arsdale, J.E. Glittenberg, and R.A. Aldrich. 1980. *The Biocultural Basis of Health*. St. Louis: C.V. Mosby.

Neiburger, E.K. 1987. Paleopathology and the Health Status of the American Indian. *Paleopathology Newsletter*, 59:11–14.

Niswander, J.D., M.V. Barrow, and F.J. Bingle. 1975. Congenital Malformations in the American Indian. *Social Biology*, 22:203–215.

Nordin, B.E.C. 1966. International Patterns of Osteoporosis. *Clinica Ortopedica*, 45:17–30.

______. 1983. Osteoporosis with Particular Reference to the Menopause. In L.V. Avioli, ed., *The Osteoporotic Syndrome*, pages 13–44. New York: Grune and Stratton.

Nutrition Canada. 1980. *Anthropometry Report*. Ottawa: National Health and Welfare.

Ottenhoff, T.H.M., P. Torres, J.T. de las Aguas, R. Fernandez, W. van Eden, R.R.P. de Vries, and J.L. Stanford. 1986. Evidence for an HLA-DR4Associated Immune-Response Gene for *Mycobacterium tuberculosis*. *The Lancet*, 9:310–313.

Perzigian, A.J., and L. Widmer. 1978. Evidence for Tuberculosis in a Prehistoric Population. *Journal of the American Medical Association*, 241:2643–2646.

Pfeiffer, S. 1980. Why Bother with Burials? *Newsletter, Museum of Indian Archaeology*, 2:4.

______. 1984. Paleopathology in an Iroquoian Ossuary, with Special Reference to Tuberculosis. *American Journal of Physical Anthropology*, 65:181–189.

Pfeiffer, S., and P. King. 1983. Cortical Bone Formation and Diet among Protohistoric Iroquoians. *American Journal of Physical Anthropology*, 60:23–28.

Price, J.A. 1979. *Indians of Canada*. Scarborough, Canada: Prentice-Hall.

Purifoy, F.E. 1981. Endocrine-Environment Interaction in Human Variability. *Annual Review of Anthropology*, 10:141–162.

Ribot, C., F. Tremollieres, J.-M. Bonneu, F. Germain, and J.P. Louvet. 1988. Obesity and Post-Menopausal Bone Loss: The Influence of Obesity on Vertebral Density and Bone Turnover in Postmenopausal Women. *Bone*, 8:327–331.

Ruff, C.B., and W.C. Hayes. 1983. Cross-Sectional Geometry of Pecos Pueblo Femora and Tibiae—A Biomechanical Investigation, vol. 11. Sex, Age and Side Differences. *American Journal of Physical Anthropology*, 60:383–400.

Saul, F.P. 1972. The Human Skeletal Remains of Altar de Sacrificios. *Papers of the Peabody Museum, Archaeology and Ethnology*, 63(2).

Schaefer, O. 1971. Otitis Media and Bottle-Feeding. An Epidemiological Study of Infant Feeding Habits and Incidence of Recurrent and Chronic Middle Ear Disease in Canadian Eskimos. *Canadian Journal of Public Health*, 62:478–489.

Short, C.L. 1974. The Antiquity of Rheumatoid Arthritis. *Arthritis and Rheumatism*, 17:193–205.

Sievers, M.L., and J.R. Fisher. 1981. Diseases of North American Indians. In H. Rothschild, ed., *Biocultural Aspects of Disease*, 191–252. New York: Academic Press.

Statistics Canada. 1984. *Canada's Native People*. Ottawa: Minister of Supply and Services.

Thompson, D.D., and M. Gunness-Hey. 1981. Bone Mineral-Osteon Analysis of Yupik-Inupiaq Skeletons. *American Journal of Physical Anthropology*, 55:1–7.

Timmermans, F.J., and S. Gerson. 1980. Chronic Granulomatous Otitis Media in Bottle-Fed Inuit Children. *Canadian Medical Association Journal*, 122:545–547.

Tobias, P.V. 1983. Hominid Evolution in Africa. *Canadian Journal of Anthropology*, 3:163–186.

Trigger, B.G. 1969. *The Huron, Farmers of the North*. New York: Holt, Rinehart and Winston.

______. 1976. *The Children of Aataentsic, a History of the Huron People to 1660*. Montreal: McGill-Queen's University Press.

Tungavik Federation of Nunavut. 1987. Land Claims, National Parks, Protected Areas and Renewable Resource Economy. In J.G. Nelson, R. Needham, and L. Norton, eds., *Arctic Heritage*, 285–297. Proceedings of a symposium, August 24–28, 1985, Banff, Alberta, Canada. Ottawa: Association of Canadian Universities for Northern Studies.

Walker, J.M. 1975. Generalized Joint Laxity in Igloolik Eskimos and in Island Lake Amerindians. *Human Biology*, 47:263–275.

Watts, E.S. 1981. The Biological Race Concept and Diseases of Modern Man. In H.R. Rothschild, ed., *Biocultural Aspects of New York*, 3–24. New York: Academic Press.

Wellmann, K.F. 1972. New Mexico's Mutilated Hand: Finger Mutilation and Polydactylism in North American Indian Rock Art. *Journal of the American Medical Association*, 219:1609–1610.

Weiss, K.M., R.E. Ferrell, and C.L. Hanis. 1984. A New World Syndrome of Metabolic Diseases with a Genetic and Evolutionary Basis. *Yearbook of Physical Anthropology*, 27:153–178.

Wong, P.C.N. 1966. Fracture Epidemiology in a Mixed Southeastern Asian Community (Singapore). *Clinica Ortopedica*, 45:55–61.

Yano, K., R.D. Wasnich, J.M. Vogel, and L. Heilbrun. 1984. Bone Mineral Measurements among Middle-Aged and Elderly Japanese Residents in Hawaii. *American Journal of Epidemiology*, 119:751–764.

SUMMARY OF AUDIENCE DISCUSSION: There are limits of time and funding for research in paleopathology. Given those limitations one may question whether or not it is realistic to develop an elaborate data protocol and research design as part of our research endeavors. If one cannot draw reliable conclusions from the published reports on paleopathological specimens, many of the really important questions in paleopathology will not be answered. There is a threshold of content and quality that needs to be met or the published research is likely to be of minimal value.

The establishment of general data protocols need not unduly limit the creativity of a researcher. There cannot be significant progress on many paleopathological problems unless there is a minimal base of reliable data that can be used in proposing generalizations and building theory. To do that will require generally accepted descriptive terms and a classificatory system that will allow us to at least count the number of cases in different categories. Furthermore, since lesions are critical to classification, we must study every available bone for evidence of disease. That is a lot of work but there can be no paleoepidemiology if that is not done.

On zoonoses and their relevance to paleopathology

Don R. Brothwell

During the process of preparing this paper, at least one national newspaper called attention to the concern of the British health authorities about a sheep disease which is at present affecting pregnant women. In farming areas in particular, infection of sheep with a *Chlamydia* species not only causes abortion in sheep, but has posed a health problem for many women living near farms. It is interesting that this same genus of microorganism causes specific human conditions of the eye and lymphogranuloma venereum, although closely related parasites in rodents suggest that these are again zoonoses, but with a longer history.

What is zoonosis? One might have expected international agreement on such a commonly used term, but in fact this is not so. Zoonoses can simply be taken to be "diseases and infections transmitted naturally between vertebrate animals and man" (White and Jordan 1963). Alternatively, it can have the broader definition of a "disease of animals—as opposed to disease of man" (Fiennes 1967). Halpin (1975) believes the true meaning to be "a disease or infection shared by both animals and man." The implication here is that parasites can be shunted in both directions, and indeed it is known, for instance, that humans can act as a reservoir for *Mycobacterium bovis* and reintroduce this parasite back into tuberculosis-free livestock.

Perhaps the right emphasis is achieved by regarding human groups as participants in "infection chains" (Schwabe 1964), sharing certain infectious diseases with other genera for varying periods of time. As Aristotle and his world was aware of rabies as a zoonosis and even some Third World tribal groups understand certain disease links between species (the Masai realize that anthrax can be caught from contaminated meat), the concept is likely to have had a long history. Without doubt the implications of zoonoses are relevant even to our study of the prehistory of disease. For we all too often consider human diseases in relative isolation (other than for the intrusive parasites themselves). So the zoonoses provide a focus for considering a continuum and evolution of diseases beyond the species level.

Although more than one classification of the various zoonoses has been elaborated, I do not wish to expand on the alternative classifications here. These are fully discussed in Schwabe (1964). My concern is to emphasize, in general overall terms, the importance of viewing human groups and their patterns of disease against an environmental background in which there is "movement" of parasites over time through to the hominids. This mosaic of disease has a prehistory as long as that of the vertebrates. From our point of view, diseases are linked to the hominids in the following ways:(a) by adaptively following along the course of primate-hominid evolution;(b) by direct links between hominids and other vertebrates; during the Pleistocene, there must have been profound changes associated with greater reliance on hunting;(c) the closer association of human groups with other species, especially domestic livestock, as a result of agriculture;(d) the development of high-density urban populations, with enhanced or continued association with other vertebrates;(e) the elaboration of cultural factors, from the keeping of pets and the long-distance transportation of exotic animals, to the improvement of hygiene.

Has this kind of overall speculation about zoonoses any great relevance to paleopathology? I think it has, and there seems little doubt that anyone concerned with general issues of past disease ecology, or paleoepidemiology, should not ignore this subject. While zoonoses have received attention at meetings for over a century, and the first London meeting of the International Congress of Hygiene and Demography devoted a special section to it (Shelley 1892), anthropology and archeology have been remarkably slow to appreciate the importance of disease in biological and environmental studies. Indeed, all aspects of the environment may be relevant to a reconstruction of suitable habitats for disease vectors. For example, the survival of the disease-carrying tick *Ixodes ricinus* in Europe depends on moisture and vegetational cover, and it is interesting to speculate on changes in its distribution with the spread of farming and expansion of permanent and well-managed pastures (detrimental to these ticks). Similarly, the survival of tsetse fly in Africa, and thus the transmission of trypanosomiasis, depends on the availability of woodland or savanna.

There is no doubt that there are many aspects of human disease, past and present, which will continue to benefit from a balanced consideration of zoonoses in relation to other aspects of human health. In an earlier largely agricultural society for instance, can we consider the apparent historic evidence for leprosy without also taking account of the possibility of confusion with, say, contagious pustular dermatitis (known as "Orf")? And should one evaluate Burkitt's lymphoma and other conditions of uncertain etiology without taking into account that they may have resulted from a virus zoonosis?

In terms of distances over which disease can be intruded into populations, birds are clearly a major group of relevance. Even today in Britain, their numbers exceed humans by two to one, and many are migrants. If they don't directly affect human groups, wild birds can transport numerous diseases to our domesticates (Keymer 1958), including anthrax, foot and mouth disease, and salmonellosis. The bird–mosquito–Japanese encephalitis link is perhaps one of the more complex epidemiologies in this respect (McClure 1963).

Parasite evolution and disease changes

Viewing hominid diseases against a broader background of vertebrate diseases provides more opportunity for reflecting on the adaptive evolutionary changes which must have occurred in a variety of parasite species. Lambrecht (1967), for instance, provides a convincing reconstruction of the sequence of events which could have resulted in the intrusion of trypanosomiasis from nonprimate mammals into hominids living in savanna biomes. Eventually, *Trypanosoma rhodesiense*-type parasites evolved specifically in association with the hominids. Then, in post-Pleistocene times, the early pastoralists moving south with their livestock were to be severely affected by "nagana" in their animals, other trypanosome species transferred by *Glossina* from reservoirs in wild mammal species.

A very different evolutionary scheme is needed to explain the differentiation of mycobacteria causing tuberculosis and leprosy in the vertebrates. While Grmek (1983) suggests that this parasite has a very long history of association with vertebrates, extending perhaps over 300 million years, he views the last 25,000 years as a critical period for the differentiation of varieties of tuberculosis and leprosy in mammals. But what ecological or other biological factors are responsible for this late microevolution is not easy to resolve.

While the differentiation of human pathogenic mycobacteria has received attention recently, further evidence of the possible rate of change which can occur in that group could be provided by paratuberculosis (Johne's disease). So far, only cattle and sheep appear to be infected. In northern Europe, three types have been described, including a distinctive Icelandic form (Hungerford 1959). What is interesting from an evolutionary point of view is that the Icelandic variety is only just over fifty years old, the disease carriers having been imported from Germany in 1933. The disease probably did not exist in the Icelandic sheep population before then, but within the first fifteen years 70,000 had been killed by it on the island (Halpin 1975). Has the disease changed, or are Icelandic sheep sufficiently different (?genetically-immunologically) to determine the difference, or are environmental factors influencing the manifestation of the disease?

A major problem in understanding parasite evolution and dispersal from a primary host to other species is that we don't know enough yet about potential host resistance. *Shigella*, for instance, is restricted in the number of mammal species it normally infects, yet it has been relatively successful in primates (Fiennes 1978). Moreover, the virulence and often fatal nature of human shigellosis, caused by our own evolved pathogen *Shigella dysenteriae*, suggests that it diverged relatively recently from the monkey parasite *S. flexneri*. Could this have been due to the closer association of hominids with a wide variety of other higher primate species as a result of increased hunting during later Pleistocene times?

Zoonoses in relation to hunting, farming, and urbanism

During hominid evolution, profound changes have occurred in terms of food resource exploitation and the development of urbanism. This is not the place to enter the debate on the actual antiquity of Pleistocene hunting, but food bone debris and butchery marks certainly suggest that hominids were widely hunting and becoming more closely associated with their prey (including meat processing and skin preparation) by at least half a million years ago. Compared with more herbivorous primates, this closer association with other mammals could have greatly assisted in establishing certain zoonoses in the hominids.

Psittacosis (i.e., all types of ornithosis), for instance, would have been a potential danger to all those handling birds infected with the causative microorganisms (Beaudette 1955). This disease is not restricted to parrots, and indeed pigeons are now an important reservoir of the infection. Another condition one could associate with increased hunting would be the tick-borne infection tularemia. Toxoplasmosis could have been a zoonosis of worldwide importance. While cats have probably been the most important group to carry these sporozoans, these days sheep and pigs are commonly infected. Poorly cooked meat would enable the infection to be passed on. In the indigenous South African hunter-gatherer peoples, there is serological evidence of toxoplasmosis in 6% to 27% of the groups studied (Nurse and Jenkins 1977).

Was it also in the Pleistocene that tuberculosis expanded its horizons and became established in human groups? If so, was it a mutant of *Mycobacteria bovis* as generally believed, or could it have been derived from *M. avium?* What further questions should we be asking of these species in relation to *M. tuberculosis* to answer more satisfactorily the ancestral relationships of one with the other? In terms of posing a human threat, the development of dairying in association with some early urban societies would have greatly increased the chances of spreading bovine tuberculosis, of course, but this time span of five or six thousand years is surely too short to allow for the distinctive separation of the human tuberculosis variety—or was it?

It has to be kept in mind that in the past, as more recently, tuberculosis could be passed on from farm to farm by more mobile domesticates. Dogs are certainly capable of carrying the infection. They can also be a health threat in various other ways, of course, from rabies to the nematode *Toxocara canis*. Zoonoses can occur which are known to be established only in domesticates. Louping ill, of the central nervous system, is such a condition, and the virus can be transmitted to humans.

It has been pointed out (Fiennes 1967) that, as a result of agricultural developments and urbanism, another possible change affecting zoonoses was the exploitation and adaptation of various rodent species to crops and settlements. Evidence of the infestation of habitations is provided early in Egypt, as well as the actual evidence of mice from Catal Huyuk in Turkey (Brothwell 1981). Perhaps the most infamous of the rodent-carried diseases is typhus, which even in this century has been highly destructive of human life. There seems little doubt that murine typhus was the early established primary disease and that by the increasingly close proximity of rodents to people (and the adopted parasite-transmitting role of human lice from rodent fleas), the classic human form of typhus evolved. So here we may well have a relatively new disease of only six thousand years or so.

If on epidemiological grounds, Fenner (1971) is correct in stating that the viability of measles in a community depends on about 3000 cases a year in a population of 300,000, then this disease also was dependent on the emergence of larger neolithic groups and urbanism. We need to look then for an ancestor, and Fiennes (1978) suggests that of the pseudomyxoviruses of the measles-distemper-Rinderpest triad, distemper could well have the greatest antiquity, extending back from domestic dogs to the wolf ancestor. However, measles could be relatively recent in this sequence of parasite microevolution, so should we again be viewing the human disease as only a few millennia old?

Finally, as regards the impact of mammalian domesticates on human population health, it should be noted that according to the World Health Organization (1962), milk has permitted the transmission of over thirty distinct diseases, including a number mentioned here. The advent of dairying and its eventual wide distribution must then have made a further significant contribution to the spread of evolving zoonoses.

Zoonoses and the organic remains of humans

So far, I have been discussing in general terms the importance of considering zoonoses in relation to changing human disease patterns, societies and environments in the past. This may seem to be a somewhat theoretical matter to consider at a meeting specifically concerned with paleopathology. But of course ancient pathology is a step toward the reconstruction of changing disease ecologies through time, of paleoepidemiology. Also, the whole question of accurate diagnosis of pathology rests on a good knowledge of the disease alternatives which may have occurred within a particular environment. As yet, there is a tendency to consider evidence of pathology somewhat in a vacuum, neglecting if not ignoring some disease ecology and the probability of microevolution in specific parasites (and consequent changes in disease expression).

To what extent, for instance, has histoplasmosis been considered in the differential diagnosis of bone pathology? It may be considered uncommon enough today to escape attention, but has it always been so? It can result in considerable skeletal pathology, especially the African form, caused by *Histoplasma duboisii* (Cockshott 1961; Cockshott and Lucas 1964), simulating to some extent metastatic deposits. New World morbidity evidence suggests that it can still be picked up from visiting caves, where the floor may have bat feces containing histoplasma; indeed it is sometimes called "cave disease." As well as eventual bone changes, histoplasmosis more commonly produces lesions of the lung, and these may calcify (and could be confused with tubercular calcifications). How often have the burials of cave dwellers been considered for this environmental problem? The answer is probably never, and the likelihood of the interior of the rib cages being carefully excavated for calcified masses is even more remote!

Ortner and Putschar (1981) have rightly pointed out that glanders, primarily a disease of horses, can occasionally today cause human skeletal lesions. These could be confused with treponemal disease and to some extent leprosy (in skull and long bones), although the exact nature of the dry-bone pathology is not really known. There may also be infectious arthropathies, especially at the knee, elbow and ankle, a fact which on present knowledge is likely to be missed for what it is by rheumatologists concerned with arthropathies in the past.

While glanders is kept under relative control in most countries today, and thus few human cases would be expected to occur, this may not have been the situation in the past, and indeed serious horse "plagues" are known to have occurred in antiquity. The very considerable loss of horses by Charlemagne while fighting the Huns was possibly the result of glanders. To what extent this disease has changed in its degree of impact on human groups must remain for the present a matter of speculation, but this does not mean that the disease should be ignored in differential diagnosis.

Perhaps one of the most interesting yet neglected of the zoonoses is brucellosis. Until early in the 19th century, it was not clearly differentiated from malaria and certain other infections. The closely related species of *Brucella* are mainly but not exclusively pathogens of goats, cattle, and pigs (*B. melitensis*, *B. abortus*, and *B. suis*, respectively). Although diagnosis in humans is often not easy to establish, nevertheless it can clearly build up to large numbers of infected people (between 1945 and 1949 in the U.S.A. over 26,000 cases were recorded). Commonly, infection is transmitted to humans via milk or cheese, but contaminated meat and even close proximity to livestock can significantly increase chances of infection (Dalrymple-Champneys 1960).

It could be significant, in terms of how recent human groups became commonly infected, that skeletal changes do not normally occur in other mammals with brucellosis. Human bone changes today occur in from 2% to 70% of infected groups, but could the average frequency have declined through time? The spine is particularly involved, and may show multifocal surface osteitis or cavitating abscesses or a "parrot beak spondylitis." In some of the vertebral changes as well as in other pathology, for instance in the articular bone rarefaction at the hip joint (Zammit 1961), the pathology may mimic that of tuberculosis (though vertebral collapse is not typical). The fact that joint involvement in other mammals is relatively mild and affects only the joint soft tissues could surely argue for a long adaptation time to *Brucella*, while the human pathology could suggest a more recent impact of the disease. As dairying has a prehistory extending back less that 10,000 years, we may be viewing in the as yet very limited paleopathology, evidence of a relatively short adaptive microevolution of brucellosis in human populations. There is certainly a need to keep this zoonosis in mind when considering especially vertebral arthropathies and any pathology suggestive of early-stage tuberculosis.

Conclusions

Although disease in human groups can be viewed clinically in isolation, any broader view of these diseases in adaptive and evolutionary terms demands that we extend our perspectives to include social changes, environmental factors, and even other host species. Some diseases very probably evolved within the period of hominid evolution, but others may have primate or other mammal precursors.

Sorting out these categories of disease in evolutionary terms demands that we be acquainted with zoonoses, not only those which leave their mark in ancient bones, but also those which contribute to the more theoretical aspects of studying ancient human diseases. This may perhaps seem to be teaching one's grandmother to suck eggs, but I for one confess to being all too forgetful of the degree to which human diseases are in fact zoonoses of very varying antiquity.

It would be nice to think that we might eventually be able to contribute to a *comparative* paleopathology. A disease such as tuberculosis might yield to this in a decade or two. Moreover, now that sieving and flotation techniques are producing numerous small mammal bones from some sites, there is even the possibility of eventually solving the origins of leprosy. Murine leprosy can show incidences of between 1% and 5% in wild rodents (Rankin and McDiarmid 1968) and presumably could have been brought into much closer contact with humans with the emergence of high-density urbanism. As yet, it is not conventional to look at rat and mouse bones for signs of bone inflammation which might be suggestive of infection by *Mycobacterium lepraemurium*, but this will eventually have to be done.

Finally, mention should be made of the fact that there is clearly much progress in the field of helminthology in relation to the past. Parasite eggs in particular promise to yield an increasing amount of information on certain zoonoses, some facts having particular relevance for human communities. For instance, Zimmerman (1980) records in his study of an ancient Alaskan Eskimo body that the intestinal tract contained eggs of the fish trematode *Cryptocotyle lingua*. Other genera of helminths which inhabit fish have also been described, and clearly indicated fishing and fish eating. With further studies of latrine residues and coprolite material, one hopes from earlier and earlier deposits, there is thus a chance that zoonoses will even provide extra information on diet.

Literature cited

Beaudette, F.R., ed. 1955. *Psittacosis. Diagnosis, Epidemiology and Control*. New Brunswick, N.J.: Rutgers University Press.

Brothwell, D.R. 1981. The Pleistocene and Holocene Archaeology of the House Mouse and Related Species. In R.J. Berry, ed., *Biology of House Mouse*, 1–13. London: Academic Press.

Cockshott, P. 1961. Mycetoma. In H. Middlemiss, ed., *Tropical Radiology*, 38–53. London: Heinemann.

Cockshott, W.P., and A.O. Lucas. 1964. Radiological Findings in *Histoplasma duboisii* Infections. *British Journal of Radiology*, 37:653–660.

Dalrymple-Champneys, W. 1960. *Brucella Infection and Undulant Fever in Man*. London: Oxford University Press.

Fenner, F. 1971. Infectious Disease and Social Change. *Medical Journal of Australia*, 1:1043, 1099.

Fiennes, R. 1967. *Zoonoses of Primates*. London: Weidenfield and Nicolson.

______. 1978. *Zoonoses and the Origins and Ecology of Human Disease*. London: Academic Press.

Grmek, M.D. 1983. *Les Maladies à l'Aube de la Civilisation Occidentale*. Paris: Payot.

Halpin, B. 1975. *Patterns of Animal Disease*. London: Baillière, Tindall and Cox.

Hungerford, T.G. 1959. *Diseases of Livestock*. London: Angus and Robertson. Keymer, I.F. 1958. A Survey and Review of the Causes of Mortality in British Birds and the Significance of Wild Birds as Disseminators of Disease. *Veterinary Records*, 70:713–720, 736–740.

Lambrecht, F.L. 1967. Trypanosomiasis in Prehistoric and Later Human Populations, a Tentative Reconstruction. In D. Brothwell and A.T. Sandison, eds., *Diseases in Antiquity*, 132–151. Springfield, Ill.: Charles C Thomas.

McClure, H.E. 1963. Birds and the Epidemiology of Japanese Encephalitis. *Proceedings of the 13th International Ornithological Congress*, 604–610.

Nurse, G.T., and T. Jenkins. 1977. Health and the Hunter-Gatherer. *Monographs in Human Genetics*, 8. Basel, Switzerland: Karger.

Ortner, D.J., and W.G.J. Putschar. 1981. Identification of Pathological Conditions in Human Skeletal Remains. *Smithsonian Contributions to Anthropology*, 28. Washington, D.C.: Smithsonian Institution Press.

Rankin, J.D., and A. McDiarmid. 1968. Mycobacterial Infections in Free-Living Wild Animals. In A. McDiarmid, ed., *Diseases in Free-Living Wild Animals*, 119–131. London: Academic Press.

Schwabe, C.W. 1964. *Veterinary Medicine and Human Health*. Baltimore: Williams and Wilkins.

Shelley, C.E., ed. 1892. *The Relations of the Diseases of Animals to Those of Man*. Transactions of the Seventh International Congress of Hygiene and Demography, London, 1891. London: Eyre and Spottiswoode.

White, E.G., and F.T.W. Jordan. 1963. *Veterinary Preventive Medicine*. London: Baillière, Tindall and Cox.

World Health Organization. 1962. Milk Hygiene. *World Health Organization Monograph*, 48.

Zammit, F. 1961. Brucellosis. In H. Middlemiss, ed., *Tropical Radiology*, 54–60. London: Heinemann.

Zimmerman, M.R. 1980. Aleutian and Alaskan Mummies. In A. Cockburn and E. Cockburn, eds., *Mummies, Disease and Ancient Cultures*, 118–134. Cambridge, U.K.: Cambridge University Press.

SUMMARY OF AUDIENCE DISCUSSION: Morphologic bone alterations in a bacterially infected host occur only if host resistance is sufficient to allow survival over a period long enough to allow production of the destructive and responsive skeletal changes. The routine absence of such changes in viral infections, together with the need of at least the more virulent viruses for a large, nonimmune population to maintain them, suggests that viral infections are more recent and therefore may have played a lesser role in the evolutionary history of infectious diseases. Some interesting recent reports suggest that viral agents may trigger erosive arthropathies and there may be a relationship of distemper to Paget's disease.

A thorough search of ancient North American bison bones could make a major contribution to the question of whether bovine tuberculosis in the New World preceded or followed the human form. Tuberculosis could have developed in zoonoses in very early periods, been lost, and emerged again in later zoonoses. We also need more precise information of brucellosis-generated bone changes so we may search for them in archeological samples and trace the dairy product-linked diseases.

Tuberculosis and leprosy: Evidence for interaction of disease

Keith Manchester

Leprosy and tuberculosis are chronic infective diseases of mankind, caused by bacteria of the genus *Mycobacterium*. Tuberculosis is not solely a human disease, but is also encountered in lower mammals, in birds, and in certain cold-blooded animals. In contrast, although a leprosy-like disease has been identified in chimpanzee and in "wild" armadillo, as an important and relevant epidemiological entity it is essentially a human disease. Historically, on current evidence, both infections are relative newcomers to the spectrum of human disease. Perhaps more than any other disease in human history, leprosy has generated strong social reaction, which persists overtly or as undercurrent in many parts of the world today, has stimulated legislation, and given rise to abhorrence. These emotive aspects of disease have spanned many centuries. The reactions at epidemics of plague were, undoubtedly, of greater intensity, but these were short-lived and, in themselves, were epidemic. It is likely therefore that the manifold reactions and opprobrium of leprosy were due to the chronicity of the disease, its mutilating and pitiful presentation, and to ill-founded theology. But, to what extent this last was a reactive root is not known; Christian and non-Christian medieval communities both practiced segregation and demonstrated ambivalence of attitude, harsh and beneficent, toward the leprosy sufferer. Tuberculosis was the subject of Touching for the King's Evil in the European Middle Ages, but little other public reaction was engendered. In terms of mortality, tuberculosis was the "Captain of all the men of death."

Bacteria of the genus *Mycobacterium* are responsible for more human suffering and misery than any other bacteria. Both tuberculosis and leprosy are eliminated as serious health problems in Western Europe and yet, in past centuries both were of immense significance therein. Today both are still of immense significance in many parts of the world, particularly the tropics and subtropics. In Western Europe, the increasing eradication of tuberculosis is due largely to improved socioeconomic conditions and, more particularly, to prudent public health measures. Eradication of bovine tuberculosis, mass immunological screening of juveniles, and appropriate vaccination are eliminating the disease. The demise of tuberculosis is, therefore, due to human agency. Not so with leprosy. The changing patterns in leprosy prevalence and in clinical intensity have been, and to some extent still are, totally independent of any human activity directed toward control and eradication.

This paper seeks to review the history of tuberculosis and leprosy in antiquity and to consider the historic changing patterns within the concept of modern epidemiology and immunology.

Bacteriology

The bacteria responsible for the human diseases of leprosy and tuberculosis are members of the genus *Mycobacterium*. This genus, which contains about 30 species, is characterized by the ability of the bacilli to retain staining by fuchsin and related bacteriological stains in the laboratory after exposure to weak acids. This acid-fast property is not, however, unique to the mycobacteria. Of greater genus specificity and taxonomic value is the nature of the lipids within the bacterial cell walls. It is likely that the virulence or pathogenicity of each species is influenced by the specific lipid content. It is also likely that the host immunological response to invasion by the bacteria is influenced by the lipid content.

Of all the mycobacterial species, *M. leprae* is unique in that it is not possible, with present laboratory methods, to culture the bacterium *in vitro*. As a pathogen, its natural host is mankind, although, as noted, a leprosy-like disease has been recorded in chimpanzee and armadillo. Antigenically, *M. leprae* has been shown to be related to *M. vaccae*, an environmental saprophyte (Grange 1980:30). An evolutionary significance of this finding has not, as yet, been demonstrated.

M. tuberculosis, in common with all other mycobacteria except *M. leprae*, is culturable in vitro. *M. tuberculosis* is, however, unique among the culturable mycobacteria in possessing no environmental saprophytic strains. It is an obligate pathogen. Many of the other culturable mycobacteria may be responsible for disease in man or animals, and Collins and Grange (1983:18) remark that not all mycobacteria isolated from clinical material are tubercle bacilli. Such bacteria may be regarded as opportunist pathogens.

At present, there is not total agreement on the taxonomy of the bacilli causing tuberculosis in man and in cattle. One taxonomic opinion regards the responsible mycobacteria as separate species, *M. tuberculosis*, *M. bovis*, and *M. africanum*. Alternatively, these are considered to be variants of the single species *M. tuberculosis*, and there are noted to be five recognizable variants of this species (Collins and Grange 1983:19). Differentiation, regardless of taxonomic debate, is based on in vitro growth characteristics, aerobic status, and enzymatic properties (Grange 1980:22; Collins and Grange 1983:17), but differentiation into these separate strains has no clinical value.

However, while the dialectic of taxonomy may be relevant to the evolution of tuberculosis as a human disease, the immunity induced by infection by the various bacilli is common to them all.

Immunology

The basis of host defense against infection is twofold: innate nonspecific immunity, and acquired specific immunity.

Natural or innate immunity is not directed at any specific invading organism. It is of multifactorial component and consists of biological host factors such as secretory and mechanical barriers to invasion by pathogens, bactericidal properties of body fluids, phagocytic cellular activity, and determinants such as general health and nutritional status, age, and hormonal balance (Weir 1986:42–44). It is thus dependent upon the *milieu interieur*. In socioeconomically unstable archaic communities this innate immunity must surely have played an important role in the epidemic and endemic infections of antiquity. This is mirrored today, perhaps, in the famine and war-torn peoples of the Third World, and the attendant endemic and fulminating epidemic infections.

But it is the second immune mechanism which is of particular interest in *M. leprae* and *M. tuberculosis* interrelationship. This is the acquired mechanism of adaptive immunity, characterized by memory, specificity, and the recognition of "non self" (Roitt 1980:1). The recognition of non self is axiomatic and refers to the specific antigens, in the present case *M. leprae* and *M. tuberculosis*. Memory too is implicit; exposure to the specific pathogen (antigen) induces long-term host protection to future infection and development of clinical disease. Specificity in the immune response depends principally upon the host synthesis of antibody (immunoglobulin) to a specific antigen and its release into the blood and other body fluids. This is the very basis of humoral immunity. There is no evidence that humoral immunity plays any significant role in the defense mechanisms against mycobacterial infection. It is another distinct acquired immune mechanism which is active in mycobacterial infection: cell mediated immunity (CMI). *M. leprae* and *M. tuberculosis* are intracellular facultative parasites inducing, ipso facto, pathological change and clinical disease. Upon invasion of the body, the mycobacteria are ingested by phagocytic cells (macrophages), and the bacterial intracellular faculty stimulates action by T lymphocytes. The stimulated T lymphocytes produce and release a biologically active molecule called lymphokine. Within this group of substances are factors which influence the activity and movement of macrophages. The macrophage so influenced produces a greater intracellular content within itself of lysosomal enzyme. This increased enzyme content heightens the ability of the macrophage to kill intracellular parasites contained within it.

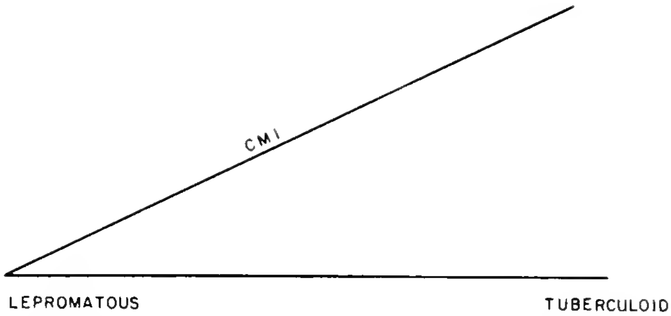


FIGURE 1. The spectrum of immunity and clinical disease in leprosy. (After Jopling 1982:296)

This, the basis of CMI, and considered very superficially here, is complex when applied to *M. leprae* infection with the broad immune, and consequent clinical spectrum. In tuberculosis, host immunity is absolute; clinical response to infection is not dependent upon and modified by a gradation of immunity, either innate or acquired. By contrast, it has been demonstrated (Ridley and Jopling 1966; Jopling 1982:296) that there is a spectrum of host immunity to *M. leprae* infections, and it is the status of the individual within this spectrum which determines the severity and type of clinical disease, and its infectivity, within the individual (Figure 1). The concept has also been applied in paleopathology (Andersen 1982:223). At one end of the spectrum is the state of absence of immunity, that is, low resistance to the pathogenic effects of invasion by *M. leprae*. The resultant clinical disease is lepromatous leprosy, a multibacillary condition characterized by high infectivity. At the other end of the spectrum is the state of high immunity, or high resistance to the pathogen. In this state the clinical disease is tuberculoid leprosy, paucibacillary and of low infectivity. There is gradation of clinical presentation and infectivity between these extremes. Indeed, beyond the high-resistant tuberculoid end of the spectrum lies the concept of subclinical infection, a state of noninfective bacterial presence within the host, but without pathological manifestation. Paleopathologically, the low-resistant, lepromatous or near lepromatous state is uniquely differentiated by its development of rhinomaxillary change (Andersen 1982:223). It is a reality, dependent upon such factors as pregnancy, intercurrent infection, and malnutrition, for an infected individual of relatively high resistance type of leprosy to downgrade toward the lepromatous pole. This concept of immune spectrum is of epidemiological importance and is of significance in the history and development of leprosy.

However, the immunity in the cell mediated response is not absolutely specific. Mackaness (1967:337) has demonstrated that the simultaneous exposure of *M. tuberculosis* and another bacillus, *Listeria monocytogenes*, to a host sensitized by previous exposure to *M. tuberculosis* induces immunity to both pathogens. The lone exposure of *L. monocytogenes* to the *M. tuberculosis*-sensitized host does not, however, induce immunity. It is the dual and coincident exposure which is significant in immunity induction. To what extent this duality of exposure and immunity applies in *M. leprae* and *M. tuberculosis* in clinical context is not, at present, known. If the same conditions apply as in *M. tuberculosis* and *Listeria* invasion, then there may be relevance to human exposure and immunity in the medieval period in Britain when both infections were present and when tuberculosis was of increasing incidence.

A further response of CMI, of diagnostic significance in tuberculosis and of pathological tissue significance in both leprosy and tuberculosis, is delayed hypersensitivity reaction. This reaction in which granulomata, lesions typical of leprosy and tuberculosis, are induced, is not considered significant in the present discussion of bacterial interrelationship.

Within the context of immunity, prophylactic immunization by Bacille Calmette Guerin (BCG) is relevant to tuberculosis and to leprosy. BCG, produced from *M. bovis*, is used in clinical circumstances to induce immunity to tuberculosis in persons demonstrated by Mantoux hypersensitivity testing to be nonimmune to the infection. The phenomenon of BCG immunization does, itself, demonstrate the phenomenon of mycobacterial cross specificity, since the vaccine is not produced from *M. tuberculosis* but is produced from *M. bovis*, a subspecies or variant. In clinical trials in areas of the world in which leprosy is endemic, BCG immunization has a proven, but variable, efficacy in the prevention of leprosy. The range of variability is from 20% efficacy in a Burmese trial to 80% efficacy in Uganda. Fine (1984:147) suggested a number of reasons for this variability. Differences between these populations exhibiting such diverse BCG efficacy may, in part, be attributable to a degree of immunity to leprosy acquired by contact with environmental mycobacteria. Administration of BCG vaccine to a people already in possession of such partial immunity may merely augment the overall population immunity to leprosy, and indeed to tuberculosis also, thereby falsely overestimating the value of BCG vaccination.

In summary, the acquired defensive mechanism in tuberculosis and leprosy is CMI. The immune reaction is not absolutely pathogen specific, but a degree of cross immunity between the pathogens is noted. Delayed hypersensitivity reaction is of significance in the development of post primary tuberculosis and probably in the granulomatous development in leprosy. Genetic immunity is of unknown, but probably little, significance in leprosy and tuberculosis. Innate immunity is a nonspecific entity and is dependent upon biological adaptive mechanisms in the host to environmental and metabolic change and to intercurrent infection.

There is no evidence to suggest that the bacteria responsible for the clinical diseases of leprosy and tuberculosis were in any way different in antiquity from those of today. Neither is there evidence to indicate that the immunological mechanism of the host to bacterial invasion has changed through time.

It is temporal change in the immunological status of populations, modified by previous bacterial exposure, which is, in part, the basis of the present hypothesis.

Epidemiology

Because, in the main, tuberculosis and leprosy are diseases of the undeveloped and developing nations, in which statistics are either absent or unreliable, the prevalence of these diseases in the world at the present time is not known. It is estimated, however, that there are 11.5 million cases of leprosy (World Health Organization 1985:10), although it is suggested (Andersen 1987) that the figure may be nearer to 20 million. The total number of persons with tuberculosis is not known, but it is estimated that 10 million persons develop tuberculosis each year and at least 3 million die of the disease (World Health Organization 1982:10).

Against this stark statistic it is necessary to consider aspects of the epidemiology of these two diseases. Much of the data are determined from current practice with only minimal input from paleopathological studies. Because, as osteoarcheological evidence indicates, there has been no change in host tissue response to infection by *M. tuberculosis* or *M. leprae*, and there has, thereby, been no change in clinical presentation through time, it is considered justifiable to extrapolate current epidemiological data to archaic populations. The inherent risks in moving from the known to the unknown are accepted.

TRANSMISSION AND DEVELOPMENT

There are, essentially, two portals of entry of the organisms causing tuberculosis in man: ingestion of bacilli or inhalation of bacilli. Reference to the taxonomic debate on *M. bovis* and *M. tuberculosis* has already been made, and the irrelevance of this in immunology is noted. However, in terms of transmission, and in the context of a hypothesis of historic development of disease, the two bacilli, be they strains or species, are of significance. Transmission of *M. bovis* is via the gastrointestinal tract from the ingestion of cattle meat or milk infected with *M. bovis*. The primary tuberculous lesion is therefore in the gut. Transmission of *M. tuberculosis* is via the respiratory tract from inhalation of droplets infected by *M. tuberculosis* and exhaled by a person with open, infectious, pulmonary tuberculosis. The primary lesion is therefore in the respiratory tract, either tonsillar bed or, more commonly, lung. Because of this mode of transmission, pulmonary tuberculosis may be considered to be a population density-dependent disease *(v.i.)*.

The incubation period, that is the latent interval between implantation of bacillus and the development of clinical disease, in tuberculosis is long. Dependent upon the course of development of tuberculous infection, the incubation period may vary from two years to several decades. In terms of immunity, these figures are largely meaningless and depend upon interpretation of clinical disease. The first manifestation of infection, termed primary tuberculosis, consists of an infected lesion at the site of entry of bacilli, and associated infective change in regional lymph nodes. This state, which may be without clinical symptoms, is associated with the development of immunity to further infection. Thereafter, and dependent upon such other factors as general health status, there may be complete resolution of the primary complex but with maintenance of immunity. Survival of the individual and restoration of total health ensues. The end result is a healthy person immune to tuberculosis and, in some measure, to other mycobacterial diseases. Alternatively, the primary infection may progress as a disseminated, fulminating, and fatal disease with distant organ involvement or miliary lesions. Whatever the mortal presentation, these individuals are removed from the scene of population immunity and are, therefore, irrelevant to the present discussion. Those individuals surviving the primary lesion and restored to health may, at some future date, subject to a deterioration of general health status or to the development of intercurrent infection, develop progressive tuberculous disease. This post-primary or secondary infection may be the result of reactivation of quiescent primary lesions, or the result of reinfection by the pathogenic organism. The difficult differentiation in the etiology of post-primary tuberculosis between reactivation or reinfection is of significance epidemiologically and may be of significance in the history of the disease. The endogenous or exogenous source of bacilli is of no significance in the clinical course of the disease or in immunological status. Post-primary infection is the familiar disease of adulthood, characterized pathologically by progressive granulomatous and caseating lesions, and clinically by progressive emaciation, cough, dyspnea, and hemoptysis in pulmonary disease and abdominal pain, distension, and pyrexia in gastrointestinal disease. Subsequently, other organ involvement may ensue, and bone and joint infection are of major importance in osteoarcheology.

To repeat and stress however, it is the primary infection in tuberculosis, with tuberculin conversion indicating immunity, which is of significance in the immune profile of the surviving individual and, through his place therein, of the population as a whole.

After many years of uncertainty, it has now been demonstrated that the clinically important mode of transmission of *M. leprae* is by inhalation of infected droplets from a leprous individual harboring bacilli in his nasal mucosa (Jopling 1982:295). Transmission of leprosy is, therefore, largely from individuals with multibacillary disease, that is, those from the low-resistant end of the immune spectrum. The criteria for such infectivity is the presence of a "highly bacilliferous nasal discharge" (Pedley and Geater 1976:97). The infectivity of paucibacillary, tuberculoid leprosy is slight. In contrast to earlier thought, it is now considered that leprosy, from lepromatous cases, is a highly infective disease, but that, because of the immune status, only a small proportion of people infected with the bacillus actually develop clinical disease.

The incubation period of leprosy is, like tuberculosis, of uncertain and long duration, probably between two and seven years (Jopling 1982:296).

Unlike tuberculosis, the pathogenesis of leprosy is not biphasic. There is no primary and secondary complex in leprosy. As previously noted, *M. leprae* is an intracellular pathogen, and has an affinity for Schwann cells of peripheral nerves and for cells of the reticuloendothelial system. After infection, tissue change and consequent clinical disease is progressive, modified only by the immune status, and change therein, of the host. Although, as yet, the stage of development of acquired immunity in leprosy is incompletely understood, it has been demonstrated by immunological testing in Micronesia and Sri Lanka that an immunity has developed from three months to two years before the onset of clinical disease (World Health Organization 1985:23).

Leprosy, unlike tuberculosis, is generally not a fatal disease. It is a relentlessly progressive and mutilating disease, but further discussion of the clinical symptoms and signs of the disease is beyond the scope of this paper.

AGE

In tuberculosis, the development of CMI is coincident with the pathological changes of primary infection. Tuberculin sensitivity is a codevelopment of CMI, albeit associated with antibody production, itself of little significance in the tuberculous defensive mechanism. Tuberculin sensitivity conversion can however be taken as a guide to the acquisition of immunity and, as a corollary, to the period of primary infection. Unfortunately, the age of conversion is incompletely known for modern developed nations and is almost completely unknown for undeveloped nations. It follows, by nature of the evidence, that the age of conversion can never be known for archaic peoples. Neither, of course, can it be known for more recent peoples before the advent of tuberculin sensitivity testing. Some other guide to primary infection, appropriate, by extrapolation, to archaic peoples must be used.

If the death rate from tuberculosis, by year of death, is examined from the prechemotherapeutic era, it is noted (Fine 1984:141) that the highest mortality occurs before the age of five years, falling dramatically thereafter and rising again markedly in the third decade (Figure 2). This may be interpreted as a high infant mortality associated with primary infection and a further high and sustained mortality with post-primary infection. If these late 19th century data can be assumed representative of medieval populations, then initial exposure to *M. tuberculosis* and the development of primary infection and consequent immunity occurred during infancy.

Because the ELISA test for leprosy infection is a relatively recent introduction which has not, as yet, had a wide application in epidemiology, age patterns in leprosy can be determined only in relation to clinical disease. The age of infection and, in consequence, the age of development of immunity, are unknown. However, the age of infection is, *post hoc propter hoc*, dependent upon the age of contact of an individual with an infective case. It is also dependent upon the intimacy of contact *(v.i.)*. In infected families young children are likely to have more intimate contact than older individuals, and therefore the young are more likely to become infected (Badger 1964:84). Clinical disease is rarely encountered below the age of five years. The incidence then rises to a plateau in the fourth decade. With acceptance of the long and variable incubation period, it seems likely that the age of infection in leprosy may be somewhat later than that for tuberculosis. Thus the age of development of acquired immunity may, likewise, be somewhat later.

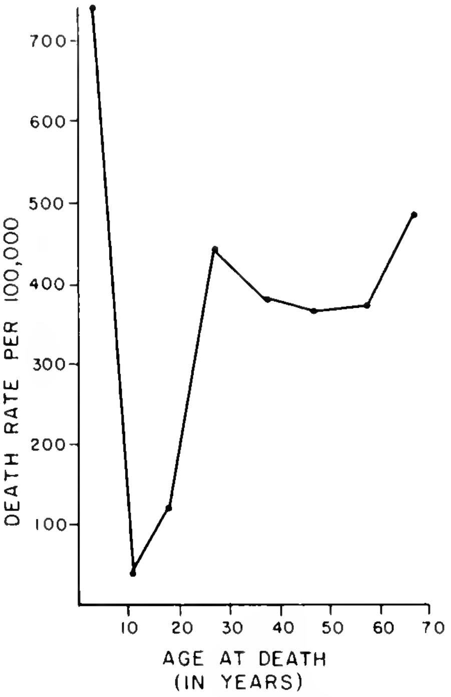


FIGURE 2. Tuberculosis death rates by year of death, late 19th century, Massachusetts. (After Fine 1984:141)

SEX

Although variations according to age and sex in incidence rates of both tuberculosis and leprosy as clinical diseases are known, it is considered that the sex variation has little bearing on the proposed immunological interrelationship. Further analysis is, therefore, not relevant within this paper.

POPULATION DENSITY

Leprosy has been described as a "disease of the villages" (Hunter 1986:5). But against this generalization, there is considerable evidence that household contacts of patients with lepromatous disease are at a high risk of infection. Epidemiological studies in Burma, South India, and the Philippines has indicated a familial clustering of infection (World Health Organization 1985:23–24). It is noted (Badger 1964:72) that the more intimate the contact the greater the risk of infection, and the risk of infection is greater with intrafamilial than with extrafamilial contact. In keeping with this, the rate of infection is also influenced by the number of contacts to whom an individual is exposed. Such interpretation of familial susceptibility is also complicated by the influence of socioeconomic status, hygiene, nutrition, and a "genetically determined susceptibility" of blood relatives. Studies suggest that there is an association between crowding and leprosy, but the problems of poverty and mobility of peoples further complicate the picture. The inhalation mode of transmission of *M. leprae* suggests that crowding of peoples and intimacy of contact are important epidemiological factors. Therefore, it is expected that leprosy is a disease of village and urban communities alike.

Buikstra and Cook (1981:118) described tuberculosis as a population density-dependent disease, and Cockburn (1963:88) also considered the disease to be a crowd disease of urban society. However, with regard to the different modes of transmission of *M. bovis* and *M. tuberculosis* and, in consequence, to the different primary manifestations and immunity therefrom, the relation, in immunological perspective, of infection to population density may be twofold. Primary gastrointestinal disease caused by *M. bovis* is dependent upon human contact with an animal reservoir of tuberculous beast. Herd size, not human population size, is the critical factor for endemicity of human primary gastrointestinal tuberculosis (Manchester 1986). Thus, this disease is likely to affect urban and rural peoples alike, assuming the general availability of milk and flesh. The growth of markets in medieval towns and cities in Britain is likely to favor urban exposure. Pulmonary tuberculosis is, in contrast, entirely dependent upon contact with a fellow human with open infectious disease. Pulmonary tuberculosis is, therefore, a crowd disease of population density dependence. It is, of course, possible, particularly within the confines of the communal animal-human longhouse, for pulmonary infection via inhalation of *M. bovis* to develop from intimate contact with an "open" cattle infection. It is further acknowledged that post-primary infection, irrespective of primary focus, may be achieved by ingestion or inhalation. But, as already stated, it is the primary infection, and the immunity developed thereby, which is significant to the arguments of this paper. It is proposed therefore that, in antiquity, there was a baseline of *M. bovis* primary infection which was sporadic and totally independent of human population size. In addition, as primary disease, and superimposed as post-primary disease, there was *M. tuberculosis* infection. This was dependent upon population density, and followed and was a consequence of urbanization and aggregation of peoples in trade. It is unfortunate but, notwithstanding the pulmonary tuberculous interpretations of rib lesions by Kelley and Micozzi (1984), paleopathological differentiation of pulmonary and gastrointestinal disease cannot, as yet, be made. The above comments on the archaic implications of *M. bovis* and *M. tuberculosis* have not, therefore, been investigated or proven.

SOCIOECONOMY

Both tuberculosis and leprosy are diseases associated, in general terms, with poverty, poor nutrition, and poor general health status. Badger (1964:73) remarks that, in leprosy, "the greatest prevalence has been, and remains, among peoples of low economic status, with inadequate housing etc., which leads to crowding and intimate contact." In the paleopathology of tuberculosis, a demonstration of these factors has yet to be made. In the paleopathology of leprosy, Møller-Christensen (1978:117) reported that 69.7% of leprous skeletons from Næstved exhibited cribra orbitalia, while only 20.2% of contemporaneous nonleprous skeletons from Æbelholt exhibited the lesion. The work of Stuart-Macadam (1982) has indicated that cribra orbitalia is the manifestation of anemia in infancy. The inference therefore from Møller-Christensen's findings is that the leprous inmates of the Næstved leprosarium were from an anemic, nutritionally deprived section of medieval society. The possibility of chronic intestinal parasitic infestation in infancy contributing to this anemia may also suggest a poor general health status and level of poverty. Further work on cribra orbitalia, porotic hyperostosis, and latrine deposits of medieval lazar houses is needed.

History

Although, within the remit of this paper, it is necessary to consider only those regions in which leprosy and tuberculosis were coexistent in antiquity, it is appropriate to review the earliest history and development of these two infectious diseases of mankind. The development and prevalence of endemic infectious disease within a species may have relevance to a developing innate immunity and to acquired immunity within a population.

Both leprosy and tuberculosis are relative newcomers to the spectrum of human disease, and tuberculosis, in historic terms, is the older of the two.

TUBERCULOSIS

The earliest evidence of tuberculosis as a human disease is from the fourth millennium B.C., and consists of osteological and iconographic specimens. A Neolithic skeleton of this period from Italy exhibits spinal osteolytic lesions compatible with a diagnosis of osseous tuberculosis (Formicola et al. 1987). A figurine exhibiting angular kyphosis and features suggestive of the cachexia of advanced consumption has been described from fourth millennium Egypt (Morse et al. 1964). Other pre-Dynastic figurines with angular kyphosis are known, but documentary records from the early Near East do not contain descriptions suggestive of tuberculosis. The Semitic Code Laws of Hammurabi of Babylon, the Ebers Papyrus (Mercer 1964), and the medical Papyri (Cave 1939) do not record disease compatible with tuberculosis. Further eastern Mediterranean examples of skeletal tuberculosis are known (Ortner 1979), and from the first millennium B.C. tuberculosis has been identified in mummified remains from Egypt.

Further east, documentary records suggestive of tuberculosis are found in India of second millennium B.C. date, and Mesopotamia of first millennium B.C. date. Suzuki (1985) has described skeletal lesions from protohistoric Japan.

Although not within the discussion of this paper, it is of interest and probable historic significance that the earliest world evidence of animal domestication is from the eastern Mediterranean, some three millennia before the earliest world evidence of tuberculosis.

Away from the Mediterranean littoral, the earliest European evidence of tuberculosis is a skeleton exhibiting spinal lesions of Potts' disease, and dated to third/second millennium B.C. from Denmark (Sager et al. 1972). A Neolithic skeleton from Heidelberg has also been diagnosed as tuberculous on recognition of possible spinal lesions (Bartels 1907), but doubt has recently been cast on this diagnosis.

However, for the purposes of this paper examining a possible relationship between leprosy and tuberculosis, it is appropriate and necessary to consider in detail only one geographic area where the two diseases coexisted and in which their history is adequately known. For these reasons and for the convenience of the author, detailed examination of the later history will be confined to Britain. It is acknowledged, of course, that this somewhat blinkard microcosmic view is one of convenience and that the evidence, constraints, and hypotheses could equally be drawn from other regions of the world in which the two diseases coexisted in antiquity.

Probably the earliest evidence of tuberculosis in Britain is of Roman date from Cirencester (Wells 1982:181; Manchester and Roberts 1987). During the succeeding centuries, the prevalence of tuberculosis increased, upon the evidence of skeletal specimens (Manchester and Roberts 1987). Unfortunately it is impossible from skeletal evidence alone to assess the absolute prevalence of the disease. The diagnostic criteria for tuberculosis in skeletal remains are woefully inadequate, the disease being diagnosed only at an advanced stage of osseous involvement. The rate of skeletal involvement in relation to the overall prevalence of tuberculosis in antiquity is not known, and it is not justifiable to assume, unequivocally, that this was the same in antiquity as it is today, although this is likely. Neither is it possible at present from osteoarcheological specimens to determine the primary site of involvement, pulmonary or gastrointestinal.

During the reign of Edward the Confessor, a ritual was introduced for the cure of King's Evil. King's Evil was the name applied to cervical lymphadenitis. Although cervical lymphadenitis is of multiple etiology, tuberculous lymphadenitis secondary to initial tonsillar or pulmonary infection was a prominent cause in the prechemotherapeutic era. Tuberculous cervical lymphadenitis is traditionally equated with King's Evil. It may be significant, in epidemiological terms of primary site involvement, that this practice was introduced at the very time that urban development was a phenomenon and when movement of peoples in market trading was becoming established. Clearly, Touching for the King's Evil was of no anti-infective therapeutic value whatsoever, but the practice was probably of considerable spiritual and psychological benefit to the recipient, to say nothing of his or her financial improvement thereby! Introduction of this practice in England, and also in continental Europe at a similar time, around the late 10th or early 11th century, suggests that the disease was widely known, of increasing incidence, and socioeconomically significant. During the advancing Middle Ages, the practice continued and the number of patients touched increased.

By the mid 17th century it was recorded in the London Bills of Mortality that, in years free of plague, 20% of all deaths in the city were due to consumption (Clarkson 1975:39). Although there was no knowledge of bacteriology and no autopsy confirmation of diagnosis, the clinical features of advancing tuberculosis were doubtless known. Progressive untreated pulmonary tuberculosis, alternatively known as consumption or phthisis, presents with intractable cough, dyspnea, hemoptysis, and progressive emaciation. There can have been little confusion with nontuberculous pneumonia, and it is unlikely that carcinoma of the bronchus was a common disease. It is probable therefore that tuberculosis was, indeed, a common cause of death in 17th century urban centers.

In terms of polity there was, in contrast to leprosy, no segregation of the consumptive. Tuberculous individuals do not exhibit the physical mutilations of leprosy, there are no religious overtones associated with the disease, and presumably the infective nature was not suspected. Therefore, the expansion of medieval hospitals *(v.i.)* cannot be taken as evidence of an increasing incidence of tuberculosis. Neither was there legal enactment in respect of tuberculosis.

It is unfortunate that the evidence for an increase in incidence of tuberculosis during the advancing Middle Ages is circumstantial, based on documents and traditions which do not provide irrefutable proof of the disease. However, as mentioned, in current paleopathological practice, the osteoarcheological diagnosis of tuberculosis is mainly made on the spinal changes at an advanced stage of pathogenesis, at which caseous destruction of vertebral bodies and subsequent collapse has occurred. Earlier stages of the disease are rarely recognized and, as yet, diagnostic criteria for early lesions have not been established. Within the framework of constraint it has been remarked, in respect of prehistoric Amerindian peoples that "if a contagious disease like tuberculosis was present in overcrowded prehistoric populations, . . . then there should be many more cases than there have been found to date" (Morse 1978). Such statements reinforce the overwhelming need to establish criteria for the diagnosis of skeletal tuberculosis at early stages of pathogenesis in osteoarcheological contexts. Also, because the osteomyelitic and septic arthritic lesions in tuberculosis are metastatic changes from the initial site, it is not possible to establish the incidence and changing pattern of primary pulmonary and primary gastrointestinal tuberculosis. Such a facility would be of immense value in the paleoepidemiology of the disease. The considerations of rib lesions by Kelley and Micozzi (1984) may be a pointer in this differentiation, but the lesions described and also observed by Manchester and Roberts (1987) are probably the sequel of empyema which may have causes additional to pulmonary tuberculosis.

Notwithstanding these constraints of evidence, it seems certain that tuberculosis as a human disease was present in Britain at least by the Roman period, that it increased in prevalence throughout the Anglo-Saxon period, and that the incidence rate increased further during the post-Norman Conquest period. The disease did not effectively become controlled until the introduction of sanatoria and, particularly, the advent of antituberculous chemotherapy in the 20th century.

The rising incidence rate in the post-Norman Conquest period may be related to urban development and to population movement and aggregation consequent upon regular market development in the medieval period. A detailed examination of these paleodemographic changes is beyond the scope of this paper, but, in similar vein, it is noted by Allison (1979) that tuberculosis in Peruvian mummies was associated, in increasing prevalence, with the development of urban centers.

LEPROSY

The earliest evidence of leprosy is often quoted as facies leontina represented by a Canaanite jar from Beth-Shan, Palestine, dated to mid second millennium B.C. (Yoeli 1955). However, this jar is not considered representative of leprosy (Manchester and Zias, n.d.) but is, it is proposed, a portrayal of negroid facial features.

There is literary record, displaying obvious clinical acumen, of significant symptoms and signs of lepromatous leprosy in Sushruta Samhita (Dharmendra 1947:425–429), dated to 600 B.C. This, the earliest acceptably authentic evidence, indicates that the disease existed in India by the mid first millennium B.C. For a disease to have been so noteworthy to have been recorded in literature, it is likely, in antiquity, that it was present in the community for some considerable time previous. Rastogi and Rastogi (1984:541) have suggested that the word "kustha," mentioned in Indian literature in the 15th century B.C., denotes leprosy. However, this reference is without clinical description and is based on imprecise social attitudes toward a disease, the nature of which is uncertain. It is not justifiable, therefore, to attribute *M. leprae* infection to this reference.

From the second century B.C., a terra cotta figure has been considered representative of leprosy (Grmek 1983:232). Also of third century B.C. date, a Chinese Bamboo Book contains a description of physical signs and symptoms which are compatible with a diagnosis of leprosy (Skinsnes 1980).

As yet, the earliest skeletal evidence of leprosy in the world has been recorded from the Dakhleh Oasis, Egypt. Skeletons, of reputed European morphological type, have been diagnosed as leprous on rhinomaxillary change (Dzierzykray-Rogalski 1980). Professor Rogalski's suggestion is that these skeletons, a minor component of a larger negroid nonleprous cemetery, represent European individuals segregated from the Greek colonial center because of their disease. If this suggestion is valid, then such a policy of ostracism may indicate the presence of leprosy in the Mediterranean littoral for many years prior to the date of the cemetery at Dakhleh Oasis in the second century B.C. This may be compatible with a new disease appearing in Greece at about the third century B.C. and described by Straton, a physician of the Alexandrian School. Andersen (1969:45) suggests that leprosy may have been brought to the Mediterranean area from India by the returning armies of Alexander the Great, but this view is considered to be, perhaps, too simplistic. Passage of peoples from the Far East to the Near East was, surely, a phenomenon for many years at that time, and it is not necessary to attribute the transference of leprosy to such a precise event.

Further evidence of leprosy has been recorded in two Coptic mummies from Nubia. These specimens, dated to A.D. 500 exhibit peripheral skeletal and rhinomaxillary stigmata pathognomonic of lepromatous leprosy (Møller-Christensen and Hughes 1966).

From the seventh century A.D., skeletons exhibiting postcranial and rhinomaxillary changes of leprosy have been identified in the ossuaries of Byzantine period monasteries of the Judean Desert (Zias 1985).

From northern Europe, the earliest evidence of leprosy is discovered in skeletal remains from Poundbury, Dorset (Reader 1974). The skeletal remains, dated to the fifth century A.D. of late Romano-British context, consist of the lower legs and feet only, the sole fragments archeologically available for study. Although the absence of upper limbs and cranium for study reduces the validity of diagnosis, and doubt has been cast on this (T. Molleson, pers. comm. 1987), it is considered that, in differential diagnosis, leprosy is the most likely.

During succeeding centuries leprosy became widespread and of increasing prevalence throughout northern Europe and lands of the Mediterranean littoral, as attested by art form, literary, and skeletal evidence (Andersen 1969; Gladykowska-Rzeczycka 1976; Grmek 1983:227–260; Manchester 1981; Manchester and Roberts 1987; Møller-Christensen 1967; Skinsnes 1972; Wells 1962,1967).

As in the discussion of the history of tuberculosis, it is only necessary, for this paper, to discuss in more detail the history of leprosy in Britain.

Although the skeletal evidence of leprosy in post-Norman Conquest Britain is less profuse than in contemporaneous Scandinavia, a reflection of the research of Professor Møller-Christensen, the overall development and change, temporal and geographic, of leprosaria in medieval England is as well known as in any other European country. As has been discussed, the history of tuberculosis is also as well known in the period of disease contemporaneity as in any other country.

Unlike the study of other diseases, and unlike the study of leprosy in earlier centuries, the prevalence and change therein of leprosy in post-Norman Conquest Britain cannot be assessed from the isolated study of osteoarcheological remains. This is because segregation of leprosy sufferers in this period was certainly general, although not exclusive, policy. Leprous skeletons are found therefore in the cemeteries of leprosaria and less so in the cemeteries of nonlazar houses, monastic communities, and parish churches (Manchester and Roberts 1987). Thus, a diminishing number of leprous skeletons in nonlazar house cemeteries after the Norman Conquest must not be taken as indicative of a decline in the prevalence of the disease at this time. An assessment of leprosy prevalence and incidence can only be made by composite study of skeletal remains, lazar house foundations and development, lazar house records, and legal enactments. Such a multidisciplinary study is, at present, only in its infancy.

A gross numerical analysis, century by century, of recorded leprosaria in Britain shows a marked increase in their number during the period from the 11th to 13th centuries (Roberts 1986). In broad terms, the trend demonstrated by the figures is correct, and documented foundation dates are, in many cases, probably true foundation dates. However, there remains the possibility that the first literary mention of a lazar house may, erroneously, be construed as its foundation date, which may, in reality, have been many years previous. Neither should it be assumed that an observed rate of increase in lazar house foundations in unit time represents a similar arithmetic increase in the number of leprosy sufferers. No account is taken in these gross figures of lazar house foundations of the actual size of individual houses. To use a modern analogy, no account is taken of "bed state," no assessment of potential occupancy by leprosy sufferers is made. During the post-Norman Conquest years a religious fervor was developing, one outlet of which was the demonstration of personal conscience, piety, and wealth, outwardly manifest as the foundation and endowment of hospitals in the medieval period. It is noted, therefore, that not only is there an increase in lazar house foundations, but there is a similar increase in nonlazar house establishments (Roberts 1986).

Notwithstanding these constraints of interpretation, the increase in leprosaria and the legal enactments, royal dictates, and comments all indicate that leprosy as an endemic disease did increase in prevalence during the first three centuries after the Norman Conquest.

The rate of increase of lazar house foundations, or literary mentions, seems to have been maximal during the 12th and early 13th centuries, reaching a peak during the 13th century. Thereafter, foundations, although continuing, declined markedly. In the 16th century only four new houses were founded, three in the southwest of England and one in East Anglia (Richards 1977:83). As with the increase in foundations, so with the decline; this cannot be taken, in isolation, as a direct arithmetic indication of a declining incidence of leprosy. There is a decline in foundations of nonlazar hospitals, although this phenomenon postdates the lazar house decline by a century or so. This event for both may represent changing attitudes toward endowment, social changes consequent upon the decline of feudalism, or maybe a preoccupation with the ravages of the Hundred Years War and the Wars of the Roses. Additional documentary evidence does, however, indicate that the disease declined by the 15th century. The house of Sherburn was so reduced from an establishment of 65 leprosy sufferers at foundation in 1181 to two by 1434. By the mid 16th century Sherburn housed no lepers at all. Similar circumstances were recorded in this period at Ripon, Shrewsbury, and Ilford. It is unfortunate that there is, as yet, no osteoarcheological evidence to support this phenomenon of decline. The sole reason for this absence of evidence is that, hitherto, no significant excavation and postexcavation analysis of lazar houses and their cemeteries has been undertaken in Britain. The stratigraphic demonstration, by archeological excavation, of a declining number, through time, of leprosy skeletons in lazar house cemeteries would indeed be noteworthy.

Thus, it is reasonable to conclude that leprosy, as an endemic disease in Britain, was present at least by the late Roman period, increased steadily in incidence during the Anglo-Saxon and post-Norman Conquest period, reaching its zenith during the 13th century or so. Thereafter the disease declined and, except for pockets of isolation to a later century in southwest England and in northern Scotland, virtually disappeared by the 16th century.

What is not known, and would be so interesting epidemiologically, is whether there was any shift in the immunologically determined type of leprosy during its period as an endemic disease in Britain. Browne has suggested that lepromatous leprosy was the only type of leprosy of significance in antiquity. In this endemic infectious disease, one may theorize that the decline in prevalence may have been associated, *post hoc propter hoc*, with an increasing immunity and shift to the tuberculoid end of the spectrum. Although the presence of rhinomaxillary change in a skeleton is unequivocal evidence of lepromatous leprosy, the absence of this change is not a contraindication to this diagnosis. It may be that the individual of antiquity died before the development of the specific skull changes. In Professor Møller-Christensen's researches at Næstved (1961), nasal inflammatory change was found in 100% of skeletons with peripheral osseous stigmata of leprosy. Anterior nasal spine change was found in 76.2%, and alveolar process of maxilla change in 66%. This high proportion of rhinomaxillary change, perhaps as high as 100% of leprous skeletons, indicated the overwhelming prevalence of lepromatous disease in the leprosy sufferers of Næstved. There was no stratigraphic, and therefore no chronological, differentiation of the skeletons at Næstved. The prevalence of lepromatous disease is assumed to be constantly high throughout the period 1250–1550 when Næstved leprosarium was active. However, this does not affirm that the immunological pattern of the disease was universally constant throughout this period. Bone change pathognomonic of leprosy was found in 70% of the excavated skeletons at Næstved. This is much higher than the rate of involvement of bone in modern leprosy studies. The implication of this finding is threefold: the rate of bone involvement may have been higher in leprosy in antiquity than it is today; the modern figures may be clouded by therapy relatively early in the clinical course of disease, and such therapy was clearly not a feature of antiquity; that only the most severely affected leprosy sufferers, those with mutilating osseous change, were segregated in antiquity. There is no evidence, as yet, for the first two propositions. There is, however, evidence for the last proposition. Møller-Christensen discovered a leprous skeleton from his excavations at Æbelholt (1953:13–14), and Manchester and Roberts (1987), and Henderson (1985) have discovered leprous skeletons from Norton Priory and Guildford Friary. These sites are not associated with established medieval leprosaria. It is enigmatic however that these "extramural" skeletons exhibit advanced rhinomaxillary features of lepromatous disease. If the last proposition is correct, and more research is obviously necessary, then the high Næstved presence of lepromatous disease may not reflect the overall population immunological trend in the Middle Ages. The leprous population not segregated may have developed a shift, through time, toward the high-resistant end of the immunological spectrum. In so doing, by a reduction in the rate of bone involvement, the very evidence necessary in paleopathology may be nonexistent. The further hypothetical implication for infectivity in antiquity will be explored.

Interpretation

Tuberculosis and leprosy are diseases which coexist in many underdeveloped countries of the world today. Within the time scale of modern epidemiological observation, there has been little change in their natural prevalence, and there has not been any comparative change in incidence rate, one disease against the other. Analysis of tuberculosis and leprosy, separate and intercurrent, in individuals has suggested that there is no relationship between these two infectious diseases in mankind (Chaudhuri and Ghosh 1975:302). The essentially epidemiological research of these two workers examines the disease status of patients at a specific time. The data do not, and indeed cannot in the absence of a predetermined immune status, identify to which pathogen, *M. tuberculosis* or *M. leprae*, the host was first exposed. Contrary to earlier opinion, it is now known that there is no increased susceptibility in lepromatous leprosy to tuberculosis (Jopling 1982:305). Tuberculosis has been, however, a major cause of death in modern leprosaria. Although there are immunological implications of this fact, the main reason for the high number of tuberculous deaths in leprosaria probably lies in the environment with the facility for respiratory transmission of *M. tuberculosis*.

While epidemiology examines these two diseases and the changing patterns thereof through decades, paleoepidemiology examines through centuries. Precision in epidemiology, even with the constraints of Third World data, is obviously greater than in paleopathology, and in archaic contexts can never be complete. The multitude of environmental, social, biological, and therapeutic factors that influenced changing patterns of disease in antiquity can never be fully known. There must, therefore, be an element of surmise and calculated guesswork in the interpretation of disease in antiquity.

It is clear, as has been demonstrated *(v.s.)*, that tuberculosis and leprosy coexisted, at least for many centuries, in British antiquity. It is clear also that leprosy, as an endemic disease, declined and disappeared in the high Middle Ages, while tuberculosis continued unabated into modern times.

Leprosy, as a disease of single mode of transmission, demonstrates prevalence and incidence rates which are dependent upon population density, intrapopulation contact, intimacy of contact, and economic status. The increasing prevalence during the Anglo-Saxon period and, particularly, the early Middle Ages, is due to increasing population, to increasing population density, and to increasing contact between peoples. As a natural disease per se, it is expected that the prevalence of the early Middle Ages would be maintained or even increased during the later Middle Ages. This is demonstrated to be not so.

It is suggested that tuberculosis, a disease of animal and human host, and a human disease of two modes of transmission, was a biphasic human disease. In the earlier phases of tuberculosis history in Britain, it was a primary gastrointestinal disease by *M. bovis* transmitted from infected cattle. In the pre-urban era of low population density village society, tuberculosis was a sporadic disease. Therefore, a significant reservoir of immune, that is, tuberculin positive, population did not exist. Ikwueke (1984:1357) proposes that a new disease in a community is relatively virulent and tends to affect young generations. It is likely therefore that, in this first phase of development, many of the tuberculous individuals with primary gastrointestinal disease died, thereby removing them from the immune pool.

It is further suggested that, with increasing population, mobility of peoples, and urbanization, there was a biological adaptation of the tubercle bacillus to a respiratory mode of transmission. The increased population density, increased contact between peoples, and, perhaps, reduced hygienic standards, developing, *pari passu*, with urbanization, favored a widespread population exposure to *M. tuberculosis*. In antiquity, just as today, primary tuberculous infection occurred in young children. According to the concept of disease aging in the community (Ikwueke 1984:1356), there is a gradual decline in the severity of disease during its historic presence in mankind. Therefore, although many young children probably succumbed to the primary infection, many more survived with their consequent acquired immunity, manifest today by tuberculin sensitivity conversion. It is suggested that the urbanized population during the advancing Middle Ages became, in increasing numbers, immune to tuberculosis. Although village inhabitants had gradually developing contacts among themselves and with urban dwellers through market trading, the increasing incidence of tuberculosis may have been slight relative to the urban community. There may also have been a higher proportion of *M. bovis* infection relative to *M. tuberculosis* in the village, but this is of no significance in individual tuberculous immunity.

How far then may the coexistent developments of tuberculosis and leprosy in the medieval period in Britain be seen as independent entities, and how far may they be considered to demonstrate an interaction of infectious disease according to modern immunological concepts?

INDEPENDENT CHANGE

Similarities of bacterial form and properties of *M. tuberculosis* and *M. leprae* have been discussed. The respiratory mode of transmission is common to both infections, although the additional mode of gastrointestinal tuberculosis is distinct to the tubercle bacillus. Pulmonary tuberculosis and leprosy are, therefore, diseases dependent for their transference upon population density and intimacy of contact. Both diseases are more common in socially and economically disadvantaged groups; they are, in the main, diseases of the poor.

Thus it is expected that those factors, significant in the epidemiology of the two diseases, would influence infection by *M. tuberculosis* and *M. leprae* in the same direction. For example, in the absence of other influences, a deterioration or improvement in the socioeconomic status of the population would be expected to produce, respectively, an increase or a decrease in the incidence rates of both tuberculosis and leprosy. Increasing population density would be expected to favor increasing incidence rates of both diseases.

But, in spite of these common influential factors, the two diseases did not move in parallel in British antiquity. The increasing prevalence of both diseases during the late Anglo-Saxon and early post-Norman Conquest periods has been outlined, but the continued increase of tuberculous prevalence and the simultaneous and rapid decline of leprosy in the later Middle Ages is a major fact.

Ikwueke has proposed (1984:1357) that in the concept of aging disease, there is a gradual decline in severity, and that disease eventually declines and dies. This phenomenon is proposed in the absence of effective therapy. From the centuries of coexistence in antiquity, osteoarcheological and documentary evidence does not support the tenet of declining severity in tuberculosis and leprosy. Within the constraints of archeological stratigraphy already considered, there is no paleopathological evidence to suggest that leprosy, during its period of epidemiological decline, declined in severity. But in this context it is acknowledged that a decline in severity, that is, a "population upgrade in immunity" from lepromatous to tuberculoid leprosy, may be accompanied by a gradual disappearance of pathognomonic bone change. Thus, an absence of osteological evidence of severity decline may not be truly evidence of absence. This problem remains unresolved. The corollary of the decline in severity proposed by Ikwueke is the decline and disappearance of the disease. It is true, of course, that both diseases have declined in Britain, and that leprosy has disappeared as an endemic disease. The decline of tuberculosis was, however, a phenomenon of the post-Industrial Revolution era, even before the advent of antituberculous chemotherapy, whereas leprosy decline was some 500 years or so previous and was a much more rapid phenomenon. Was, perhaps, segregation a factor influencing this medieval decline of leprosy? It has been noted that the incubation period of leprosy is long, during which time there are no clinical manifestations to warrant segregation, and during which time the infected individual was, himself, infectious. As a preventative of transmission, segregation was clearly of limited value because the practice was akin to "shutting the stable door when the bacterial horse has bolted." Also, as noted *(v.s.)*, segregation of infected individuals in the medieval community was not absolute.

Genetic immunity is of unknown, but probably little, significance in tuberculosis and leprosy. Ikwueke comments (1984:1357) that there is growing evidence which makes transmission of acquired features a feasible proposition. How far this is applicable to immunity is unknown.

An equally vexed problem is that of genetic susceptibility to the two diseases. Fine (1981:452) remarked that there is evidence, albeit contentious, for some role of genetic factors in determining responses in leprosy and tuberculosis, although a "major role of genetic polymorphisms in determining epidemiological patterns" is unconvincing. Thus, although there may be some genetic influence in the initial setting of an individual on the immune spectrum in leprosy, it is doubtful if genetic factors are an influence, *ab initio*, in the susceptibility of individuals to infection by *M. tuberculosis* or *M. leprae*.

INTERACTION

As already discussed, immunity in both leprosy and tuberculosis is cell mediated, and antibodies have been considered unimportant, although in leprosy this matter is under review (Harboe 1981:5–6). The cell mediated immunity is not absolutely pathogen specific, as indicated in the varying response to infection by *M. leprae* following prior inoculation with BCG.

The concept of cross immunity between leprosy and tuberculosis was examined in depth by Chaussinand (1948, 1950), and by Lowe and McNulty (1953). Chaussinand's proposals are based on epidemiological observations of the two diseases in Africa, India, Indochina, and Japan. Immunological evidence in support of cross immunity is outlined earlier in this paper *(v.s.)*. Consideration of cross immunity in relation to the history of the two diseases in mankind has also been made by Steinbock (1976:197–198), Grmek (1983:291–306), Manchester (1984), and Clark et al. (1987:50–51).

What support then does paleoepidemiology afford to the concept of cross immunity and interaction of disease?

It is suggested that, within the village-orientated and relatively isolated communities of the sub-Roman and Anglo-Saxon periods, tuberculosis was of primary gastrointestinal focus due to *M. bovis*. Infection was sporadic and overall population immunity was insignificant. Both leprosy and tuberculosis smoldered unabated. Toward the end of the Anglo-Saxon period, townships and markets developed. These aspects developed particularly with the Norman and later towns and cities. Population density, consequent upon this development, prompted respiratory transmission of *M. tuberculosis*. The introduction of Touching for King's Evil, an indication of droplet transmission, is in support. Crowding of peoples facilitated the widespread urban, and to a lesser extent rural, exposure to the tubercle bacillus. Then, as in more modern context, the primary infection was probably in the young below the age of five years. Those infants surviving the primary infection were, in consequence, tuberculin positive and had "lifelong" immunity to tuberculosis. As noted, the age of exposure to *M. leprae* is, even today, not known, but it is suggested that, epidemiologically, this may be at a somewhat older age than *M. tuberculosis* exposure. Thus, exposure to *M. leprae* may have been, in the population as a whole, to a people already immune to tuberculosis by previous primary infection. The protection afforded thereby may have prevented the establishment of clinical leprosy, except in those individuals whose immune response was compromised by poverty, malnutrition, or intercurrent disease. Any improvement in these features during the advancing Middle Ages would favor the development of an immune population. Furthermore, as has been noted, the simultaneous presentation of *M. tuberculosis* and *M. leprae* to a person sensitized by previous exposure to *M. tuberculosis* may prevent the development of both infections. In communities in which both diseases were common, such simultaneous exposure may, indeed, have occurred.

Furthermore, in those individuals who developed clinical disease, the immunity conferred by prior exposure to *M. tuberculosis* may have caused a "right shift" along the Ridley-Jopling spectrum toward tuberculoid leprosy. This clinical manifestation of *M. leprae* infection is associated with relative noninfectivity. Thus, this diminution in leprous population infectivity may have contributed to the eradication of the disease.

It is possible therefore that the rapid decline of leprosy from a population in which the more virulent disease of tuberculosis was both widespread and increasing may be paleoepidemiological evidence of a cross immunity between the two diseases.

The coexistence and increasing prevalence of the two diseases took several centuries to develop. The decline of leprosy, consequent upon and subsequent to the increasing prevalence of tuberculosis, likewise took several centuries to occur. Modern epidemiological studies of these two diseases, influenced as they are by effective therapy, lack the prolonged time dimension which only paleopathology, paleoepidemiology, and medical history can supply.

Disease is not solely a phenomenon of the modern world, to be studied only in its 20th century context. Disease has a past, it has a present, and will have a future. No one facet should be studied in isolation and ignorance of the others.

Literature cited

Allison, M.J. 1979. Paleopathology in Peru. *Natural History,* 88:74–82.

Andersen, J.G. 1969. *Studies in the Medieval Diagnosis of Leprosy in Denmark.* Copenhagen: Costers Bogtrykkeri.

———. 1982. The Osteo-Archaeological Diagnosis of Leprosy. *Proceedings of Paleopathology Association 4th European Meeting,* 221–226. Middleburg/Antwerpen.

Badger, L.F. 1964. Epidemiology. In R.G. Cochrane and T.F. Davey, eds., *Leprosy in Theory and Practice.* Bristol, U.K.: John Wright and Sons.

Bartels, P. 1907. Tuberkulose (Wirbelkaries) in der jungeren Steimheit. *Archiv fur Anthropologie,* 6:243–255.

Buikstra, J.E., and D.C. Cook 1981. Pre-Columbian Tuberculosis in WestCentral Illinois: Prehistoric Disease in Biocultural Perspective. In J.E. Buikstra, ed., Prehistoric Tuberculosis in the Americas. *Northwestern University Archeological Program Scientific Papers,* 5:115–139. Evanston, Ill.: Northwestern University.

Cave, A.J.E. 1939. Evidence for the Incidence of Tuberculosis in Ancient Egypt. *British Journal of Tuberculosis,* 33:142–152.

Chaudhuri, S., and S. Ghosh. 1975. Leprosy and Tuberculosis: Immune Allergic Relationship. *Leprosy in India,* 47:295–306.

Chaussinand, R. 1948. Tuberculosis and Leprosy-Antagonistic Illnesses. *International Journal of Leprosy,* 16:431–438.

———. 1950. *La Lepre.* Paris: Expansion Scientifique Française.

Clark, G.A., M.A. Kelley, J.M. Grange, and M. Cassandra. 1987. The Evolution of Mycobacterial Disease in Human Populations. *Current Anthropology,* 28:45–62.

Clarkson, L. 1975. *Death, Disease and Famine in Pre-Industrial England.* Dublin: Gill and MacMillan.

Cockburn, A. 1963. *The Evolution and Eradication of Infectious Diseases.* Baltimore: Johns Hopkins University Press.

Collins, C.H., and J.M. Grange. 1983. The Bovine Tubercle Bacillus. *Journal of Applied Bacteriology,* 55:13–29.

Dharmendra. 1947. Leprosy in Ancient Indian Medicine. *International Journal of Leprosy,* 15:424–430.

Dzierzykray-Rogalski, T. 1980. Paleopathology of the Ptolemaic Inhabitants of Dakhleh Oasis (Egypt). *Journal of Human Evolution,* 9:71–74.

Fine, P.E.M. 1981. Immunogenetics of Susceptibility to Leprosy, Tuberculosis and Leishmaniasis. An Epidemiological Perspective. *International Journal of Leprosy,* 49:437–454.

———. 1984. Leprosy and Tuberculosis—An Epidemiological Comparison. *Tubercle,* 65:137–153.

Formicola, V., Q. Milanesi, and C. Scarsini. 1987. Evidence of Spinal Tuberculosis at the Beginning of the Fourth Millennium B.C. from Arena Candide Cave (Liguria, Italy). *American Journal of Physical Anthropology,* 72:1–7.

Gladykowska-Rzeczycka, J. 1976. Case of Leprosy from a Medieval Burial Ground. *Folia Morphologia,* 35:253–264. Warsaw.

Grange, J.M. 1980. *Mycobacterial Diseases.* London: Edward Arnold.

Grmek, M.D. 1983. *Les Maladies a l'Aube de la Civilisation Occidentale.* Paris: Payot.

Harboe, M. 1981. *Mycobacterium leprae* and the Host Response. *Leprosy Review,* supplement, 1:1–14.

Hunter, J.M. 1986. Hansen's Disease and Towns in Africa. Part 1. *The Star,* 43:4–7.

Ikwueke, K. 1984. The Changing Pattern of Infectious Disease. *British Medical Journal,* 289:1355–1358.

Jopling, W.H. 1982. Clinical Aspects of Leprosy. *Tubercle,* 63:295–305.

Kelley, M.A., and M.S. Micozzi. 1984. Rib Lesions in Chronic Pulmonary Tuberculosis. *American Journal of Physical Anthropology,* 65:381–387.

Lowe, J., and F. McNulty. 1953. Tuberculosis and Leprosy: Immunological Studies in Healthy Persons. *British Medical Journal*, 579–584.

Mackaness, G.B. 1967. The Immunology of Antituberculous Immunity. *American Review of Respiratory Disease*, 97:337.

Manchester, K. 1981. A Leprous Skeleton of the 7th Century from Eccles, Kent, and the Present Evidence of Leprosy in Early Britain. *Journal of Archaeological Science*, 8:205–209.

———. 1984. Tuberculosis and Leprosy in Antiquity: An Interpretation. *Medical History*, 28:162–173.

———. 1986. Tuberculosis: An Evolutionary Model. *Proceedings of Paleopathology Association 6th European Meeting, 189–198*. Madrid.

Manchester, K., and C.A. Roberts. 1987. *Paleopathological Evidence of Leprosy and Tuberculosis in Britain*. SERC Research Report. Bradford, U.K.: University of Bradford.

Manchester, K., and J. Zias. n.d. The Leprous Jar from Beth-Shan: A Reappraisal. Manuscript on file.

Mercer, W. 1964. Then and Now: History of Skeletal Tuberculosis. *Journal of Royal College of Surgeons of Edinburgh*, 9:243–254.

Møller-Christensen, V. 1953. *Ten Lepers from Næstved in Denmark*. Copenhagen: Danish Science Press.

———. 1961. *Bone Changes in Leprosy*. Copenhagen: Munksgaard.

———. 1967. Evidence of Leprosy in Earlier Peoples. In D.R. Brothwell and A.T. Sandison, eds., *Diseases in Antiquity*. Springfield, Ill.: Charles C Thomas.

———. 1978. *Leprosy Changes of the Skull*. Odense, Sweden: University Press.

Møller-Christensen, V., and D.R. Hughes. 1966. An Early Case of Leprosy from Nubia. *Man*, 1:242–243.

Morse, D. 1978. *Ancient Disease in the Midwest*. Springfield, Ill.: Illinois State Museum.

Morse, D., D.R. Brothwell, and P.J. Ucko. 1964. Tuberculosis in Ancient Egypt. *American Review of Respiratory Disease*, 90:524–543.

Ortner, D.J. 1979. Disease and Mortality in the Early Bronze Age People of Bab edh-Dhra, Jordan. *American Journal of Physical Anthropology*, 51:589–598.

Pedley, J.C., and J.G. Geater. 1976. Does Droplet Infection Play a Role in the Transmission of Leprosy? *Leprosy Review*, 47:97–102.

Rastogi, R.C., and N. Rastogi. 1984. Leprosy in Ancient India. *International Journal of Leprosy*, 32:541–543.

Reader, R. 1974. New Evidence for the Antiquity of Leprosy in Early Britain. *Journal of Archaeological Science*, 1:205–207.

Richards, P. 1977. *The Medieval Leper and His Northern Heirs*. Cambridge, U.K.: D.S. Brewer.

Ridley, D.S., and W.H. Jopling. 1966. Classification of Leprosy According to Immunity. A Five-Group System. *International Journal of Leprosy*, 34:255–273.

Roberts, C. 1986. Leprosy and Leprosaria in Medieval Britain. *MASCA Journal*, 4:15–21.

Roitt, I.M. 1980. *Essential Immunology*. 4th edition. London: Blackwell Scientific Publications.

Sager, P.L., M. Schalimtzek, V. Møller-Christensen. 1972. A Case of Spondylitis Tuberculosa in the Danish Neolithic Age. *Danish Medical Bulletin*, 19:176–180.

Skinsnes, O.K. 1972. Leprosy Sketches from a Chinese Brush *International Journal of Leprosy*, 40:309–313.

———. 1980. Leprosy in Archaeologically Recovered Bamboo Book in China. *International Journal of Leprosy*, 48:333.

Steinbock, R.T. 1976. *Paleopathological Diagnosis and Interpretation*. Springfield, Ill.: Charles C Thomas.

Stuart-Macadam, P.L. 1982. A Correlative Study of a Palaeopathology of the Skull. Ph.D. dissertation, Department of Anthropology, Cambridge University, Cambridge, U.K.

Suzuki, T. 1985. Paleopathological Diagnosis of Bone TB in the Lumbosacral Region. *Journal of Anthropological Society of Nippon*, 93:381–390.

Weir, D.M. 1986. *Aids to Immunology*. London: Churchill Livingstone.

Wells, C. 1962. A Possible Case of Leprosy from a Saxon Cemetery at Beckford. *Medical History*, 6:383–386.

———. 1967. A Leper Cemetery at South Acre, Norfolk. *Medieval Archaeology*, 11:242–248.

———. 1982. The Human Burials. In A. McWhirr, L. Viner, and C. Wells, *Romano-British Cemeteries at Cirencester*, 185. Cirencester, U.K.: Cirencester Excavations Committee.

World Health Organization. 1982. Tuberculosis Control. *World Health Organization Technical Report Series*, 671. Geneva: World Health Organization.

———. 1985. Epidemiology of Leprosy in Relation to Control. *World Health Organization Technical Report Series*, 716. Geneva: World Health Organization.

Yoeli, M. 1955. "Facies Leontina" of Leprosy on an Ancient Canaanite Jar. *Journal of the History of Medicine*, 10:331–335.

Zias, J. 1985. Leprosy in the Byzantine Monasteries of the Judean Desert. *Koroth*, 9:242–247.

SUMMARY OF AUDIENCE DISCUSSION: The immune status as defined by the skin tuberculin test has only a minimal influence on susceptibility to infection with contraction of clinical tuberculous disease, but it is the dominant factor in determining the form of the infection. A nonimmune individual is more apt to develop the rapid and often lethal course of tuberculous pneumonia or hematogenous spread of "miliary" tuberculosis, while the contained form of the disease (usually restricted to the lung) is more apt to occur in the tuberculin-positive ("immune") person. In the United States the death rate fell dramatically during the century preceding 1960, unaffected by the laboratory identification of the tubercle bacillus, isolation of patients in sanatoria, or specific chemotherapy (the latter available only about the final 10 years of the period). Furthermore, except for the final decade, most people during that period were tuberculin-positive by age 18. Neither the immune status nor therapy appears to have been responsible for the straight-line decline. More probable contributions include a reduction in the "dose" (i.e., number of inhaled organisms) secondary to reduction in crowding and better housing which tend to reduce rebreathing of others' exhaled air. At least today tuberculosis is a population-density disease.

Recognition of empyema's characteristic rib periostitis lesions as described by Kelley may double the frequency of identifiable tuberculous lesions in skeletal populations, although the frequency of nontuberculous empyema secondary to pyogenic pneumonia in antiquity is unknown—a problem resolvable through the study of mummies with soft tissue preservation.

Porotic hyperostosis: Changing interpretations

Patty Stuart-Macadam

Porotic hyperostosis is a paleopathologic condition that has intrigued researchers for over 100 years. This time period has seen a development of thought concerning both etiology and interpretation of the biological significance of the lesions. In the past a profusion of terminology existed, but today the term porotic hyperostosis (after Angel 1966) is commonly used to describe characteristic bone lesions seen in human skeletal material. These lesions are usually symmetrical in distribution and occur mainly on the orbits (also known as cribra orbitalia) and the skull vault, particularly the frontal, parietal and occipital bones. The normally smooth, dense outer compact bone is replaced by small holes of varying size and density. In addition, the middle layer of bone, or diploe, is often increased in thickness. A review of past and current ideas on porotic hyperostosis illustrates how science progresses by building upon the work of earlier researchers and by the development of new interpretations of data as the result of changing belief structures or paradigms.

Early researchers focused on descriptions of the lesions and identification of the etiology. Although Welcker (1888) is credited with the first good description of porotic hyperostosis, Owen in 1859 may have been the first researcher to comment on the lesions observed in skeletal material. In his examination of a collection of early 19th century crania from East Asia, Owen came across some striking cases of porotic hyperostosis. He noticed great thickening of the cranial vault and commented that one skull in particular "is chiefly remarkable as exemplifying the rare disease of hypertrophous thickening of the parietal bones." Ideas about etiology proliferated, with suggestions such as pressure from cradle boards (Williams 1929), stress of carrying water jugs on the head (Wood-Jones 1910), toxic disorders (Hrdlicka 1914), genetic trait (Adachi 1904), or nutritional deficiencies (Williams 1929).

In 1929 two researchers independently suggested that anemia may be the causative factor (Moore 1929; Williams 1929). They based their opinion on the striking similarity between radiographs of skulls with porotic hyperostosis and those from clinical cases of various hemolytic anemias. In both cases, bone changes include an increase in the diploe, loss of outer compact bone integrity, and a "hair-on-end" appearance of trabeculae. Initially a genetic anemia was implicated but iron deficiency anemia became a possibility when it was realized that it too could produce the characteristic skull changes.

Angel (1964, 1966, 1967) popularized the idea that a genetic anemia, particularly thalassemia, could be responsible for lesions of porotic hyperostosis in earlier skeletal material. He was one of the first researchers to develop a population approach to this issue and to have an evolutionary perspective. Angel's work on Greek skeletal material suggested to him that thalassemia may have occurred as an adaptation to some disease such as malaria or amoebiasis. Moseley (1961) was the first researcher to suggest that iron deficiency anemia may also be a factor in porotic hyperostosis. Hengen, in 1971, put forward the hypothesis that iron deficiency anemia was the exclusive cause of cribra orbitalia. He had a broad perspective and saw the population in terms of its interaction with the environment. He considered that parasitic infestation and/or an iron deficient diet, consequent on local conditions, were responsible for porotic hyperostosis. He said: "Changes of the hygienic conditions and of the incidence of iron deficiency anemias in former times depended without doubt largely on deviations of the climate, differences of the habits of daily life, procuring and preparation of food, types of housing, keeping of domestic animals, disposal of excrement and so on."

Later researchers continued to use a population approach and to explore the complex interaction of factors behind the occurrence of anemia. Carlson et al. (1974) speculated that poor diet, parasitic infection, and weanling diarrhea contributed to the development of iron deficiency anemia in Nubian populations. Lallo et al. (1977) suggested a synergistic relationship between microbial infection, malabsorption due to weanling diarrhea, and the nutrient depletion that occurs with rapid growth. Mensforth et al. (1978) also stressed the multifactorial nature of the problem and illustrated that infectious diseases, represented by periosteal reactions, can also play a role in the story.

By the 1970s porotic hyperostosis was considered to be a good stress marker for assessing the health and nutritional status of past human skeletal populations. Skeletal populations with porotic hyperostosis were considered to be "less successful" in adapting to their environment than those without lesions. The general feeling was that females had a greater incidence of porotic hyperostosis than males, and that this corresponded to modern incidences of iron deficiency anemia. It was also believed that juveniles had a much higher incidence of porotic hyperostosis, hence iron deficiency anemia, than adults. Some researchers began to equate the presence of porotic hyperostosis with iron deficient diets.

By the 1980s some of the assumptions behind these interpretations began to be challenged. Researchers showed that even groups who consumed diets rich in iron suffered from porotic hyperostosis (Walker 1986). The belief that porotic hyperostosis was more common in females was shown to be not true. Statistical analysis of a number of studies revealed that in almost every case there was no significant difference between males and females in incidence of porotic hyperostosis (Stuart-Macadam 1982); if porotic hyperostosis was due to iron deficiency anemia, then why were there not significant differences between males and females similar to those that have been observed in clinical and population studies for as long as iron deficiency anemia has been documented?

Consideration of bone marrow physiology and data on iron deficiency anemia from clinical studies suggests that lesions of porotic hyperostosis in adults are not representative of an episode of anemia that was current or had occurred within a relatively short period prior to death (Stuart-Macadam 1985). In fact, porotic hyperostosis seen in adults is probably indicative of a childhood episode of anemia, with the resultant bone lesions showing incomplete remodeling. This would explain the discrepancy between modern demographic patterns of iron deficiency anemia and the pattern of porotic hyperostosis in earlier human populations. In adults, marrow hyperplasia can occur without putting undue stress on the available marrow space and producing bony response (Stuart-Macadam 1985). There is no evidence from clinical studies to suggest that bone changes as a result of marrow hyperplasia can occur in an adult who has only recently acquired iron deficiency anemia. In young children, however, the factors of great bone malleability and marrow space already filled to capacity with red marrow are likely to lead to bone change in response to increased demands for red blood cells.

These ideas have implications for the interpretation of porotic hyperostosis seen in past human skeletal populations. If iron deficiency anemia acquired in adulthood does not lead to bone change then the higher incidence of porotic hyperostosis (therefore anemia) observed in juveniles does not necessarily reflect reality. Juveniles may or may not have had a higher incidence of anemia than adults; it is simply not possible to assess the impact that anemia may have had on adults. If porotic hyperostosis in adults does reflect a childhood episode of anemia, then the search for causative factors should concentrate on the juvenile sector of the population. It is also important to be aware that the total number of individuals affected by anemia will always be underrepresented in a skeletal population. At most, 50–75% of clinical patients with anemias that are associated with bone change show changes which can be seen radiographically (Stuart-Macadam 1985). As a result of all these factors it may be impossible to assess the true pattern of anemia in any past human skeletal population.

Stuart-Macadam (1982,1987b) expanded on the ideas of some of the earlier researchers by undertaking a detailed comparison of radiographs from clinical cases of anemia with radiographs of skulls with porotic hyperostosis. On the basis of seven criteria, it was felt that the data strongly supported the hypothesis that porotic hyperostosis was indeed the result of an anemia. In addition, three lines of evidence supported the hypothesis that porotic hyperostosis is more likely due to iron deficiency anemia than a genetic anemia:

1. Calculations based on the highest gene frequencies for genetic anemias seen today show that the probability of finding individuals from archeological collections with skeletal changes due to genetic anemia is quite low (Stuart-Macadam 1982).
2. There are high levels of porotic hyperostosis in skeletal groups from northern Europe and North America, areas where genetic anemias did not exist in the past.
3. The severe bone changes associated with genetic anemias, particularly postcranially, have not been substantiated for any individuals from archeological collections.

Recently, Stuart-Macadam (1987a) has questioned the biological significance of porotic hyperostosis. Formerly its presence in a skeletal population has been seen as an inability to adapt, a negative response on the part of the body. It has been assumed that skeletal groups with high levels of porotic hyperostosis were less "successful" than groups with lower levels of porotic hyperostosis. It may be that it is actually a positive response and a sign of a healthy defense system. This view has arisen out of changing perspectives of the immune system, and iron deficiency in particular. A large body of data supports the concept that iron deficiency may not always be detrimental, but may actually strengthen the body's defenses against infection (Strauss 1978). Lowered iron levels may be a natural protective response which discourages bacteria and other pathogens. Microbes are dependent upon assimilation of iron and actually synthesize substances which have the ability to bind iron. In this situation hypoferremia should be advantageous to the host and disadvantageous to the microbial invader. In vitro, in vivo, and population studies show that this is very often the case (Lukens 1975; Strauss 1978; Wadsworth 1975).

Normally there is a balance between the role of iron in the defense system and the body's requirements for iron. Iron metabolism is basically a closed system, since losses are normally very low and iron obtained from destruction of old blood cells by the reticuloendothelial system is recycled within the body. In times of extra iron requirements the intestine absorbs a greater percentage of the iron available in the diet. It is possible that only continual and repeated exposure to a large number of pathogens can destroy this balance in a normal individual and ultimately lead to iron deficiency anemia. In this view it is possible that the lesions of porotic hyperostosis reflect a positive response on the part of the body to the total pathogen load of the environment (Stuart-Macadam 1987a). In areas where the load of pathogens (viruses, bacteria, fungi, parasites) is high, it would be expected that greater numbers of the population would cross the threshold between an iron deficiency as an adaptive response, and an iron deficiency anemia.

An examination of the demographic picture of porotic hyperostosis in earlier populations should provide clues to the biological significance of the condition. Porotic hyperostosis begins to appear in Neolithic times (Stuart-Macadam 1987a), it is more prevalent the closer the group is to the equator (Hengen 1971), it occurs with greater frequency on lowland sites than highland (Hrdlicka 1914; El-Najjar et al. 1976; Ubelaker 1984), it has decreased in modern times in northern Europe (Hengen 1971), and it occurs in nonhuman primates (Nathan and Haas 1966). What is the significance of this? Building on the ideas of previous researchers who have suggested that porotic hyperostosis is the result of the interaction of customs, diet, hygiene, parasites, and infectious diseases, the concept of porotic hyperostosis as a response to high pathogen loads could explain why individuals from archeological sites who appear to have diets rich in iron and protein still have high levels of porotic hyperostosis. It could explain the fact that porotic hyperostosis is more common in areas such as lowland sites and tropical areas: these are areas of higher pathogen loads. It could also explain why porotic hyperostosis starts becoming common in the Neolithic, not because of agriculture or changes in diet per se, but because of greater population density which means greater exposure to pathogens.

In the past, signs of chronic disease in skeletal material have been interpreted as an inability to adapt to the environment. However, it is becoming more widely accepted that disease is the defense system's fight for health; in this view evidence for chronic disease on the skeleton is seen as a positive adaptation, a fight for health against the pathogen. As Powell (1986) has said, "since bone lesions typically occur relatively late in the progress of the disease after considerable soft tissue involvement has commenced, their very presence is indicative of long-term immune response to infection." By a shift in paradigm, individuals with chronic bone lesions can be seen to have been more successful in adapting to their environment than individuals who did not live long enough to produce bony response to a pathogen. If this is true, then skeletal populations that show signs of chronic bone lesions may not be less "successful" than populations that do not show signs of chronic bones lesions. Of course, ultimately, fertility and longevity are the true indicators of successful adaptation.

Another shift in perception in recent years has been an understanding of the complexities of the interaction between the host, pathogen, and environment. Scrimshaw (1964) wrote a classic paper in which he emphasized that the disease agent is only one of a triad with host factors and environmental factors. He said that simply identifying the agent is not sufficient to describe its cause. It must be seen as part of a complex interrelationship which is dynamic, and unique to every individual. These views are reflected in the more recent work on porotic hyperostosis, as researchers carefully examine as many variables as possible in the complex interaction between a population and its environment.

This paper has presented a review of the research on porotic hyperostosis and the development of thought that has occurred over the past century regarding etiology and interpretation. Most earlier work would fit into Armelagos et al.'s (1982) descriptive-historical model of research. This refers to research which focuses mainly on data description and discussion of individual cases of paleopathology. In recent years, there has been a shift to a population approach that stresses functional interpretation of data. At the same time, concepts of health and disease have been changing, along with perceptions of the body and its immune system. These trends have had a profound effect on research into porotic hyperostosis and have stimulated new ideas and interpretations of data.

Literature cited

Adachi, B. 1904. Die porositat des schadels. *Zeitschrift fur Morphologie und Anthropologie*, 7:373.

Angel, J.L. 1964. Osteoporosis: Thalassemia? *American Journal of Physical Anthropology*, 41:103–106.

______. 1966. Porotic Hyperostosis, Anemias, Malarias and the Marshes in Prehistoric Eastern Mediterranean. *Science*, 153:760–762.

______. 1967. Porotic Hyperostosis or Osteoporosis Symmetrica. In D. Brothwell and A.T. Sandison, eds., *Diseases in Antiquity*, 378–389. Springfield, Ill.: Charles C Thomas.

Armelagos, G.J., D.S. Carlson, and D.P. van Gerven. 1982. The Theoretical Foundation and Development of Skeletal Biology. In F. Spencer, ed., *A History of American Physical Anthropology 1930–1980*, 305–328. New York: Academic Press.

Carlson, D., G.J. Armelagos, and D. van Gerven. 1974. Factors Influencing the Etiology of Cribra Orbitalia in Prehistoric Nubia. *Journal of Human Evolution*, 3:405–410.

El-Najjar, M., D.J. Ryan, C.G. Turner II, and B. Lozoff. 1976. The Etiology of Porotic Hyperostosis Among the Prehistoric and Historic Anasazi Indians of the Southwestern U.S. *American Journal of Physical Anthropology*, 44:477–488.

Hengen, O.P. 1971. Cribra Orbitalia: Pathogenesis and Probably Etiology. *Homo*, 22:57–75.

Hrdlicka, A. 1914. Anthropological Work in Peru in 1913, with Notes on Pathology of Ancient Peruvians. *Smithsonian Miscellaneous Collections*, 61:1–69.

Lallo, J., G.J. Armelagos, and R.P. Mensforth. 1977. The Role of Diet, Disease and Physiology in the Origin of Porotic Hyperostosis. *Human Biology*, 49:471–483.

Lukens, J. 1975. Iron Deficiency and Infection. *American Journal of Diseases of Children*, 129:160–162.

Mensforth, R., C. Lovejoy, J. Lallo, and G.J. Armelagos. 1978. The Role of Constitutional Factors, Diet, and Infectious Disease in the Etiology of Porotic Hyperostosis and Periosteal Reactions in Prehistoric Infants and Children. *Medical Anthropology*, 2:1–59.

Moseley, J.E. 1961. Skull Changes in Chronic Iron Deficiency Anemia. *American Journal of Roentgenology and Radium Therapy*, 85:649–652.

Moore, S. 1929. Bone Changes in Sickle Cell Anemia with Note on Similar Changes Observed in Skulls of Ancient Mayan Indians. *Journal of Missouri State Medical Association*, 26:561–564.

Nathan, H., and N. Haas. 1966. The Presence of Cribra Orbitalia in Apes and Monkeys. *American Journal of Physical Anthropology*, 24:351–360.

Owen, R. 1859. Report on a Series of Skulls of Various Tribes of Mankind Inhabiting Nepal, Collected and Presented to the Museum by B.H. Hodgson. *Report British Association*. London.

Powell, M.S. 1986. Bone Lesions of Chronic Disease: A Cautionary Note for Interpretation. Paper presented at the 55th annual meeting of the American Association of Physical Anthropology. Albuquerque, New Mexico.

Scrimshaw, N. 1964. Perspectives in Nutrition. *American Journal of Clinical Nutrition*, 14:112–122.

Strauss, R. 1978. Iron Deficiency, Infection, and Immune Function: A Reassessment. *American Journal of Clinical Nutrition*, 31:660–666.

Stuart-Macadam, P.L. 1982. A Correlative Study of a Palaeopathology of the Skull. Ph.D. dissertation, Department of Physical Anthropology, Cambridge University, Cambridge, U.K.

______. 1985. Porotic Hyperostosis: Representative of a Childhood Condition. *American Journal of Physical Anthropology*, 66:391–398.

______. 1987a. Nutrition and Anaemia in Past Human Populations. *Chacmool*. Alberta, Canada: University of Calgary.

______. 1987b. A Radiographic Study of Porotic Hyperostosis. *American Journal of Physical Anthropology*, 74:511–520.

Ubelaker, D.H. 1984. Prehistoric Human Biology of Ecuador: Possible Temporal Trends and Cultural Correlations. In M.N. Cohen and G.J. Armelagos, eds., *Paleopathology at the Origins of Agriculture*, 491–513. New York: Academic Press.

Wadsworth, G.R. 1975. Nutritional Factors in Anemia. *World Review of Nutrition and Dietetics*, 21:75–150.

Walker, P.L. 1986. Porotic Hyperostosis in a Marine-Dependent California Indian Population. *American Journal of Physical Anthropology*, 69:345–354.

Welcker, H. 1888. Cribra Orbitalia, ein ethologisch-diagnostisches merkmal am schadel mehrerer menschrassen. *Archive fur Anthropologie*, 17:1.

Williams, H. 1929. Human Paleopathology. *Archives of Pathology*, 7:839.

Wood-Jones, F. 1910. General Pathology (Including Diseases of the Teeth). In G.E. Smith and F. Wood-Jones, eds., *Report on the Human Remains. The Archaeological Survey of Nubia*. Report for 1907–1908, vol. 2. Cairo.

SUMMARY OF AUDIENCE DISCUSSION: Porotic hyperostosis presents problems of both diagnosis and etiology. Minor osteoporotic pitting or outer table erosion above the temporal lines and on frontal squama as well as above the occipital crest without frank thickening should probably be given a descriptive label which does not bear the etiological implications presumed for porotic hyperostosis. Site specificity can also be a problem. While the material reviewed in this presentation demonstrated involvement of both cranial porotic hyperostosis and orbital cribra orbitalia in 90% of the individuals studied, the range of such associations varies from 0 to 100% in different groups. It should be remembered that porotic hyperostosis is an indicator of childhood, not adult, anemia, and that iron deficiency anemia may be the consequence of blood loss or pathological absorptive conditions even in individuals consuming diets of normally adequate iron content. Some orbital porous lesions appear to be of infectious origin; in sickle-cell anemia the orbital cases occur at 4–5 years of age.

Diagnosis of occupationally related paleopathology: Can it be done?

Ann Stirland

The occupations followed by people in the past and the activities in which they most widely indulged have probably not changed greatly until recently. The development of modern technology has brought changes in occupations, tool types and usages that may in time produce characteristic skeletal variations. There are already reports in the clinical literature relating such changes to occupation (Mintz and Fraga 1973). In the past, activity-related pathology may have been expressed as stress fractures of the tibia in hunter-gatherer groups which "ran down" their large prey. No matter how good the preservation and recovery of artifacts from an archeological site, however, the interpretation of their use and the activities involved relies heavily on assumptions based on ethnographic and historical parallels. Such interpretation is speculative.

It is a truism that an archeological site usually has no documentary records. The occupations and activities of its population are, therefore, unknown except from their artifacts. Paleopathological diagnosis brings its own problems. Human bone has only two responses to any insult: either normal bone is lost or new bone is added. Such limited responses lead to difficulties in equating specific pathological lesions with particular occupations. Even in a modern clinical context diagnosis is an inexact science, and many of the relationships between occupation and pathology are still not clearly understood. Much occupational pathology will be confined to soft tissue and will leave no record on the skeleton. There is also the problem of distinguishing lesions which are attributable to a direct traumatic event, age degeneration, or developmental defects from those specifically related to occupation.

Paleopathologists have only recently attempted to relate some pathology to occupation, and they are constantly made aware of the problems and limitations of such interpretation of the material. There are, however, some positive aspects. Unlike the clinician, the paleopathologist is working with the whole dry skeleton. Changes in bone are seen in their very early stages and conditions which may be clinically symptomless can be detected.

Occupationally related paleopathology is, by definition, nonrandom and habitual. If, despite the problems, some occupations can be diagnosed then this will provide an important tool in archeological reconstruction. The ability to identify some skills, trades or professions can lead to the extrapolation of this evidence into other sites and periods, making a further valuable contribution to archeology.

Skeletal studies

The only comprehensive skeletal study of activity-induced pathology so far produced is that by Merbs (1983). Although the Sadlermiut series analyzed was not particularly large, consisting of 41 male and 50 female adult skeletons, the group was thought to meet many of the necessary criteria for such a study (Merbs 1983:4,5). These include a limited number of specialized, but known, activities, good skeletal preservation and recovery, a relatively narrow time span, and both cultural and genetic isolation. The paleopathological lesions in the group were evaluated in six categories: osteoarthritis, osteophytosis, vertebral compression, other degenerative features of the vertebral column (porosity of the articular body surfaces, Schmorl's nodes and laminal spurs), spondylolysis, and anterior tooth loss. The largest category was that of osteoarthritis. Merbs analyzed Sadlermiut activity patterns and discussed their possible stresses on areas of the skeleton (1983:147–156). He correlated the patterns of activity and pathology. In his conclusions, Merbs argued that the osteophytosis of the vertebral columns is a normal degenerative condition and a consequence of bipedalism. A number of the other pathological lesions, however, were correlated with particular activities, known or reconstructed. Both sex and side differences were shown to be important.

Of particular interest were some elements of specific activities which had not been anticipated during Merbs's reconstruction of Sadlermiut behavior patterns. These elements were suggested, however, by particular patterns of pathology (Merbs 1983:184). This unexpected result provides an optimistic conclusion to the study and suggests further potential for such reconstruction.

Another activity-related skeletal study by Dutour (1986) postulates enthesopathies as indicators of activities in two Neolithic populations. There are, however, problems with this work. Both groups studied were small, consisting of only 41 skeletons in total. Of these 21 were unsexable. Dutour does not differentiate between enthesis, attachments of muscles, and syndesmoses, attachments of ligaments (1986:224). He calls all his observed lesions enthesopathies although some are in areas of ligamentous and not tendinous insertion. This is particularly important since it concerns his reconstruction of a possible archer (1986:222). Here, the enthesopathies of the right radius have been combined with degenerative changes to the synovial joints at both elbows and with developments of muscle insertions on both humeri. All these changes are not enthesopathies and the synovial joint changes may be age related. Dutour does not appear to take such age-related degeneration into account when discussing the etiology of the lesions. It is clear, however, from the literature that enthesopathies are degenerative in nature and are "common in older individuals" (Resnick and Niwayama 1981:1297). Apart from using such categories as "juvenile," "mature adult" and, in one case, "aged male," no indication of age ranges is given. In attempting to diagnose activity-related changes, the relative ages of the individuals concerned are of importance. When enthesopathies which are not metabolic or inflammatory in origin are thought to be degenerative in nature, then age becomes a key question. However, as the changes concerned may be accelerated by trauma or chronic stress (Resnick and Niwayama 1981:1300) and, therefore, activity, the picture may be more complex than would appear from Dutour's paper. The problem is once again one of specificity.

In a recent paper, Waldron (1987) discussed a site in which there is some documentary evidence of occupation from burial registers. Although this work is only in its early stages it is of great interest. Of the 336 adults so far examined from Christ Church, Spitalfields, 37.2% have osteoarthritis. This occurs most commonly in the spine, but the shoulders and hands are also involved. The hands are of particular interest since Spitalfields was a very important silk-weaving center, and the disease here may be related to occupational factors.

One of the problems associated with the diagnosis of occupationally related pathology is concerned with current terminology. An example is the use of the term "degenerative change." Some specialists use it to denote age-related changes, while others are concerned with the deterioration of joint surfaces resulting in destruction or proliferation of bone. Ortner (1968:141) studied two large skeletal groups in order to classify degenerative change in the humeral elbow. This study uses the term in the latter sense and stresses the importance of handedness, sex, and cultural practices in degenerative change. Ortner (1968:144) also emphasizes the significance of age in the degree of degeneration in a joint, and discusses mechanical stress, anatomy, and heredity as other determining factors.

These skeletal studies, while subject to inherent problems, do attempt to describe possible occupationally related changes and to categorize some of them.

Clinical studies

The traditional clinical postulate has been that in osteoarthritis, particularly of the spine, an occupational component is present. In the public as well as the specialist mind, pathology of joints has often been associated with usage. This is reflected in the popular terminology often associated with overuse syndromes, such as "tennis elbow." However, the modern clinical view of the role of activity in the development of osteoarthritis is, to say the least, ambivalent. The traditional view is represented by studies such as those on coal miners (Lawrence 1955), dockyard workers (Anderson and Duthie 1963), and by some theoretical works (Radin et al. 1972). Other work, however, such as that by Hadler, presents a different view. In his first study, Hadler stresses the anecdotal nature of much of the clinical correlation between activity and some musculoskeletal disease, and points out that the amount of true clinical information is somewhat inadequate. He suggests alternative strategies for clinical studies to gather the necessary information (1977:1023). These studies are implemented in later work on patterns of repetitive, stereotyped usage in the hands of female textile workers (Hadler et al. 1978; Hadler 1980). It is clear that this later work considers degenerative joint disease to be a highly prevalent process of aging throughout the entire population, and not confined to specific groups; repetitive, stereotyped tasks and side are both involved in the degenerative syndrome.

Anderson found in a study of manual workers that, after allowing for age, heavier work significantly increased the likelihood of rheumatic complaints in general and degenerative disc disease in particular, but not of osteoarthritis of the other joints (1974:523). He also concluded that such diseases are likely to develop earlier among heavy manual workers (1974:524). In a later study, Anderson identified the importance of side, age, biochemical changes and their genetic bases, and chronic irritation in the development of osteoarthritis (1984:431). He argued "that the development of degenerative changes can be triggered in those at risk" either by a severe impact which damages the cartilaginous joint lining, or by chronic stress over a long period producing fatigue in a joint. Both posture and mobility were seen to be important in these changes.

Lockshin et al. (1969) surveyed arthritic complaints in more than 1100 men from three mining communities in West Virginia. The main differences between the groups were that in the 60–69 age group, 68.9% of miners had osteoarthritis of the cervical spine grades 2–4, compared with 56.9% of nonminers (1969:24). Unlike similar British work (Lawrence 1955), these authors found no difference in the prevalence of disc degeneration in the lumbar spine between miners and nonminers. This was attributed either to an unexplained higher prevalence in nonminers or to a difference in working environments in the two countries. Similarly, in a Finnish study of lumberjacks, no correlation could be found between the period of heavy work and disc degeneration when the age factor was removed (Sairanen et al. 1981). The authors concluded that various factors seem to be involved in the etiology of disc degeneration, as well as age and mechanical stress. Heredity and autoimmunization may be involved in these changes, making it difficult to assess the role of heavy work. Osteoarthritis was thought to have a multifactorial etiology (Sairanen et al. 1981:27), and ergonomically correct work was found to be very important in its lack of development. Lindberg and Danielsson (1984) were also unable in their study to demonstrate any relationship between occupation and coxarthrosis in shipyard workers involved in heavy labor.

Some of the preceding papers, and much of the fundamental work in this field, are excellently reviewed by Hagberg (1984). In this review, the controversial nature of the etiology of the degenerative arthropathies, the enthesopathies, and their relationship to occupational stress are clearly discussed and evaluated in the context of disorders of the neck and shoulder (1984:270–275). This work emphasizes what must by now be clear. The role of occupational stress as a factor in osteoarthritis is far from unequivocal. This is partly a function of the classification of work by occupation (e.g., "miners"), rather than by actual task or evaluation of stress, or by loading on skeletal areas. The necessary information is also spread over many different specialized medical fields and its interpretation can suffer from the constraints of differing opinions.

The changes due to osteoarthritis or to degenerative joint disease are not the only ones that may be related to activity in the skeleton. Lesions may be present whose pathogenesis is clear but in a different context. An example is the group of enthesopathies. In this case, the lesion may be degenerative (related to age) in one context, but occur in a young, robust individual in another. If direct trauma or disease can be eliminated as a causative agent, then another explanation has to be found for the lesions.

Other morphological variants used as occupational stress markers are supinator crests and fossae of ulnae (Kennedy 1983). The hypertrophy of the crests, deepening of the fossae, and "ridging" of the insertion of the anconeus muscle were found in prehistoric samples in males "known" to have used missile weapons such as spears (Kennedy 1983:872). Similar changes also occurred in modern populations, in both sexes, who were habitually engaged in occupational or athletic activities involving similar patterns of arm movement. Changes at the elbow which can be directly related to occupation are also apparent in baseball players (Bennett 1959). Damage to hyaline cartilage, olecranon fractures, and spur formation occur as a result of persistent, chronic strain. Chronic bursitis and fraying of both supraspinatus and biceps tendons are recorded as a result of strain and overuse.

The previous examples resulted in the formation of proliferative new bone at various sites in the upper girdle. Another syndrome associated with activity-related stress is osteolysis of the distal clavicle (Kaplan and Resnick 1986). This pathology is known to occur clinically after acute traumatic injury to the shoulder. In the cases cited here, it was also found to occur secondary to repeated microtrauma of the acromioclavicular joint. The main case is of a 39-year-old male who worked in a bakery, lifting heavy pans of rolls in and out of ovens all day. There had been no single traumatic event. Similar, atraumatic pathological changes have been noted in other occupations involving loading, such as air-hammer operator or oxygen tank delivery man, and also in athletes, such as weight-lifters and handball players.

The clinical existence and reporting of pathological changes other than those associated with osteoarthritis in the human skeleton is encouraging. Although the arthropathies are the most common pathological changes observed in archeological skeletal material, other lesions do occur. While some of these are truly pathological and are a consequence of a disease process, others quite clearly are not. It is common to attribute the term "pathological" to anything that is abnormal. Such abnormalities include some of the changes already discussed, such as the enthesopathies, and those due to overuse or activity. Can such abnormal changes be used in the diagnosis of occupationally related paleopathology?

The *Mary Rose*

One of the more serious problems encountered in the analysis and interpretation of archeological human skeletal material is the lack of spatial and temporal controls for a particular site. This problem is part of the wider one already referred to, namely, the general lack of any documentary evidence. Very rarely is a site excavated in which these problems are minimized. Such a site is Henry VIII's flagship, the *Mary Rose*.

The *Mary Rose* was sunk on the morning of 19 July 1545 after having emerged from her home port of Portsmouth, England, at the head of the English fleet. The object was to engage the French fleet moored nearby. The ship apparently executed a bad turn to starboard and, in attempting to raise sail, took in water through open gun ports, heeled over and sank rapidly, settling heavily into the soft seabed silts. Of the crew of 415 men all but about 35 drowned, including the captain, the master, and the vice admiral. Most were trapped under the stout antiboarding netting which covered the decks. The ship came to rest on her starboard side and silted up within a matter of months. The exposed port side was eroded by the sea until it collapsed, leaving intact the complete starboard half. The wreck was then sealed in the 16th century by a hard, shelly seabed and remained hidden, apart from the occasional severe winter storm, until it was discovered in 1968. The rapid silting and sealing provided a perfect anaerobic environment in which much organic material, including the human skeletal remains, was exceptionally well preserved. The position and condition of the wreck led to a commingling of the skeletal material both within individual deck sectors and probably, in some cases, from one deck to another. An account of the finding, excavation, and raising of the *Mary Rose* may be found in Rule (1982,1983).

Henry VIII had a list of his ships compiled by Anthony Anthony and this was completed in 1546 (Rule 1982:26–28). This list contains the only contemporary picture of the *Mary Rose* and includes crew numbers and their occupations (Rule 1982:27). It also includes lists of all the ordinance and equipment for the war. In the case of the men of the *Mary Rose*, therefore, the actual date and cause of death is known as is their equipment and occupations. This sample, like Merbs's Sadlermiut, meets many of the criteria for the study of activity- or occupationally related pathology. There is, however, one problem that Merbs did not have; it is a very commingled group. Because of the unique nature of this sample, its importance and the possibility of undertaking such a study, it was decided to try to re-sort the bones where possible into individuals. This was undertaken initially for the purposes of the bone report submitted in 1985. Because of pressure of time (money), the re-sorting was accomplished only within the deck sectors and not attempted from one deck to another. A skull and mandible count produced a minimum number of 179 individuals; the re-sorting generated 91 fairly complete skeletons, some more complete than others.

Experience suggests that an archeological sample of human remains generally displays a fairly common collection of pathological lesions. Many of these lesions are expressed in joint changes as arthropathies. Apart from the arthropathies, healed fractures are commonly seen as in nonspecific periostitis and, in the New World at least, porotic hyperostosis and cribra orbitalia. In some specific groups and time periods, evidence for tuberculosis and for leprosy can be identified. Other lesions occur generally much less commonly in such material, although they may have a more widespread clinical expression. They include such examples as osteochondritis dissecans, Osgood-Schlatter's and Scheuermann's diseases. There are other conditions, such as os acromiale, which are very rare and have a low expression in any group, and there are morphological changes, such as the enthesopathies, which have a much higher frequency in some groups than in others. In the sample from the *Mary Rose*, we are concerned with the last three groups rather than the more common ones.

Predictably for a fighting ship, most of the men of the *Mary Rose* were young and some of them were very young. There was a predominance of individuals ranging from 15 to 25 years with a smaller group in their late twenties/early thirties. There was also a very small number of both very young (10–15) and older (35–45+) men. Before discussing the patterns of pathology, it must be pointed out that, owing to the problems of mixing, pathology in this group has so far been assessed by bone, rather than by individual. It is accepted that, for the arthropathies at least, this situation is undesirable (Rogers et al. 1987), and discussion on arthritic changes has, therefore, been omitted.

The area of the skeleton displaying the most pathology is the vertebral column. There are nine cases of spondylolysis, including one of spondylolisthesis but, apart from these, most of the lesions are in the thoracic segment. The most common of these lesions is Schmorl's nodes, and they occur in some subpubescent spines where the centra are still very billowed and the epiphyseal ring is unfused. Such cartilaginous nodes have a varied etiology (Resnick and Niwayama 1981:1404). If they are not degenerative or related to various diseases, however, they are often a consequence of trauma. One of the diseases in which Schmorl's nodes occurs is Scheuermann's disease. This condition also has a high frequency in the sample, in the thoracic spine. As it is a juvenile condition, it occurs in very young spines, although the evidence is also retained in older individuals. Much of the modern work on Scheuermann's disease suggests that it is probably a manifestation of the node formation and a result of the failure of the cartilaginous end plate under stress which can be traumatically induced. In the sample there are also marked changes in the shape of the centra in the thoracic spine, producing an expansion, often in an anterior direction. Thoracic scoliosis is also apparent, as is twisting of some apophyseal joints, particularly at the lower end where there is also some gross enlargement of such joints. This is particularly true in the case of three of the matched, fairly complete skeletons. They are all young, in their early to mid-twenties, and they all exhibit a great deal of stress to the thoracic and lumbar areas. One in particular has extraordinary vertebrae. All are large and robust with huge apophyseal joints, particularly T10–12. Both T11 and T12 look like lumbar vertebrae, with characteristic curved articulations (Figure 1), and the sacrum and innominates are also very large with deep articulations. All three of these skeletons were found on the Main Gun deck in close association with one of the very heavy bronze cannon. These cannons weighed up to two tons and were operated entirely by hand by a gun crew, being hauled in and out of the gun port on a wooden carriage. The ball shot used was also very heavy and had to be fetched in baskets from the Orlop deck below.

Two other conditions which are a consequence of trauma resulting in damage to an epiphysis are osteochondritis dissecans and Osgood-Schlatter's disease. Both are more frequent clinically in the young and both affect boys more than girls. Neither are seen commonly in archeological samples. In the sample from the *Mary Rose*, there are high frequencies of both. The most common site for osteochondritis dissecans is the first metatarso-phalangeal joint. There are 23 affected bones: 16 examples occur on the distal humeral condyles and 7 on the distal femoral condyles, some bilaterally (Figure 2). There are six cases, two left and four right, of unusual pits occurring in femoral heads, superior to the fovea (Figure 3).

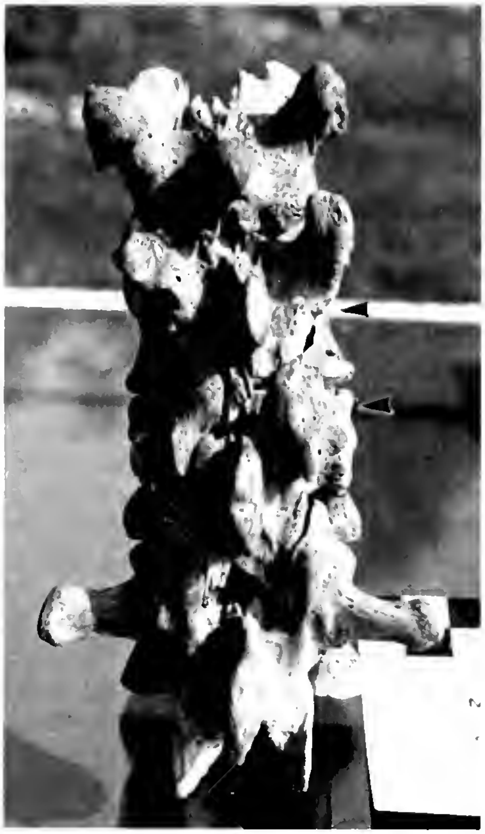


FIGURE 1. Gross enlargement of lower thoracic apophyseal joints *(arrows)*. (All specimens illustrated in this paper are courtesy of the *Mary Rose* Trust.)

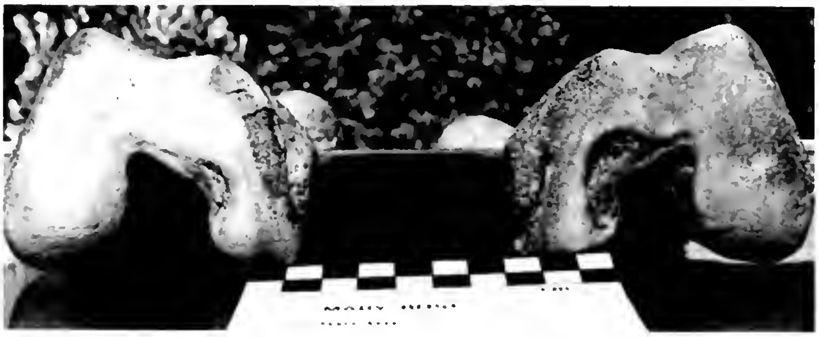


FIGURE 2. Bilateral osteochondritis dissecans.

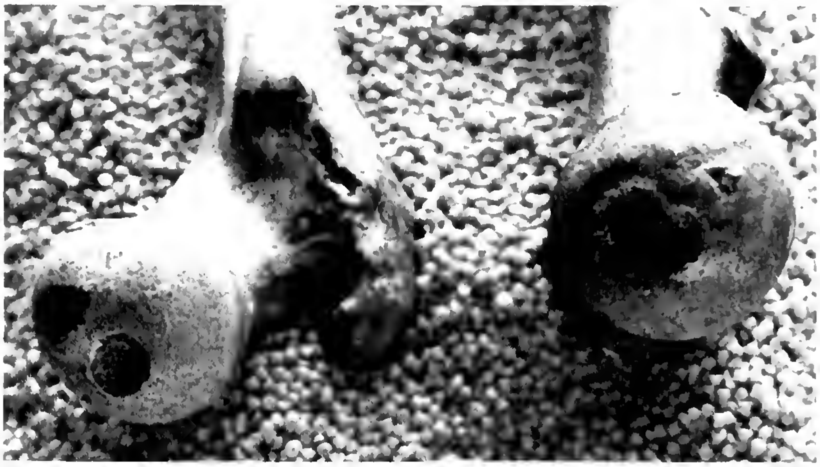


FIGURE 3. Pits in femoral heads.

These have also been identified as osteochondritis dissecans (I. Watt 1984, pers. comm.), although an alternative diagnosis of avascular necrosis has been suggested (D. Birkett 1986, pers. comm.). Eleven left and 12 right tibiae exhibit Osgood-Schlatter's disease. Some are very young with newly fused epiphysis and in some cases it is bilateral (Figure 4).

Os acromiale is a rare anomaly which has already been described for this group (Stirland 1987). The data in this earlier paper have now been updated and the frequency is 12.5%. With a normal frequency of from 2% to 6%, the values for this sample are high. It is argued that os acromiale in this case may be related to long-term use of the very heavy longbows by the professional archers on the ship. The persistent use of this weapon, with its draw weight of about 57 kg (125 lb), from a very early age was responsible for long-term shearing stresses on the acromion which inhibited fusion of the final element. The inflammatory nature of the unfused elements suggests the surfaces were subjected to such stresses in these cases (Figure 5).

A defect in the rim of the acetabulum, usually in the posterior portion, has been called an acetabular flange lesion (Knowles 1983). In the absence of other serious pathology of the acetabulum or the femoral head its etiology is unclear. It has been suggested that it is produced by a "transient, incomplete, upward dislocation of the femoral head" (Knowles 1983:65), which does not affect the femur. Eleven innominates, four left and seven right, from this group are affected by this lesion (Figure 6). Another suggestion offered is that it is a defect in the fusion of the pelvic elements (I. Watt 1986, pers. comm.). The appearance of these particular innominates, however, where there is pitting of the affected rim, suggests a traumatic origin associated with activity but not serious enough to be permanently disabling.

Enthesopathies are widespread in the burials from the *Mary Rose*. These range from development of the linea aspera, gluteal ridge, and hypotrochanteric fossa on the posterior femur (Figure 7) to lesions at the insertions of pectoralis and teres major on the humerus and of biceps on the radius (Figure 8). There are many other examples and all are widespread throughout the sample. Enthesopathies also occur in the form of spurring, especially of the trochanters, calcaneus and, to a lesser extent, the olecranon process of the ulna. Some syndesmoses, particularly of the costoclavicular ligament, show similar changes (Figure 9), with lesions and some bony buildup. The exuberant nature of these changes, which far exceed those normally seen in archeological material, must be explained in terms of processes.

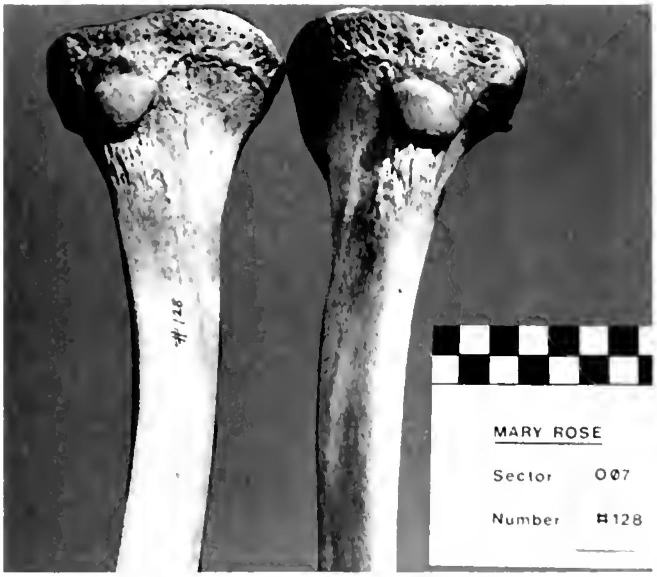


FIGURE 4. Bilateral Osgood-Schlatter's disease.

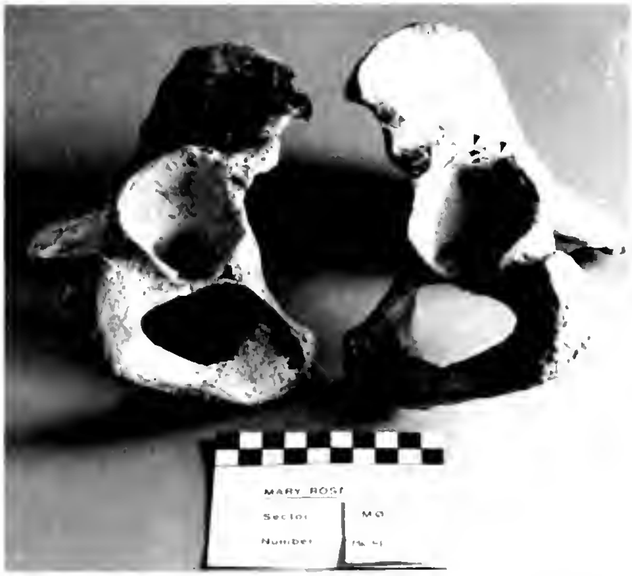


FIGURE 6. Acetabular flange lesion of left innominate *(arrows)*.

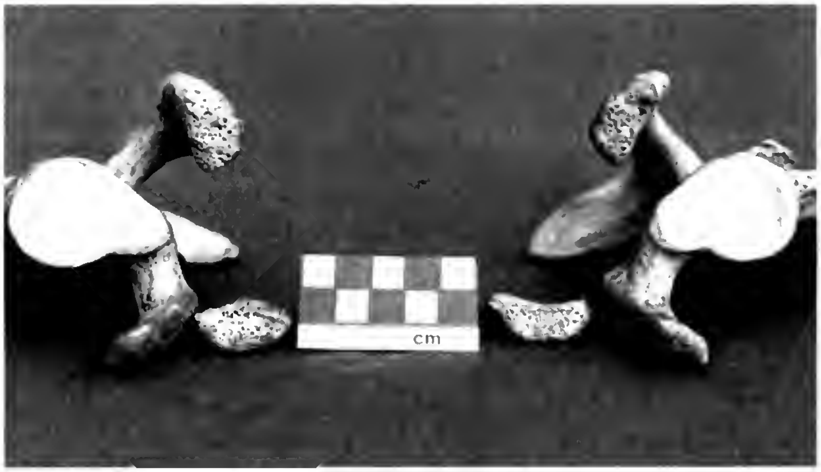


FIGURE 5. Bilateral os acromiale.

The rigorous physical demands of their working environment must have put heavy stresses on the developing skeletons of these young men. The mariners were working with four masts, all of which bore canvas and required servicing. The ship had a small keel and very little ballast and was, therefore, unstable at the best of times. Men and boys must have fallen and slipped while trying to release or furl sails. The loading stresses associated with the operation of the guns and with the longbows has already been discussed. The amount and type of pathology and the degree of bony remodeling of various fibrous insertions in this sample has to be explained in terms of their environment. These would appear to be changes due to occupation in some members of the crew of the *Mary Rose*. Some of these are pathological and some morphological. The former may be due to work-related trauma, while the latter appear to be related to loading stresses. All, therefore, may well be a consequence of occupation. The relationships between these changes and specific occupations is, however, another matter.

Conclusions

The concept of relating specific skeletal changes to particular activities is a comparatively recent one. As has been shown, clinical work is controversial and paleopathology is faced with problems associated with the nature of the material and the lack of proper controls. Some samples, however, are able to overcome these problems to a greater or lesser extent and these should be utilized to implement further studies. An example is the work of Merbs already cited. The sample from the *Mary Rose* would appear to be another.

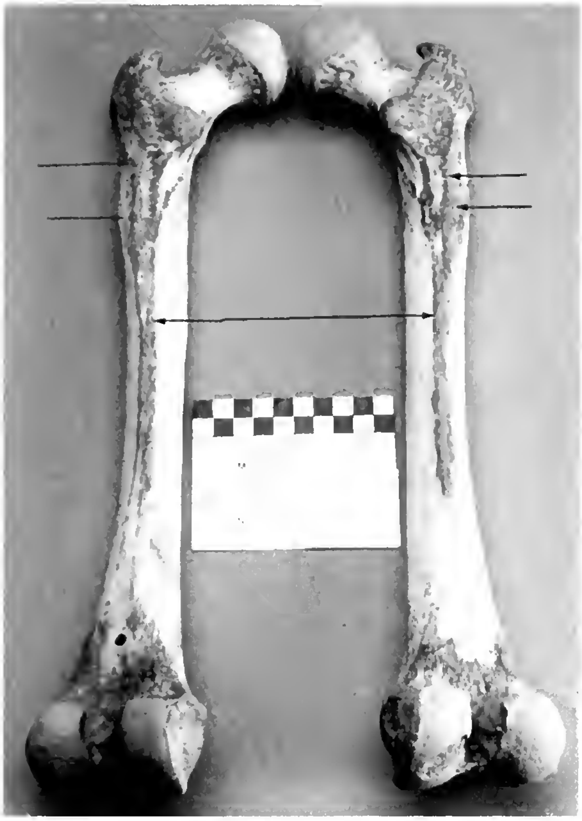


FIGURE 7. Development of linea aspera, gluteal ridge, and hypotrochantic fossa *(arrows)*.

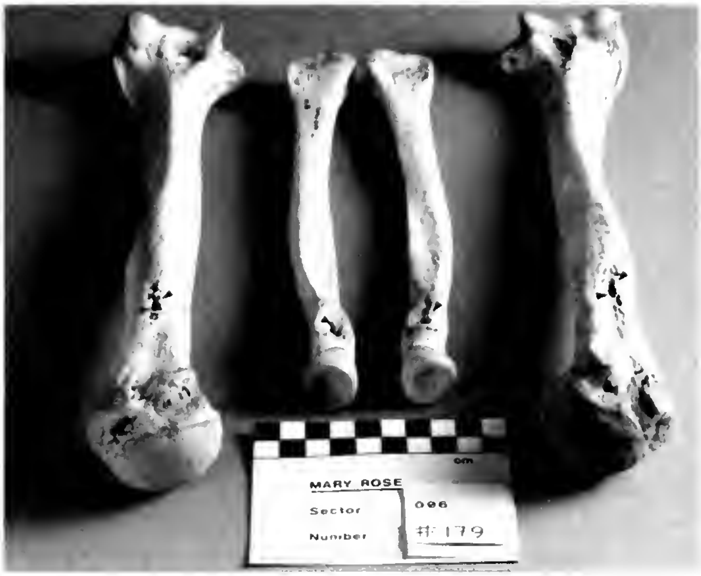


FIGURE 8. Enthesopathies of humerus and radius *(arrows)*.

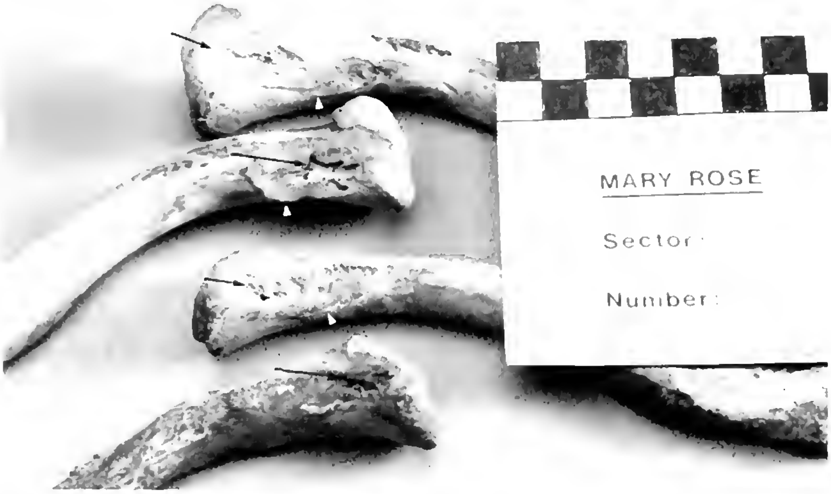


FIGURE 9. Lesions of clavicular syndesmoses *(arrows)*.

Although such work is difficult, some contribution may be made. In an archeological sample, it will never be possible to extrapolate from the general to the particular and assign an individual's occupation from a group study. In a personal sense, I will never be able to say: "This man was an archer." What needs to be done, however, is to compare specific groups. Such comparison must obviously be as rigorous as possible, so that groups from the same time period and/or geographical area should be compared. What is needed is similarity studies (Waldron 1987, pers. comm.). In this way, groups could be compared for similarities and differences. When all other factors, such as age, sex, and side, are equal, then differences, such as have been described for the *Mary Rose* crew, can be related to a group activity or occupation.

With regard to the *Mary Rose*, a research program has been undertaken which will implement such group studies. Other large medieval groups from southern England will be compared with that sample and the differences will be evaluated. Perhaps then it will be possible to say with more confidence that, all else being equal, the sample from the *Mary Rose* is different and these differences are due to the occupations followed by the crew.

Literature cited

Anderson, J.A.D. 1974. Occupation as a Modifying Factor in the Diagnosis and Treatment of Rheumatic Diseases. *Current Medical Research and Opinion*, 2(9).

———. 1984. Arthrosis and its Relation to Work. *Scandinavian Journal of Work Health*, 10:429–433.

Anderson, J.A.D., and J.J.R. Duthie. 1963. Rheumatic Complaints in Dockyard Workers. *Annals of the Rheumatic Diseases*, 22:401–409.

Bennett, G.E. 1959. Elbow and Shoulder Lesions of Baseball Players. *American Journal of Surgery*, 98.

Dutour, O. 1986. Enthesopathies (Lesions of Muscular Insertions) as Indicators of the Activities of Neolithic Saharan Populations. *American Journal of Physical Anthropology*, 71:221–224.

Hadler, N.M. 1977. Industrial Rheumatology: Clinical Investigations into the Influence of the Pattern of Usage on the Pattern of Regional Musculoskeletal Disease. *Arthritis and Rheumatism*, 20(4).

———. 1980. The Variable of Usage in the Epidemiology of Osteoarthrosis. In J.G. Peyron, ed., *Epidemiology of Osteoarthritis*. Symposium Paris, Juin.

Hadler, N.M., D.B. Gillings, H.R. Imbus, P.M. Levitin, D. Makuc, P.D. Utsinger, W.J. Yount, D. Slusser, and N. Moskovitz. 1978. Hand Structure and Function in an Industrial Setting. *Arthritis and Rheumatism*, 21(2).

Hagberg, M. 1984. Occupational Musculoskeletal Stress and Disorders of the Neck and Shoulder: A Review of Possible Pathophysiology. *International Archives of Occupational and Environmental Health*, 53:269–278.

Kaplan, P.A., and D. Resnick. 1986. Stress-Induced Osteolysis of the Clavicle. *Radiology*, 158:139–140.

Kennedy, K.A.R. 1983. Morphological Variations in Ulnar Supinator Crests and Fossae as Identifying Markers of Occupational Stress. *Journal of Forensic Sciences*, 28:871–876.

Knowles, A.K. 1983. Acute Traumatic Lesions. In G.D. Hart, ed., *Disease in Ancient Man*, 65. Toronto: Clarke Irwin.

Lawrence, J.S. 1955. Rheumatism in Coal Miners Part III: Occupational Factors. *British Journal of Industrial Medicine*, 12:249–261.

Lindberg, H., and L.G. Danielsson. 1984. The Relation between Labor and Coxarthrosis. *Clinical Orthopaedics and Related Research*, 19(1).

Lockshin, M.D., I.T.T. Higgins, M.W. Higgins, H.J. Dodge, and N. Canale. 1969. Rheumatism in Mining Communities in Marion County, West Virginia. *American Journal of Epidemiology*, 90(1).

Merbs, C.F. 1983. *Patterns of Activity-Induced Pathology in a Canadian Inuit Population*. Ottawa: National Museums of Canada.

Mintz, G., and A. Fraga. 1973. Severe Osteoarthritis of the Elbow in Foundry Workers. *Archives of Environmental Health*, 27:78–80.

Ortner, D.J. 1968. Description and Classification of Degenerative Bone Changes in the Distal Joint Surfaces of the Humerus. *American Journal of Physical Anthropology*, 28:139–156.

Radin, E.L., I.L. Paul, and R.M. Rose. 1972. Hypothesis: Role of Mechanical Factors in the Pathogenesis of Primary Osteoarthritis. *Lancet*, 1:519–521.

Resnick, D., and G. Niwayama. 1981. *Diagnosis of Bone and Joint Disorders*, vol. 2. Philadelphia: W.B. Saunders.

Rogers, J., T. Waldron, P. Dieppe, and I. Watt. 1987. Arthropathies in Paleopathology: The Basis of Classification According to Most Probable Cause. *Journal of Archeological Science*, 14:179–193.

Rule, M. 1982. *The Mary Rose: The Excavation and Raising of Henry VIII's Flagship*. Leicester, U.K.: Windward Press.

———. 1983. Henry VIII's Lost Warship. *National Geographic*, 163:646–675.

Sairanen, E., L. Brushaber, and M. Kaskinen. 1981. Felling Work, Low Back Pain and Osteoarthritis. *Scandinavian Journal of Work and Environmental Health*, 7:18–30.

Stirland, A. 1987. A Possible Correlation between Os Acromiale and Occupation in the Burials from the Mary Rose. *Proceedings of the 5th European Meeting of the Paleopathology Association*, 327–334. Siena, Italy, 1984.

Waldron, T. 1987. Osteoarthritis at Christ Church, Spitalfields: An Interim Report. Paper presented at the 14th annual meeting of the Paleopathology Association, April 1–2. New York.

SUMMARY OF AUDIENCE DISCUSSION: Discussion quickly made it clear that the audience was divided on the question of whether the lesions demonstrated as osteochondritis dissecans were of metabolic, traumatic, or even developmental nature. Several felt they had seen similar lesions under circumstances making it reasonable to relate them to occupations resulting from prolonged and repetitive minor trauma such as a recent (19th century) military, young adult group under heavy stress. By law Renaissance youths initiated longbow training (which placed a 300-pound pressure on each shoulder) at age of six years. It is conceivable that application of such stress to a growing bone may induce lesions to which a mature bone would be resistant. All agreed an investigational, radiologically based study on modern individuals with known, selected occupations could make a major contribution to the identification of osteologic lesions useful for prediction of handedness and occupations.

Methods

Recovery of bone and serum proteins from human skeletal tissue: IgG, osteonectin, and albumin

Noreen Tuross

The most common substrates from which to infer or deduce vertebrate paleopathology are bones and teeth. Physical anthropologists have explored the significant contribution that morphology and histology of mineralized tissues can make to our understanding of paleopathology. This study describes information at the molecular level that remains in the bones and teeth of some of our ancestors.

The presence of amino acids in fossil bones and teeth was the first evidence that proteins indigenous to the animal might remain in the mineralized tissues (Abelson 1956; Ho 1965). The preservation of the amino acids from the major bone protein, collagen, was documented in a variety of fossil bones from many locations (Ho 1965; Wyckoff and Doberenz 1965; Tuross and Hare 1978). In fossil bones much of the collagen exists as a heterogeneous mixture of degradation products relative to the original gene product (Tuross et al. 1980). Isotopically, however, the degraded collagen is assumed to retain the pertinent information for archeometric use. Radiocarbon from collagen has been used to date fossils (Libby 1955; Taylor 1987) and the stable isotopes of carbon and nitrogen from bone collagen have been used in paleodietary interpretations (Schoeninger et al. 1983; Schoeninger and DeNiro 1984). Degradation of collagen, particularly the relative loss of the amino acid, glycine, can perturb the carbon and nitrogen stable isotope values obtained from fossil bones and teeth (Tuross et al. 1988). Better methods of isolation and characterization of proteins from fossil bones and teeth will contribute to the accuracy of archeometric isotopic applications.

Many proteins other than collagen can be found in modern bone. Both serum-derived and bone-cell-produced proteins can be extracted from bone. The complexity of the mixture of proteins found in bone can be seen in a two-dimensional gel electrophoresis analysis of bovine bone where approximately 200 separate proteins were observed (Delmas et al. 1984). Extraction techniques developed by John Termine and coworkers allow for the mineral and nonmineral associated proteins to be solubilized (Termine et al. 1981). Five proteins from the mineral compartment of developing human bone have been purified and partially characterized (Fisher et al. 1987).

Of greatest potential interest to the paleopathologist is the preservation of serum-derived proteins in bone. Two serum proteins, albumin and alpha–2-HS, concentrate in bone and make up 13% of the noncollagenous bone matrix proteins extracted from fetal human subperiosteal bone (Robey et al. 1988). Smaller amounts of many other serum-derived proteins, including transferrin and the immunoglobulins (IgG, IgA, IgM and IgE), can be isolated from modern bone.

Noncollagenous proteins, osteonectin, albumin, IgG, and transferrin, have been identified at their original molecular weight in several individuals from the Windover archeological site in Florida. This 7000-year-old site, excavated under the direction of Glen Doran, yielded in excess of 150 human skeletons. The preservation of the mineralized tissue from the Windover site was variable. Generally, however, the neutral peat environment provided an anoxic, reducing atmosphere that was conducive to protein preservation in these bones.

This study examines the preservation of noncollagenous proteins—osteonectin, IgG, and albumin—in protein extracts from human skeletal material excavated at the Sully and Mobridge sites in South Dakota. These cemeteries were excavated in the 1920s and the 1950s, and the collections reside in the National Museum of Natural History, Smithsonian Institution. Associated materials at these sites date the skeletal remains at two to three hundred years of age. These skeletons are a classic museum collection, and provide the opportunity to assess the preservation of protein in bones that have been disinterred for 30 to 50 years.

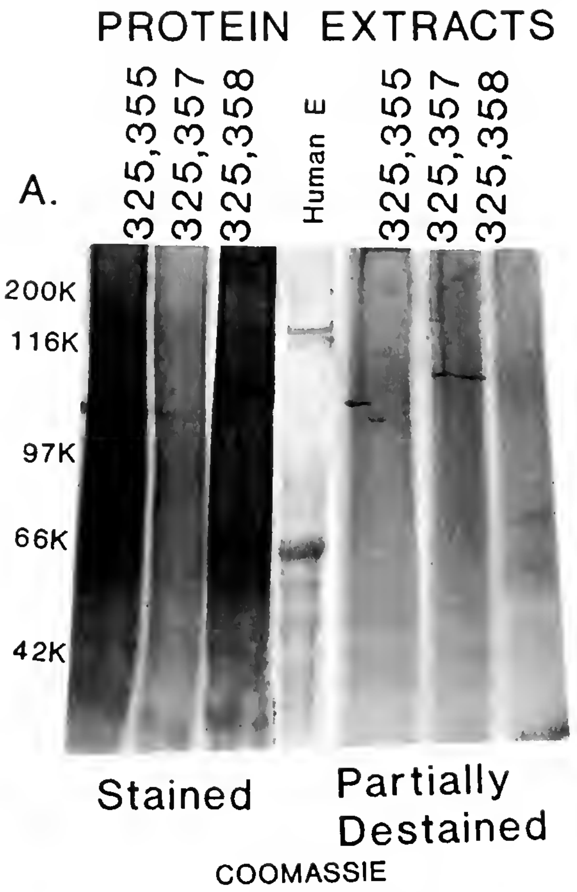


FIGURE 1. Protein extracts from three Mobridge site individuals on an SDS 4–20% polyacrylamide gel stained with Coomassie Brilliant Blue. Fully stained extracts are a smear of collagen degradation products over the entire molecular weight range of the gel. Equivalent extracts, partially destained in methanol/acetic acid/water, have discrete bands approximately 70 Ka and below. Multiple bands are apparent in each sample. Sharp, highly stained gashes above 97 Ka region are due to large amounts of protein (500 μg) applied to top of each lane.

Materials and methods

Thirteen rib fragments, weighing from 2 to 5 g, were taken from individuals (catalog numbers 325348, 325352–325358, 381159, 381163, 381193, 381342 and 381345, 381346) and extracted in 4M guanidine HCl/0.5M EDTA at 4°C for two days. This procedure partially demineralized the samples. The guanidine/EDTA was removed from the soluble protein by desalting 35 ml of the solution over a P6DG column. The proteins were monitored at 254 nm, eluted in 100 mM ammonium acetate, and lyophilized.

Fractions of the desalted protein were concentrated by Centricon (Amicon) filtration, and protein above 30,000 molecular weight retained on the filter was electrophoresed. Mini-gradient SDS gels, 4–20% acrylamide (Novex) were used in a traditional Laemmli (Laemmli 1970) buffer system. The gels were stained with Coomassie Blue, and partially destained for up to four days.

Bacterial collagenase (Advanced Biofractures) digestions of up to 1 mg of protein were done at 37°C for 4 to 8 hours. The fossil protein digests were then subjected to Centricon filtration with a membrane that would retain all proteins above 10,000 molecular weight. The filters were extracted with gel sample buffer and gel electrophoresed as described above.

Electroblotting of collagenase digested proteins onto nitrocellulose was performed according to the method of Towbin et al. (1979). Nitrocellulose electrotransfers were processed for immunodetection by using 1:1000 dilution of rabbit antihuman osteonectin and albumin (Cappel Laboratories) and a 1:2000 dilution of peroxidase conjugated goat antirabbit IgG (Kirkegaard and Perry Laboratories) and 4-chloro–1-naphthol color reagent. IgG detection was achieved with a 1:1000 dilution of rabbit antihuman peroxidase conjugated IgG (Capell Laboratories).

Results and discussion

The protein extracts from all bone samples were collagen-like in their amino acid pattern. Gel electrophoresis (Figure 1) of the whole protein extract gave a smear of Coomassie stainable material that ranged in molecular weight from > 200,000 to the 30,000 retained on the Centricon filter. This collagen smear is common in electrophoresed fossil bone extracts (Tuross et al. 1980) and results from multiple peptide bond breaks along the collagen molecule. Partial destaining of these Coomassie stained gels revealed the presence of bands of protein originally obscured in the fully stained gel (Figure 1).

The fully stained fossil protein extracts and the equivalent partially destained extracts are shown in relation to the proteins extracted by guanidine/EDTA in a modern human fetal calvarium. In modern bone, collagen is largely insoluble when subjected to the dissociative, demineralizing conditions of guanidine/EDTA (Termine 1983). In fossil bone, however, the partial breakdown of the collagen molecule renders this protein soluble to the same dissociative demineralizing conditions.

This increased solubility of the collagen degradation products makes the isolation and characterization of any remaining intact noncollagenous proteins or native collagen more difficult. Generally, large amounts of protein (up to 1 mg) from each skeletal sample must be applied to a gel in order to visualize any "bandable" protein upon partial destaining of a gel. Applying a large amount of protein to a gel can lead to several type of distortions, including the short, sharp, gashlike disconformities seen at the top of several lines in Figure 1.

Allowing an electrophoresed total protein extract to partially destain after Coomassie Blue staining produced discrete bands of protein in 12 out of the 13 skeletal fragments analyzed. The number and the molecular weight of these bands varied among bone fragments, but generally, the most common band observed was at approximately 68 Ka. Several bands were also observed at 60 Ka, 45 Ka, and 42 Ka. The ability to see bands of protein as the collagen degradation products destain is due to the fact that collagen is a relatively poor Coomassie binder. Therefore different bands could be observed at varying stages of the destaining process, and any protein that binds Coomassie Blue equally or less well than collagen would not be seen at all. Another problem with the extensive collagen degradation products is the interference in transferring the fossil protein to nitrocellulose paper for immunological detection. Because a percentage of the protein moves from the gel to the paper with time, it is difficult to transfer enough of the putative intact protein in a reasonable amount of time.

The total protein extracts were digested with bacterial collagenase. Bacterial collagenase is an enzyme that degrades any string of amino acids, Gly-X-Pro or Hypro, to tripeptides. Since only collagen has repeats of Gly-X-Pro or Hypro, this is the only protein that will be affected by the collagenase treatment. All of the skeletal fragments produced protein that was partially degradable by bacterial collagenase, and all digested fossil bone protein extracts exhibited at least one Coomassie stainable band upon postdigestion electrophoresis (Figure 2).

The ability to digest the background smear on the gels with bacterial collagenase is proof that the staining was caused by collagen breakdown products. The existence of Coomassie stainable bands after collagenase treatment is proof of the preservation of several noncollagenous proteins in these fossil bones.

While preliminary identification of these bands can be made based on their molecular weight, immunological identification by reacting the transferred bands to purified antibodies is the basic criterion upon which protein identification should be made. This immunodetection technique (commonly called Western blotting) requires that one and only one band at the appropriate molecular weight recognize the antibody being used.

In preliminary analysis of the collagenase digested electrotransfers, the proteins albumin, osteonectin, and IgG were identified from these skeletal remains. Albumin was by far the most prevalent among the preserved noncollagenous proteins in the samples. The identification of IgG in some of the collagenase extracts demonstrates the ability to detect intact proteins from fossil extracts even in the absence of a clear Coomassie stained band on the gel.

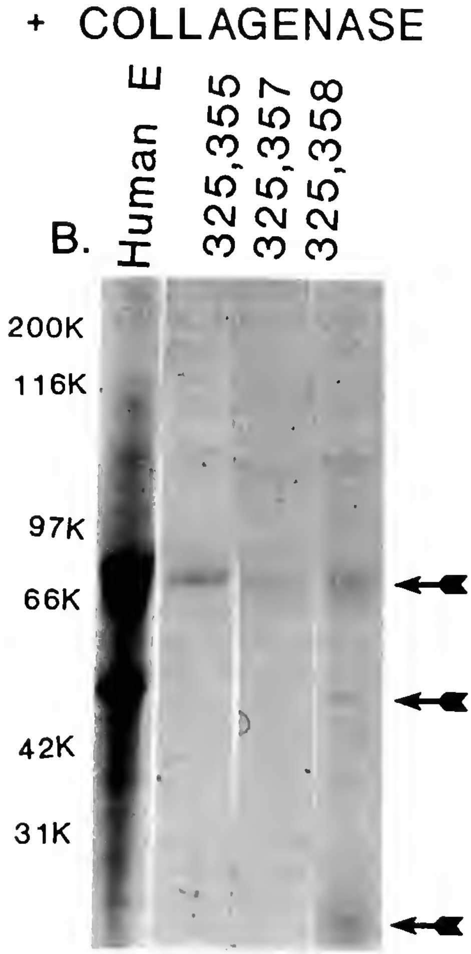


Figure 2. Bacterial collagenase digestion of bone protein from three Mobridge site individuals on an SDS 4–20% polyacrylamide gel stained with Coomassie Brilliant Blue. This is a fully stained (Coomassie) gel of protein bands remaining after bacterial collagenase digestion of 1 mg of bone protein extract. The smear of degradation products from Figure 1 is removed, and intact, collagenase-resistant proteins can be seen. A band just above the 66 Ka marker was positive to the rabbit antihuman albumin antibody upon electrotransfer, and two of the three individuals (325357 and 325358) had bands that recognized rabbit antihuman osteonectin antibody.

The demonstration of preservation of noncollagenous proteins in fossil bones that have been washed, treated and stored in a museum collection is an important addition to the growing evidence that fossil mineralized tissue contains molecular information of importance to the paleopathologist. Digestion of fossil bone protein extracts with bacterial collagenase will increase our ability to screen these samples for molecular preservation. The preservation of immunoglobulins is particularly intriguing, and opens up the possibility that an independent record of disease states remains in the bones of many individuals.

Literature cited

Abelson, P.H. 1956. Paleobiochemistry. *Scientific American*, 195:83–92.

Delmas, P.D., R.P. Tracy, B.L. Riggs, and K.G. Mann. 1984. Identification of Noncollagenous Proteins of Bovine Bone by Two-Dimensional Gel Electrophoresis. *Calcified Tissue International*, 36:308–316.

Fisher, L.W., G.R. Hawkins, N. Tuross, and J.D. Termine. 1987. Purification and Partial Characterization of Small Proteoglycans I and II, Bone Sialoproteins I and II, and Osteonectin from the Mineral Compartment of Developing Human Bone. *Journal of Biological Chemistry*, 262:9702–9708.

Ho, T.Y. 1965. Amino Acid Composition of Bone and Tooth Proteins in Late Pleistocene Mammals. *Proceedings of the National Academy of Science*, 54:26–31.

Laemmli, U.K. 1970. Cleavage of Structural Proteins during the Assembly of the Head of Bacteriophage T4. *Nature*, 227:680–685.

Libby, W. 1955. *Radiocarbon Dating*. 2d edition. Chicago: University of Chicago Press.

Robey, P.G., L.W. Fisher, M.F. Young, and J.D. Termine. 1988. The Biochemistry of Bone. In B.L. Riggs and L.J. Melton, eds., *Osteoporosis: Etiology, Diagnosis and Management*, 95–109. New York: Raven Press.

Schoeninger, M.J., and M.J. DeNiro. 1984. Nitrogen and Carbon Isotopic Composition of Bone Collagen from Marine and Terrestrial Animals. *Geochimica et Cosmochimica Acta*, 48:625–639.

Schoeninger, M.J., M.J. DeNiro, and H. Tauber. 1983. Stable Nitrogen Isotope Ratios of Bone Collagen Reflect Marine and Terrestrial Components of Prehistoric Human Diet. *Science*, 220:1381–1383.

Taylor, R.E. 1987. *Radiocarbon Dating: An Archaeological Perspective*. Orlando, Fla.: Academic Press.

Termine, J.D. 1983. Osteonectin and Other Newly Described Proteins of Developing Bone. In W.A. Peck, ed., *Bone and Mineral Research*, 144–156. Amsterdam: Excerpta Medica.

Termine, J.D., A.B. Belcourt, K.M. Conn, and H.K. Kleinman. 1981. Mineral and Collagen-Binding Proteins of Fetal Calf Bone. *Journal of Biological Chemistry*, 256(20):10403–10408.

Towbin, H., T. Staehilin, and J. Gordon. 1979. Electrophoretic Transfer of Proteins from Polyacrylamide Gel to Nitrocellulose Sheets: Procedure and Some Applications. *Proceedings of the National Academy of Science*, 76:4350–4354.

Tuross, N., D.R. Eyre, M.E. Holtrop, M.J. Glimcher, and P.E. Hare. 1980. Collagen in Fossil Bones. In P.E. Hare, ed., *Biogeochemistry of Amino Acids*, 53–63. New York: Wiley and Sons.

Tuross, N., M. Fogel, and P.E. Hare. 1988. Variability in the Preservation of the Isotopic Composition of Collagen from Fossil Bone. *Geochimica et Cosmochimica Acta*, 52:929–935.

Tuross, N., and P.E. Hare. 1978. Collagen in Fossil Bone. *Carnegie Institution of Washington Yearbook*, 77:891.

Wyckoff, R.W.G., and A.R. Doberenz. 1965. Electron Microscopy of Rancho La Brea Bone. *Proceedings of the National Academy of Science*, 53:230–233.

SUMMARY OF AUDIENCE DISCUSSION: The ability to identify in archeological human remains the serum antibodies present at the time of death would provide access to an incredibly valuable legacy of the history of specific infectious diseases suffered by an ancient population. However, it must be emphasized that demonstration of the presence of a major molecular IgG fraction does not guarantee that the variable end of the light chain is preserved well enough to reveal its immunologic specificity, which is necessary for identification of the specific infectious agent against which the antibody is directed. Unfortunately the next analytical step in pursuit of that goal involves the use of chemicals strong enough by themselves to alter even the preserved protein. Recent studies on an excavated, 150-year-old seaman of the mid-19th century Franklin expedition, buried deep in North American arctic permafrost, demonstrated the research potential of cryopreserved bodies, but the surface arctic summer thawing conditions frustrate such efforts in most instances. Ancient tissues usually contain a host of polypeptides secondary to partially degraded protein, and the potential of these to react with immunological diagnostic reagents is untested. For this reason it appears desirable to include more controls than usual when applying such immunodiagnostic methods to archeological remains, and to be meticulously cautious in interpretation of their results. Investigations leading to diagnostic security in the use of immunological methods would be a major contribution to paleopathology. A recent Oxford conference presentation suggested the presence of protein within the hydroxyapatite crystals of fossil bone. The potential value of recovering intact protein there justifies serious research pursuit of that observation.

Bone histology and paleopathology: Methodological considerations

Debra L. Martin

In the last hundred years, identification and analysis of disease processes have dramatically increased in the area of paleopathology. However, prior to the last ten years, analysis had remained primarily descriptive with the aim to identify disease in space and time. Recent emphasis on the interactions between biology and culture in the disease process has proven to be fruitful, yielding information concerning human adaptability within an evolutionary framework. A further expansion of the biocultural approach involves using skeletal material as an aid in elucidating processes and bone biodynamics in health and disease states.

Given the interest in skeletal growth, pathology, maintenance, and repair, analyses should ideally proceed in a logical and complementary fashion from the gross and macroscopic analysis to the histological and microscopic level. Building on this data base, the biochemical and molecular assays can follow. In this manner, identification of pathologic conditions, patterns of growth and development, changes in gross morphology, and alterations in the rate of morbidity and mortality can form the contextual framework for the other types of analyses. While emphasis on gross morphological features and measurements remains important to the reconstruction of human paleobiology and population success in adjusting to the physical environment, evidence now exists that other skeletal parameters (such as histology) can offer valuable information on health status prior to death (Martin et al. 1985).

Bone at the microscopic level can be used as a tool, that is, as a model system or biological "window" into the past giving a view of earlier behavior and health of the skeletal system (Frost 1964). The objectives of paleohistological analyses are to assess bone remodeling activity for entire populations and to examine the association of remodeling with age, sex, stature, pathological conditions, and cultural affiliation. The bridging of macroscopic data with microscopic data is seen as an essential step toward addressing differential health status across age, sex and culture. Techniques used by anthropologists must be based on biomedical precedents which are most applicable to archeological specimens. These techniques should include measure of *bone quantity* (cortical thickness, cortical area, and rate of remodeling) and *bone quality* (quantification of the size, distribution, and level of mineralization of discrete units of bone). These measures need to be based on well-defined skeletal parameters which are accurate, replicable, and useful in comparative analyses.

Examples of the types of multidimensional analyses which utilize histology include assessment of male and female differences in bone maintenance, correlation of macroscopic features of bone with microscopic features such as growth arrest, identification of subgroups at risk with respect to problems in bone metabolism, the effect of changing levels of sociopolitical organization and nutrition on health, and the effects of agricultural intensification on growth and development.

Given what is currently known concerning the relationship between structural and physiological functions of bone, optimal conditions for normal growth and development can be hypothesized. These optimal conditions include adequate nutrition, low disease stress, proper endocrinological function, and normal age-related wear and tear on the skeletal system. If these conditions are not met, the degree of dysfunction which results will parallel the seriousness and duration of the stressing agent (Ortner 1976). Analysis of health at the microstructural level can help define not only the existence, but also the severity, of stress in individuals with altered physiological states. Health information gained in this way can be assessed in conjunction with information from other areas, such as the archeological data. The comprehensiveness of the data will allow interpretations about the biological evidence of health, the archeological reconstruction of ecological and cultural variables, and the demographic profiles resulting from accumulated morbidity and mortality at the population level.

Loss of bone (osteopenia) and lack of bone mineralization (osteoporosis) are the two most important responses that skeletal tissue makes when under physiological stress. Gross pathologic changes on bone reveal insults to which an individual may have been subjected such as infectious disease, trauma, and degenerative changes such as arthritis. Traditionally, the health status of prehistoric individuals was based on these gross indicators alone. The analysis of bone histology provides information on a more subtle level—information concerning chronic or episodic undernutrition, periods of physiological disruption which leave no trace on the outer skeletal surfaces, the long-term effects of multiparity and lactation on female calcium metabolism, and the effects of immobilization on skeletal health.

In summary, analysis of physiological aspects of skeletal remodeling provides a clue to an individual's lifestyle, diet, reproductive activity, and nutritional adequacy. These indicators of health are rarely "clinically" significant; they reflect the day-to-day physiological responses which individuals must make. It is these responses at the histological level on which current research can focus.

Bone biodynamics

A general familiarity of normal bone histology, as well as the normal processes involved with growth, development, maintenance and repair, is necessary in order to highlight and understand the range of possible responses bone can make when experiencing physiological stress. Bone is a highly specialized kind of connective tissue and it is distinguished from other tissues such as skin and cartilage by its hard and crystalline structure. Bone has a cellular matrix composed of collagen and protein fibers embedded in a ground substance high in mucopolysaccharides. The hardness of bone comes from crystalline salts of calcium, phosphate, and carbonate deposited within the organic matrix. Specialized cells mediate the deposition and withdrawal of the mineral component of bone to keep an even balance between the body fluids and the skeletal reserves (Raisz and Kream 1983). Bone exists in a dynamic equilibrium with blood, and the "bone-body continuum" is regulated by nutrients and hormones (McLean and Urist 1968).

Much of the outward appearance, and most of the histological aspects of bone reflects the biological responses to physical and structural requirements. The diverse set of functions which a skeletal system provides (for example, structural support, locomotion, storage and regulation of minerals, control of ionic concentrations in body fluids, and production of red blood cells) necessitates a high priority for maintenance. Thus, as a connective tissue, bone is an open and living system which changes constantly throughout life to meet the demands for growth, development, maintenance and repair.

Disturbance in normal patterns of growth, mineralization, and remodeling form the pathogenesis of nutritional, hormonal, disease and aging problems (Jowsey 1964). These processes can be measured, and the values can be used to define boundaries between health and disease. Quantification of histological properties of bone aids in making diagnoses and indicates the probabilities that the properties of an individual case are within healthy or diseased states (Byers 1977).

Osteons are discrete units of bone which are the major quantifiable histological features used in diagnostic analyses. In the femur, osteons measure approximately 0.25 mm in diameter and are easily viewed under low or high magnification. Osteons take a variable length of time to form. In a ten-year-old individual osteons take 46 days to attain completeness and full mineralization. In a 60-year-old, osteons can take as long as 108 days to attain completeness (Lips et al. 1978).

Both complete osteons and partial osteon fragments remain visible for many years, since at any given time only 3–5% of the skeleton of an individual undergoes active remodeling during adulthood (Frost 1964). Osteons, resorption spaces, fragments, and layers of bone without osteons (lamellae) play a central and critical role in maintaining the quality and quantity of bone.

Once bone has been shaped by growth, it can be altered by the remodeling process, or "turnover" of bone (Frost 1973). There is activity of some degree in every part of the skeleton throughout life. The two basic components of this process are formation of osteons and resorption of older osteons. Remodeling is the resorption of older units of bone and formation of and replacement with newly mineralized bone. Remodeling can be viewed as quantitative changes in osteonal size, degree of mineralization, and placement within the cortex of bone.

Resorption and formation are not independent phenomena; they are coupled. After each resorption activity, there is always a formation activity although the rate is variable. The cycle of resorption and subsequent formation can take from three months to one year (Frost 1969). The formation/resorption ratio is a relative indicator of how many cycles are in progress, but does not indicate at what point the cycles are, or if the cycles are proceeding normally.

Although remodeling continues at predictably different rates in each decade of life, the consequence is an ever-increasing population of osteons per unit volume of cortical bone. Remodeling occurs slowly enough that previously remodeled bone remains unchanged for long periods of time. The actual rate of remodeling can be measured by comparing the number of older bone units with that of newer bone (Frost and Villanueva 1960; Jowsey 1964) or by determining the amount of bone laid down over the course of the life span (Frost and Wu 1967).

A healthy individual weighing 72 kg has approximately 14 kg of skeletal tissue. Of that 14 kg of bone, half is composed of calcium (Posner 1979). Calcium is extremely important to the biochemical constitution of skeletal tissue. Approximately 99% of the calcium ingested from food goes directly to the skeletal tissue; only 1% of the calcium remains circulating in the blood (Raisz 1977). The small percentage of calcium in the circulatory system is crucial for the maintenance of cardiac and nervous system function (Marshall et al. 1976).

In summary, diet, disease, and aging are all factors which affect calcium metabolism and bone remodeling, and these are especially valuable variables to track in prehistoric skeletal populations. This brief summary of bone dynamics serves to highlight the complexity of bone as a tissue and, more importantly, to suggest the ways in which the skeletal system responds to physiological disruption and the ways that this response can be reconstructed from the histological properties. Skeletal remodeling provides a measure of skeletal health as well as an indication of the health of the individual as a whole. Of particular interest to this research is that skeletal remodeling presents an ideal form of evidence leading to an understanding of prehistoric health. By understanding the physiological properties of skeletal remodeling, a clearer and more realistic reconstruction of past health can be made.

Response to stress on a histological level

Bone tissue is most affected by three factors: aging, disease, and nutrition (Smogyi and Kodicek 1969). Of particular significance to anthropological research is the fact that bone microstructure is sensitive to these factors, and tissue at the microstructural level is frequently preserved in archeological specimens (Stout 1978).

Bone reacts to stress in a limited number of ways. In general, the skeletal response to physiological stress is one (or a combination) of three phenomena: (1) reduced bone mass, (2) increased bone mass, and (3) poorly mineralized or abnormal bone quality (Meunier et al. 1979). It is the type of response, the timing of onset, the degree of severity, the pattern, and the frequency of abnormal histological properties which aid in the interpretation of remodeling.

It is a well-documented fact that structural and physical responses of bone to biological needs are affected by the aging process (Kerley 1965). A knowledge of normal, age-related processes in bone represents a factor critical to the understanding of pathological or abnormal conditions. One important distinction to be able to make when dealing with bone loss is the one between loss due to old age and loss due to other factors such as disease, malnutrition, or hormonal imbalances. Clinical methods for delineating bone loss as a function of age and other factors are outlined in Barzel (1979), Frost (1973), Jaworski (1973), and Simmons and Kunin (1979).

Skeletal remodeling can also be significantly altered by insufficient nutrients. The effects of nutrient deficiencies can be compounded not only by inadequacies in the diet, but by further problems in malabsorption of nutrients in the system. Experimental studies on animal models have shown the effects of protein-calorie malnutrition to be systemic and generalized (Steward 1975). During protein-calorie malnutrition growth slows or ceases, remodeling rates increase, and removal of bone exceeds deposition with mineralization of existing bone greatly slowed (Dickerson and McCance 1961).

Studies conducted on clinical populations experiencing nutritional stress support the findings that a general sequence of events are followed. These include (1) retardation in long bone growth and delayed maturation for children, (2) slowed formation of new bone for adults and children, and (3) existing bone loss by resorption with a net decrease in bone mass (Garn 1970).

While bone loss can be viewed as a pathological condition, it is important to note that the loss can also be seen as an adaptive response under certain circumstances such as nutritional stress. In the face of protein-calorie malnutrition or deficiencies in minerals, skeletal reserves can be used for growth, repair or function.

Rather than searching for a single diagnostic criterion, patterns of bone growth and maintenance must be examined with the emphasis on stress markers at the different stages of skeletal activity throughout the life cycle. Remodeling activities need to be carefully examined in conjunction with other variables such as age, sex, and pathologic conditions in order to interpret the nature and severity of nutritional stress.

Use of histology in anthropological research

As early as 1849, researchers were looking at microstructure of fossils, and in 1878 an extensive histological analysis of fossil bone and teeth demonstrated that histological structures were preserved in archeological specimens (Stout and Simmons 1979). Advances in technology led to a further understanding of the extent to which skeletal histology is preserved. Archeological specimens of varying age and from differing soil types and differing degrees of moisture exposure have been compared at the histological level for preservation. Race and co-workers (1968) found that the greatest alteration in skeletal material due to weathering was chemical in nature (not structural), and that osteons were often visible even in samples where severe chemical diagenesis had occurred.

During the early 1900s, attempts to detect abnormalities in preserved skeletons increased along with the methods used to assess pathological conditions (Brothwell and Sandison 1967). The development of radiographic techniques and the application to prehistoric specimens by Moodie (1923) made substantial progress in paleopathological research. Moodie's work often used histological sections, although analysis of the sections was not based on quantifiable measures. In spite of these early observations of morphology, no systematic analysis of histological structures was performed. Putschar stated, "one should not, however, expect too much help from the microscopic examination . . . since diagnostic microscopic bone patterns are rare" and he further emphasized that "gross examination of the surface is more important"

(1966:58–59). This lament has perhaps inhibited the growth of histological studies on prehistoric skeletal material, but the overwhelming success of more recent studies should put that attitude to rest.

Numerous methods have been developed to evaluate the absolute amount of bone present. One noninvasive technique used is photon absorptiometry. Perzigian (1973) used this technique to test the hypothesis that change in diet affected the rate of bone loss. Greater bone loss was found to occur in the agricultural group than in the gathering and hunting groups in an archeological population from Indian Knoll.

Stout and Teitelbaum (1976) offer one of the first detailed methodological considerations on both how to prepare and how to analyze a prehistoric thin section of bone. Practical information concerning embedding, staining, and mounting sections is reviewed. The authors suggest numerous avenues of potential research for the use of bone in the assessment of health and disease. Ortner (1976) also presents a review of the potential application of bone histology to ancient skeletal remains. This study emphasizes the ability of histological properties to give an indication of the aging process, nutritional adequacy, and disease status of prehistoric individuals. Martin and co-workers (1985) present a thorough review of the anthropological literature on methods for assessing quality and quantity of diet through the use of histological analysis.

An analysis of skeletal remodeling activity was undertaken for two Illinois Woodland populations representing the shift from intensive, harvest-collecting subsistence to that of corn agriculture (Stout 1976). Thin sections of rib were used and turnover rates were calculated, based on the density of remodeled osteons, to estimate actual amounts of bone formation per year in square millimeters for each individual. The results indicated a tendency toward increased bone remodeling rates in the agricultural population. The author suggested several possibilities for this, with dietary inadequacies serving to explain the findings best.

Patterns of remodeling have also been determined from microradiographs of femoral cross-sections. Martin and Armelagos (1979) combined cortical thickness and area measurements with histological analysis of osteons to examine bone loss and maintenance for an adult population from prehistoric Sudanese Nubia. Nubian females exhibit early and dramatic rates of bone loss on the organ level. Analysis of the distribution of osteons and the rate of osteonal remodeling further showed the differences between age-matched males and females. A diet low in calcium, iron, and protein, combined with endemic parasitic infestations, and the increased metabolic demands of reproduction created a negative skeletal balance for young adult Nubian females (Martin and Armelagos 1985).

Weinstein and co-workers (1981) studied the histology of a Peruvian mummy and found that the histomorphometrics were profoundly dissimilar from normal parameters. The researchers concluded that an imbalance between bone formation and resorption was the result of a dietary stress. Thompson and Gunness-Hey (1981) used microstructural analysis to examine bone loss in Kodiak Island Eskimo populations. Pfeiffer (1981) and Pfeiffer and King (1981) used osteon counts of prehistoric Canadian populations to analyze age structure and health.

Richman and co-workers (1979) looked at osteonal variations in Eskimo, Pueblo, and Arikara prehistoric populations. They were able to document a significant increase in the number of growth-arrested osteons in the Eskimos, and a dietary explanation was postulated.

In summary, the systemic and general nature of human skeletal response to stress has been profitably used on prehistoric remains to interpret the nature of the stressing agencies involved via examination of patterns of bone growth, remodeling, repair, and loss. The occurrence of physiological disruptions at different parts of the life cycle can be examined and compared to the mortality rates of the group. Information from indirect and direct examination of skeletal remains has been combined with environmental data to provide a more realistic reconstruction of the nutritional and health status of prehistoric groups.

Conclusions

In this brief and select overview of the anthropological uses of bone histology, there is ample evidence suggesting that analysis of bone histology can reveal information which far surpasses information obtained from macroscopic analyses only. Aging, disease, and nutritional stress are the main categories which can be tracked using histological analysis. Given that these categories are precisely those used to reconstruct the health dynamics of prehistoric peoples, it seems timely for histology to enter the mainstream of skeletal analyses in anthropology. For archeological populations, the aspect of nutritional and disease stress in endemic or epidemic conditions holds the most potential for interpreting prehistoric adaptation.

The use of histological analysis can highlight individuals who are experiencing health problems but who do not show gross pathologic changes. Further, subgroups within the population can be identified who are most sensitive to stresses which affect skeletal health, and by extension, stresses which affect overall patterns of morbidity and mortality.

Literature cited

Barzel, U.S., ed. 1979. *Osteoporosis II.* New York: Grune and Stratton.

Brothwell, D., and A.T. Sandison. 1967. *Diseases in Antiquity.* Springfield, Ill.: Charles C Thomas.

Byers, P.D. 1977. The Diagnostic Value of Bone Biopsies. In L.V. Avioli and S.M. Krane, eds., *Metabolic Bone Disorders*, 183–

236. New York: Academic Press.

Dickerson, J.W.T., and R.A. McCance. 1961. Severe Undernutrition in Growing and Adult Animals: 8. The Dimensions and Chemistry of the Long Bones. *British Journal of Nutrition*, 15:567–576.

Frost, H.M. 1964. Dynamics of Bone Remodeling. In H.M. Frost, ed., *Bone Biodynamics*, 315–334. Springfield, Ill.: Charles C Thomas.

———. 1969. Tetracycline Based Histological Analysis of Bone Remodeling. *Calcified Tissue Research*, 3:211–237.

———. 1973. *Bone Remodeling and its Relation to Metabolic Bone Disease*. Springfield, Ill.: Charles C Thomas.

Frost, H.M., and A.R. Villanueva. 1960. Measurement of Osteoblastic Activity in Diaphyseal Bone. *Stain Technology*, 35:179–189.

Frost, H.M., and K. Wu. 1967. Histological Measurements of Bone Formation Rates in Contemporary Archaeological and Paleontological Compact Bone. In W.D. Woode, ed., *Miscellaneous Papers in Paleopathology*, 9–22. Flagstaff: Museum of Northern Arizona.

Garn, S.M. 1970. *The Early Gain and Later Loss of Bone in Nutritional Perspective*. Springfield, Ill.: Charles C Thomas.

Jaworski, Z.F.G., ed. 1973. *Proceedings of the First Workshop on Bone Morphometry*. Ottawa: Ottawa Press.

Jowsey, J. 1964. Variations in Bone Mineral with Age and Disease. Mechanisms of Hard Tissue Destruction. *American Association for the Advancement of Science Publication*, 75:447–470.

Kerley, E.R. 1965. The Microscopic Determination of Age in Human Bone. *American Journal of Physical Anthropology*, 23:149–163.

Lips, P., P.J. Munier, and P. Courpron. 1978. Mean Wall Thickness of Trabecular Bone Packets in the Human Iliac Crest: Changes with Age. *Calcified Tissue Research*, 26:13–17.

Marshall, D.H., B.E.C. Noirdin, and R. Speed. 1976. Calcium, Phosphorus, and Magnesium Requirements. *Proceedings of the Nutrition Society*, 35:163–173.

Martin, D.L., and G.J. Armelagos. 1979. Morphometrics of Compact Bone: An Example from Sudanese Nubia. *American Journal of Physical Anthropology*, 51:571–578.

———. 1985. Skeletal Remodeling and Mineralization as Indicators of Health: An Example from Prehistoric Sudanese Nubia. *Journal of Human Evolution*, 14:527–537.

Martin, D.L., A.H. Goodman, and G.J. Armelagos. 1985. Skeletal Pathologies as Indicators of Quality and Quantity of Diet. In J. Mielke and R. Gilbert, eds., *The Analysis of Prehistoric Diets*, 227–279. New York: Academic Press.

McLean, F.C., and M.R. Urist. 1968. *Bone: Fundamentals of the Physiology of Skeletal Tissue*. Chicago: University of Chicago Press.

Meunier, P.J., P. Courpron, J.M. Geroux, C. Eduoard, J. Bernard, and G. Vignon. 1979. Bone Histomorphometry as Applied to Research in Osteoporosis and to the Diagnosis of Hyperosteoidosis States. *Calcified Tissue Research* (supplement), 21:354–360.

Moodie, R.L. 1923. *Paleopathology*. Urbana: University of Illinois Press.

Ortner, D.J. 1976. Microscopic and Molecular Biology of Human Compact Bone: An Anthropological Perspective. *Yearbook of Physical Anthropology*, 20:35–44.

Perzigian, A.J. 1973. The Antiquity of Age-Associated Bone Loss in Man. *Journal of American Geriatric Society*, 21:100–105.

Pfeiffer, S. 1981. Bone Remodeling Age Estimates Compared with Estimates by Other Techniques. *Current Anthropology*, 21:793–794.

Pfeiffer, S., and P. King. 1981. Intracortical Bone Remodeling and Decrease in Cortical Mass Among Prehistoric Amerindians. *American Journal of Physical Anthropology*, 54:262.

Posner, A. 1979. The Relationship Between Diet and Bone Mineral UltraStructure *Federal Proceedings*, 26:7717–1722.

Putschar, W.G.J. 1966. Problems in the Pathology and Paleopathology of Bone. In S. Jarcho, ed., *Human Paleopathology*, 57–65. New Haven, Conn.: Yale University Press.

Race, G.J., E.I. Fry, J.L. Mathews, H. Martin, and J.A. Lyon. 1968. The Characteristics of Ancient Nubian Bone by Collagen Content and Light and Electron Microscopic Examination. *Clinical Pathology*, 45:704–713.

Raisz, L.G. 1977. Bone Metabolism and Calcium Regulation. In A. Avioli and D. Krane, eds., *Metabolic Bone Disorders*, 223–230. New York: Grune and Stratton.

Raisz, L.G., and B.E. Kream. 1983. Regulation of Bone Formation (I and II). *New England Journal of Medicine*, 309:29–35, 83–89.

Richman, G.A., D.J. Ortner, and F.P. Schulter-Ellis. 1979. Differences in Intracortical Bone Remodeling in Three Aboriginal American Populations: Possible Dietary Factors. *Calcified Tissue Research*, 28:209–214.

Simmons, D.J., and A.S. Kunin. 1979. *Skeletal Research*. New York: Academic Press.

Smogyi, J.C., and E. Kodicek, eds. 1969. Nutritional Aspects of the Development of Bone and Connective Tissue. *Series of the Institute of Nutrition Research*, 13.

Steward, R.J.C. 1975. Bone Pathology in Experimental Malnutrition. *World Review of Nutrition and Dietetics*, 21:1–74.

Stout, S.D. 1976. Histomorphometric Analysis of Archaeological Bone. Ph.D. dissertation, Department of Anthropology, Washington University, St. Louis.

———. 1978. Histological Structure and its Preservation in Ancient Bone. *Current Anthropology*, 19:601–604. Stout, S.D., and D.J. Simmons. 1979. Use of Histology in Ancient Bone Research. *Yearbook of Physical Anthropology*, 22:228–249.

Stout, S.D., and S.L. Teitelbaum. 1976. Histological Analysis of Undecalcified Thin Sections of Archaeological Bone. *American Journal of Physical Anthropology*, 44:263–270.

Thompson, D.D., and M. Gunness-Hey. 1981. Bone Mineral Osteon Analysis of Yupik-Inupiaq Skeletons. *American Journal of Physical Anthropology*, 55:1–7.

Weinstein, R.S., D.S. Simmons, and C.O. Lovejoy. 1981. Ancient Bone Disease in a Peruvian Mummy Revealed by Quantitative Skeletal Histomorphometry. *American Journal of Physical Anthropology*, 54:321–326.

SUMMARY OF AUDIENCE DISCUSSION: In response to the question of the role of estrogen in bone remodeling, it was suggested that the hormone has a stimulating effect on endosteal osteoblasts and that grand multiparas tend to have thicker bones, although prolonged lactation may deplete bone mineral content.

8000-year-old brain tissue from the Windover site: Anatomical, cellular, and molecular analysis

William W. Hauswirth, Cynthia D. Dickel, Glen H. Doran, Philip J. Laipis, and David N. Dickel

The Windover site (8BR46) consists of a small (5400 m^2) peat deposit in a low-lying swale on the western edge of the Florida Atlantic Coastal Ridge, roughly equidistant from the Indian River coastal lagoon system and St. John's River in eastern, central Florida (Figure 1). Information from preliminary analysis of flora and fauna indicates the site was a wooded marsh from 8000 B.P. to 6900 B.P. and during this time was regularly used as a burial ground. Most bodies found at the site had been placed in a flexed position and then buried lying on their sides in anaerobic, water-saturated peat at an approximate depth of one meter.

The Windover site is a significant North American archeological site for several reasons: it is one of the oldest American sites with a large, representative human skeletal sample; it contains all age morphs; it has a large sample of prehistoric flexible fabrics; it has 91 crania containing preserved matter identifiable as brain tissue; and intracranial tissue was demonstrated to be human by cellular and biochemical techniques and by isolation of human DNA (Doran et al. 1986).

Results

RADIOCARBON DATES

During excavation four distinct types of peat strata were identified (Figure 2). The upper stratum (1.2 m thick) was composed of black sawgrass-peat (W. Spackman, Jr., and S. Stout, pers. comm. 1986). The lower levels of this black peat were dated at 4790± 100 B.P. (Beta–10763). Underlying the black peat was a 1.2-m-thick stratum of red-brown peat containing a high concentration of naturally deposited wood. The upper zone of this red-brown peat has been radiocarbon dated at 5800±80 B.P. (Beta–10764). The red-brown peat stratum had a striking preservation of intact leaves, sawgrass strands, twigs, branches, turtle bones, fish remains, nonhuman fecal material and other faunal material. The highest concentration of skeletal material was within the lowest levels of the red-brown peat stratum. Underlying the red-brown peat was an approximately 0.5-m-thick layer of "rubber" peat. The top of the rubber peat stratum has been radiocarbon dated at 7950±140 B.P. (Beta–10855). Human and nonhuman skeletal material and preserved wood decreased with increasing depth of the rubber peat. The incidence of freshwater mollusks was high in the rubber peat and may have influenced the water chemistry of the pond. Beneath the rubber peat was a 1.8-m-thick stratum of tan-brown peat

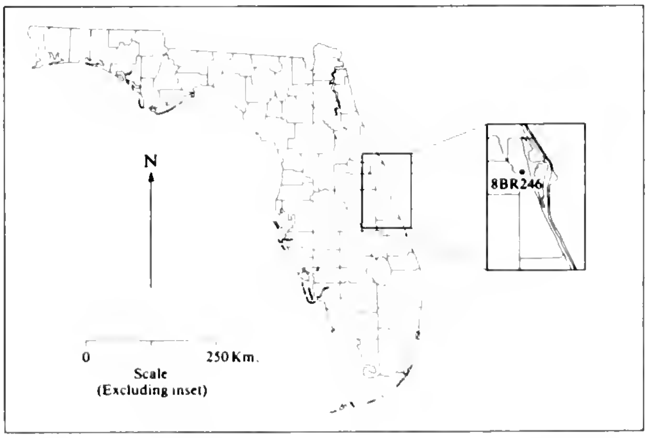


FIGURE 1. Geographic location of the Windover site, 8BR246

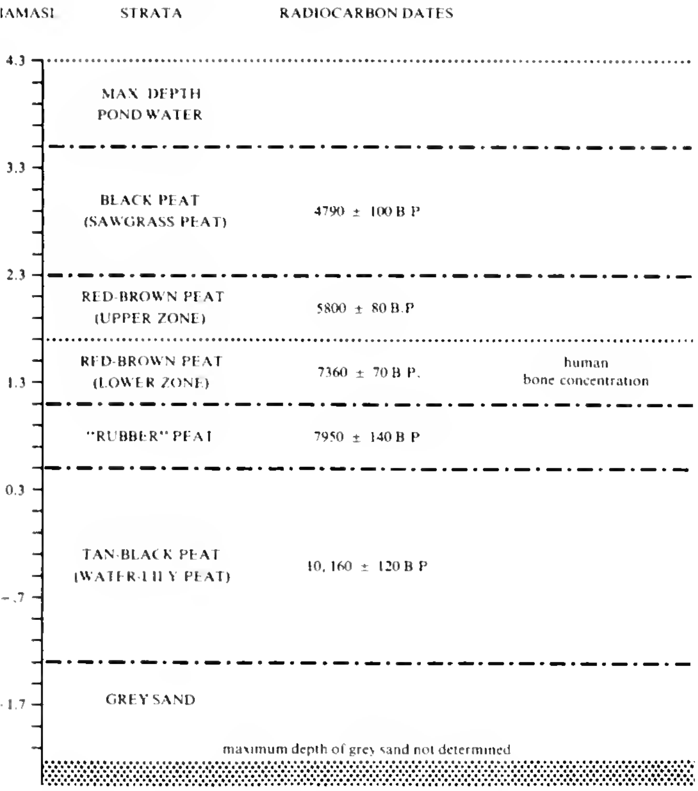


FIGURE 2. Stratigraphic profile, peat types, and uncorrected radiocarbon dates on peat from the Windover site, 8BR246. Radiocarbon dating either by Isotrace Laboratory, University of Toronto, Canada, or by Beta Analytical, Coral Gables, Florida. Sample numbers indicated in text (e.g., Beta 10763). Dates in years B.P. Correction factor of +800 yr B.P. should be added to dates between 7000 and 10,000 yr B.P. (Klein et al. 1982). *MAMASL*, meters above mean annual sea level. Vertical scale units, 20 cm.

containing no freshwater mollusks or human bone. This stratum has been described as water-lily peat (W. Spackman, Jr., and S. Stout, pers. comm. 1986) and radiocarbon dated at over 10,000 years. Underlying the entire deposit was a gray Pleistocene sand.

The chronometric placement of Windover skeletal material is based on a series of radiocarbon dates (Table 1). These dates were obtained directly from human bone, from the top and bottom of vertical burial stakes, peat above, within, and beneath human bone, and from multiple locations within the pond. Recent radiocarbon corrections (Beukens 1986) indicate the dates would cluster the human activities at Windover at approximately 7450 B.P. The radiocarbon dates place the utilization of Windover in a chronological period that is usually considered Early Archaic in the southeastern United States (Milanich and Fairbanks 1980).

TABLE 1. Culturally relevant radiocarbon dates

Date[a]	*Sample*	*Laboratory number*
6980 ± 90	Wooden stake	Beta-19316
7050 ± 80	Peat near highest bone	Beta-14132
6990 ± 70	Human bone (AMS)	TO-207
7100 ± 100	Wooden stake	Beta-19315
7210 ± 80	Human bone	Beta-7186
7290 ± 120	Bottle gourd	Beta-20450
7300 ± 70	Wooden stake	Beta-19722
7330 ± 100	Human bone	Beta-5803
7360 ± 70	Peat beneath crania	Beta-11381
7410 ± 80	Peat from brain surface	Beta-11383
7830 ± 80	Human bone (AMS)	TO-518
7930 ± 80	Wooden stake	Beta-18295
8120 ± 70	Human bone (AMS)	TO-241
8430 ± 100	Peat at base of red-brown strata	Beta-13909

a. Date in radiocarbon years B.P.

CULTURAL MATERIALS

The Windover burials were accompanied by a diverse cultural inventory. Artifacts fabricated from animal teeth, antler, bone, seed, wood, shell and stone were found. Bone awls and pins were the most commonly recovered artifact category and were manufactured from upland game including deer, canids, and felids. Drilled antler and manatee ribs and atlatl cups were also found. A bottle gourd *(Lagenaria siceraria)* accompanying a burial provides early evidence of curcurbits north of Mexico; it predates other *Lagenaria* and virtually all other known Curcurbitacae north of Mexico (Conrad et al. 1984; Kay et al. 1980; Prentice 1986). The status and morphology of the specimen is being carefully evaluated particularly in light of its early context (Newsom 1987).

Additionally, textile materials were recovered from 37 of the burials. Seven twining/weaving variants have been identified which include fine-balanced, plain-weave inner garments, more durable complex-twined materials possibly representing blanketlike items, twined globular bags, open-twined items and matting (Andrews and Adovasio 1988). Macroscopic and microscopic thin sections of plant fibers in the fabrics have been unsuccessful in identifying the plant species utilized. Morphological features that would normally prove taxonomic criterion were apparently removed during the original processing of plant fibers or simply have not been preserved for 8000 years (Andrews and Adovasio 1988; Newsom 1987). Phytolith studies also failed to provide information for the identification of plant fibers (Piperno 1987).

TABLE 2. North American material with firm dating in excess of 6500 radiocarbon years B.P. (uncorrected)

Provenience	*Date*[a]	*Sample size*	*Reference*
Republic Groves, FL	6520–5745	37	Wharton et al. 1981
Bay West, FL	6630 ±80	30–45	Beriault et al. 1981
Carrier Milles, IL	6750–5650	159	Bassett 1982
Union Lake, MI	7000 ±400	1	Black and Eyman 1963
Eva, TN, Stratum IV	7150 ±500	17	Lewis and Lewis 1961
Tecolote Point, CA	7230–3970	79	Berger et al. 1971; Protsch 1978.
Glen Annie, CA	7400–6700	7–8	Owen 1964.
Angeles Mesa, CA	7900–4050	6	Taylor et al. 1985
Modoc Rock Shelter, CO	7970–6219	24	Neumann 1967
Batiquitos Lagoon, CA	8000 ±3000	?	Bada 1985; Bada and Helfman 1975
Scripps Estates, CA	8000–4820	2	Taylor et al. 1985; Bada 1985
San Diego series, CA	8100–5000	46	Bada and Helfman 1975; Ike et al. 1979; Protsch 1978
Windover, FL	8120–6990	172	
Sunnyvale, CA	8200–3600	1	Bada and Helfman 1975; Taylor et al. 1985
San Diego, CA (SDM 16709)	8360 ±75	1	Ike et al. 1979
La Brea, CA	9000–4450	1	Taylor et al. 1985
Renier, WI	9524–6300	1	Mason and Irwin 1960
Cutler Ridge, FL	9670 ±130	5	Carr 1987
Gordon Creek, CO	9700 ±250	1	Breternitz et al. 1971
Arlington Springs, CA	10000 ±200	1	Protsch 1978
Horn Shelter, TX	10310 ±150	2	Young 1985, 1986
Sulphur Springs, AZ	10420–8200	1	Waters 1985, 1986
Mostin, CA	10470–4000	1	Kaufman in Taylor et al. 1985; Young 1986
Warm Mineral Springs, FL	10500 ±1700	21	Clausen et al. 1975; Lien 1983
Anzik/Wilsall, MN	10600 ±300	1	Taylor 1969
Marmes Rockshelter, WA	10750–6200	28	Sheppard et al. 1987
Fishbone Cave, NV	11200–10900	1	Young 1986
Wilson-Leonard, TX	13000 ±3000–9470	1	Weir 1985
Midland, TX	13400–7100 ±1000	1	Wendorf et al. 1955

NOTE: The age range and sample sizes are listed when possible
a. Date in radiocarbon years B.P.

OSTEOLOGICAL MATERIAL

Few large samples of human skeletal material earlier than 6500 B.P. have been recovered from New World deposits (Table 2). Therefore, the Windover collection provides an opportunity for detailed analysis of such a population. Data currently being obtained includes general nonmetric, metric and morphological features, disease morbidity, age, sex and other paleodemographic features. The abundant subadult material (52%) provides an excellent opportunity for the study of growth and development in this population.

Some initial data on the skeletal material has been reported. Preliminary evidence for nutritional/metabolic stress based on transverse lines, cribra orbitalia, enamel defects and cranial hyperostosis has recently been summarized (Dickel 1986). Interproximal grooves have been described and compared to similar dental modifications found in other prehistoric populations (Dickel in press). A case of severe lumbar spina bifida aperta complicated by scoliosis has also been reported (Dickel 1987; Dickel and Doran 1989). This case shows strong evidence of sensory loss in the lower limbs including the loss of the distal tibia due to a massive infectious lesion.

TABLE 3. Water chemistry of the Windover site (mg/l)

	Laboratory Number			
	8789[a]	8797[b]	10604[c]	10491[d]
Calcium	160.0	81.5	94.0	4.0
Magnesium	77.0	60.5	68.0	4.6
Total dissolved solids	1447.5	1420.0	1418.0	170
Total hardness	716.4	452.5	27.5	28.8
Carbonate hardness	31.2	8.9	ND	0.1
Noncarbonate hardness	685.2	443.6	527.5	28.8
Total alkalinity	31.2	8.9	498.2	0.1
Bicarbonate	31.2	8.9	ND	2
Carbon dioxide	38.1	34.3	6.4	1.87
Total phosphate	0.99	0.01	ND	ND
Sulfides	ND	1.6	0.05	0.04
Chlorides	189.0	472.4	ND	ND
Strontium	4.3	2.3	4.8	0.27
Sulfates	381.9	33.9	ND	ND
Fe	9.3	35.5	ND	ND
Mn	0.105	0.01	ND	ND
Cu	1.38	0.03	ND	ND
Zn	0.06	0.17	ND	ND
K	6	26.0	ND	ND
Na	200	350.0	ND	ND
pH	6.1	5.6	6.9	2.9

a. Water standing in Windover pond prior to excavation or wellpointing. This represents percolation of waters from the peat deposits with some mixture of surface runoff and rainwater.
b. Wellpoint sample taken moments after 5 wellpoints were installed - this is probably the best estimate of the waters actually saturating the peat strata.
c. Water seeping into one of the deeper excavation units, it predominantly reflects the underlying waters with some mixture of the peat waters.
d. Water from a nearby pond.

WATER CHEMISTRY

In contrast to a nearby pond, Windover water was substantially more mineralized with high amounts of calcium, magnesium, total dissolved solids, and sulfates (Flowers Chemical Laboratory, Altamont Springs, Florida). Additionally, the pH of the Windover samples are generally closer to neutral than the nearby pond water samples (Table 3).

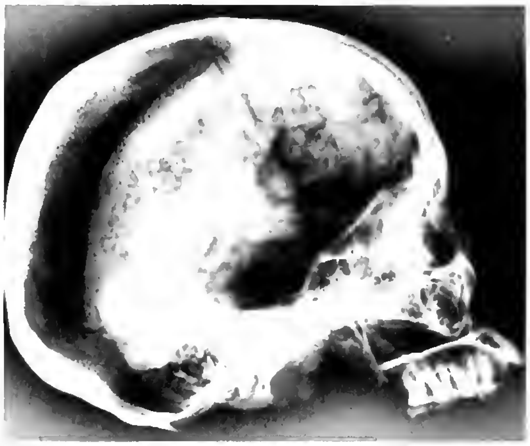


FIGURE 3. Lateral radiograph shows material that, though shrunken in comparison to the bone and settling toward the base, resembles the expected configuration of brain within the skull. This mass proved to be peat surrounding a residual brain.

ANATOMICAL AND CELLULAR ANALYSIS

Based on the number of adult crania and subadult crania and mandibles, at least 172 individuals were recovered from the Windover site. The recovered bone was well preserved and nonfriable. Ninety recovered crania contained soft tissue remnants, sometimes preserved as recognizable brains and sometimes as amorphous masses of brain tissue mixed with peat. Bone fragments from one of the crania containing a brain were dated at 6990±70 B.P. (Isotrace TO-207) by accelerator mass spectrometry (AMS) dating.

Age and sex determinations based on cranial structure, dental attrition, and limited postcranial skeletal evidence indicated the two adult crania initially analyzed were from a female at least 45 years old and a male about 25 years old. When the female intracranial mass was removed, the immediate visual impression was of a human brain; however, it was extremely fragile, making handling difficult. Therefore, analysis of the male cranium was carried out by a noninvasive appraisal using conventional x-ray imaging, computerized axial tomography, and proton magnetic resonance imaging (MR).

X-ray analysis proved useful in evaluating brain position but provided little additional information (Figure 3). Chemically different components were easily recognized and differentiated by false color MR imaging which enhanced contrast (Figure 4). In this analysis, the skull content ranges in contrast from light gray to dark gray. A high MR signal intensity is represented by light gray and low by dark gray. Living tissue yields a high signal intensity from soft tissue like fat and a lower signal intensity from dense connective tissue. When the brains were later sectioned, the dark gray region proved to be peat, whereas the light gray region was clearly brain matter (Figure 5).

Gross examination of the brain masses after removal from the skull disclosed the external gyral pattern of an atrophic human brain shrunken to approximately 1/4 the original size. No meningeal coverings or blood vessels remained. The brain was tan-gray and had a soft, granular consistency. The two cerebral hemispheres, divided by the longitudinal fissure, were identifiable, and the Sylvian fissure was visible on either one or both hemispheres. Cerebellar tissue containing visible folia was present below the occipital lobes. The region of the brain stem in all cases was amorphous and finer structure could not be identified. Transverse slices of the remaining material exposed parietal, temporal, and occipital lobes with peat filling all fissures. Additionally, internal structures such as the thalamus, basal ganglia, and ventricular system were clearly visible (Figure 5). The overall impression was that although shrunken and altered in consistency, gross anatomical features were present.

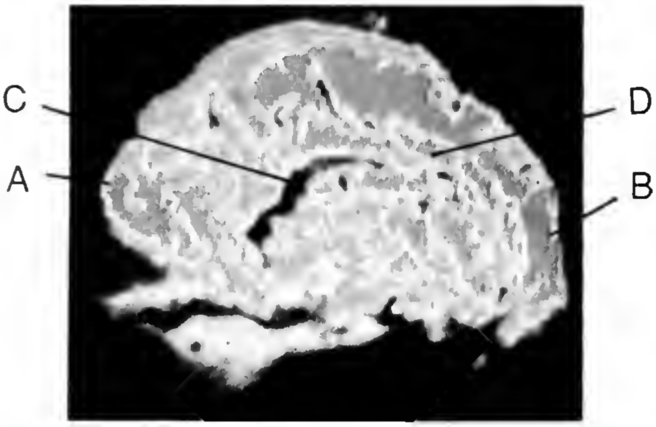


FIGURE 4. Magnetic resonance image, 1 cm thick, of medial portion of adult male brain 57-300, sagittal section, specimen facing right. Skull removed and brain embedded in agar (uniform medium gray). Brain material is light gray. Several gross anatomical features are identifiable: *A*, occipital pole; *B*, frontal pole; *C*, lateral ventricle; *D*, cingulate gyrus. Images produced by a Technical Teslacon 0.15T resistive magnet unit. Machine and pulsing techniques used in routine clinical practice.

A more detailed analysis of tissue structure was conducted by light microscopy. Representative samples taken from the cerebral hemisphere, cerebellum, and brain stem of both brains were processed for light microscopy. Sections were stained with silver axon stain (Seiver-Munger), hematoxylin and eosin (H&E) for nuclei, and luxol fast blue (LFB) for myelin (Luna 1968). The silver-stained, H&E, and LFB sections resembled cerebral cortex and contained yellow granular pigment, often in pyramidal shapes, corresponding to the remains of neurons (Figure 6A). The cyto-architectural pattern was similar to that of fresh cerebral cortex tissue. Occasional processes consisting of parallel, arrayed fibers extending to the cortical surface, as well as horizontal, arrayed fibers within the subcortical white matter, were present. No material staining as nucleic acid could be identified, although clear areas the size and shape of nuclei were apparent within the granular pigment (Figure 6B). Sections of cerebellum contained preserved Purkinje cells arranged in the spatial pattern seen in contemporary cerebellar tissue (Figure 6B). A section of the pons contained the typical pattern of crossing pontocerebellar tracts as well as cortical spinal tracts divided by remains of pontine nuclei (Figure 6C). Cellular processes in all sections were negative for silver stain but showed moderate staining throughout with myelin stains.

No connective tissue elements were identified with Masson trichrome staining for collagen. Immunoperoxidase staining for glial fibrillary acid protein (GFAP) and S-100 protein was also negative.

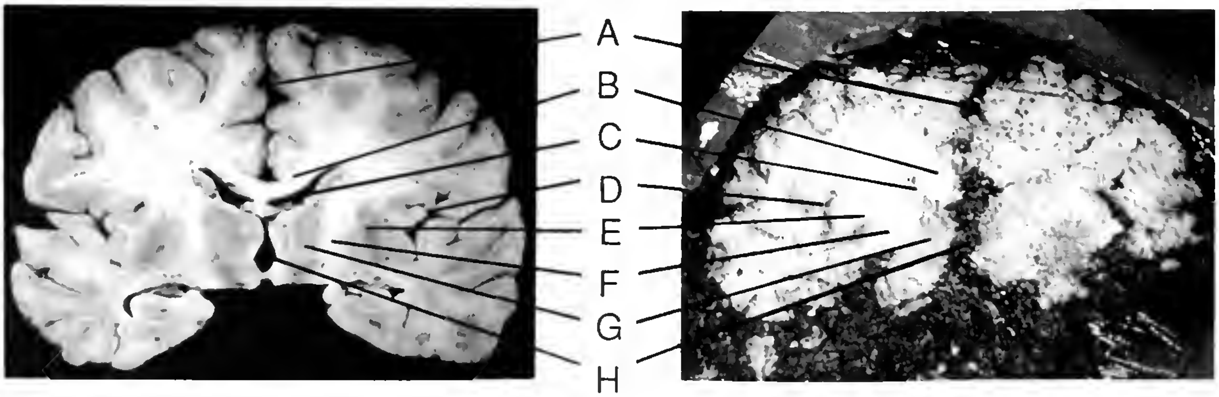


FIGURE 5. Coronal section through agarose gel-embedded ancient brain of male 57-55 *(right)*, and a similar section of a contemporary brain *(left)*. Although the old brain section is surrounded by peat and has undergone some fragmentation, many gross anatomical structures are present. Two cerebral hemispheres and their gyral patterns clearly visible. *A*, interhemispheric fissure; *B*, corpus callosum; *C*, lateral ventricle; *D*, insular cortex; *E*, putamen; *F*, internal capsule; *G*, thalamus; *H*, third ventricle. A subtle distinction between grey and white matter is still apparent.

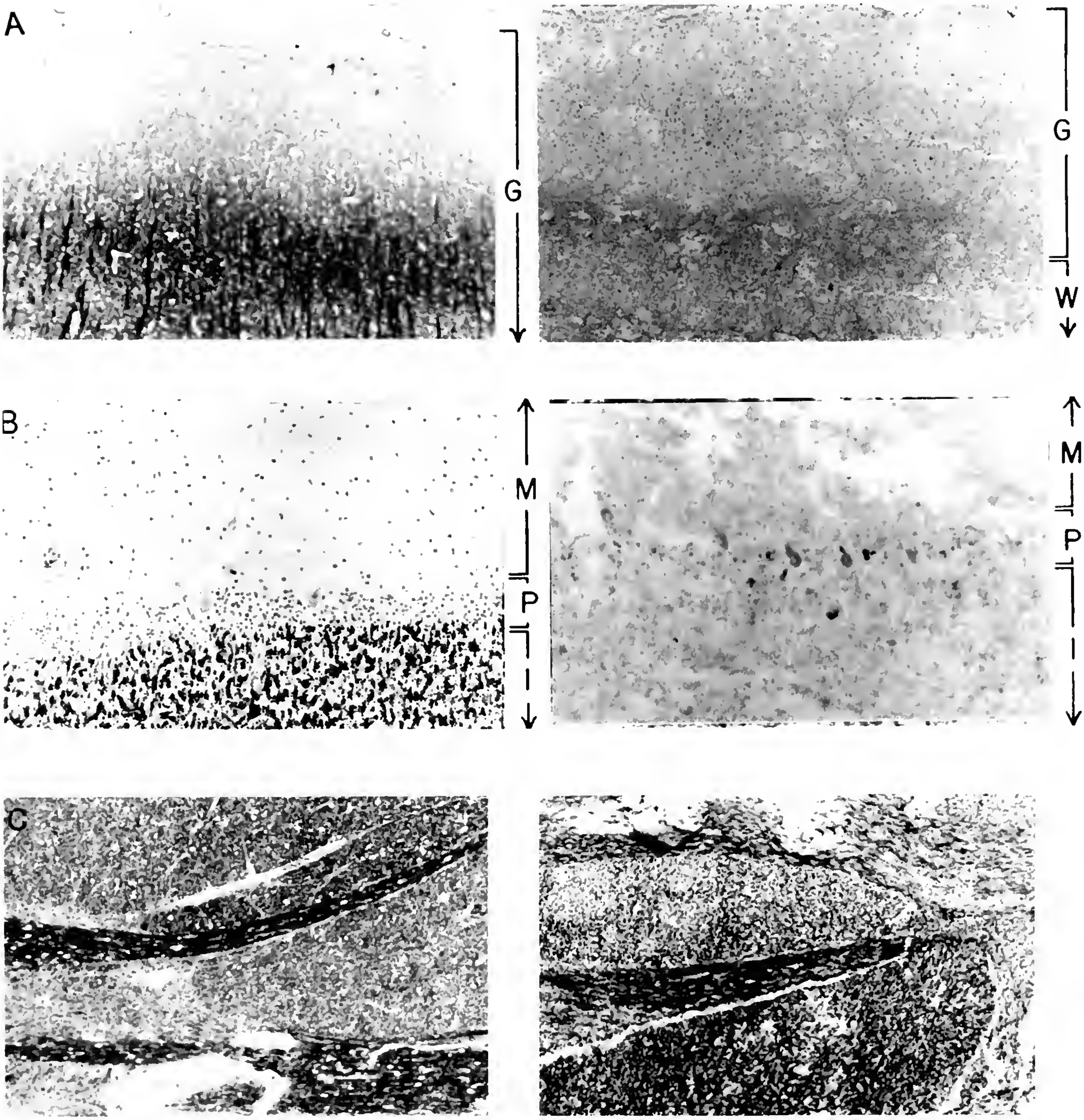


FIGURE 6: Histological comparison of contemporary brain *(left)* with ancient female brain *(right)*. *A*. Sections of cerebral cortex visualized with silver axon stain (Luna, 1968). Surface of each brain at top. Small amount of peat visible on surface of ancient brain. Division between gray and white matter indicated on ancient brain section. At same magnification, section of modern brain shows only gray matter, due to general tissue shrinkage suffered by ancient brain. Vertical axonal fibers visible in gray matter of both sections. Small darkly staining particles in ancient brain probably represent remains of neurones. (Seiver-Munger stain, ×100). *B*. Sections of cerebellum stained with hematoxylin and eosin (Luna 1968). Correlation of contemporary and ancient molecular layers *(M)*, Purkinje cell layers *(P)*, and inner granular layers *(I)* indicated on right. Clear areas in center of most Purkinje cells in ancient cerebellum are probably residual nuclei (contemporary section, ×250; ancient section, ×400). *C*. Section of pons stained with Luxol fast blue for myelin (Luna 1968). Cross-sectional views of cortical spinal tracts and longitudinal sections of pontocerebellar tracts clearly evident in both (contemporary section, ×100; ancient section, ×200).

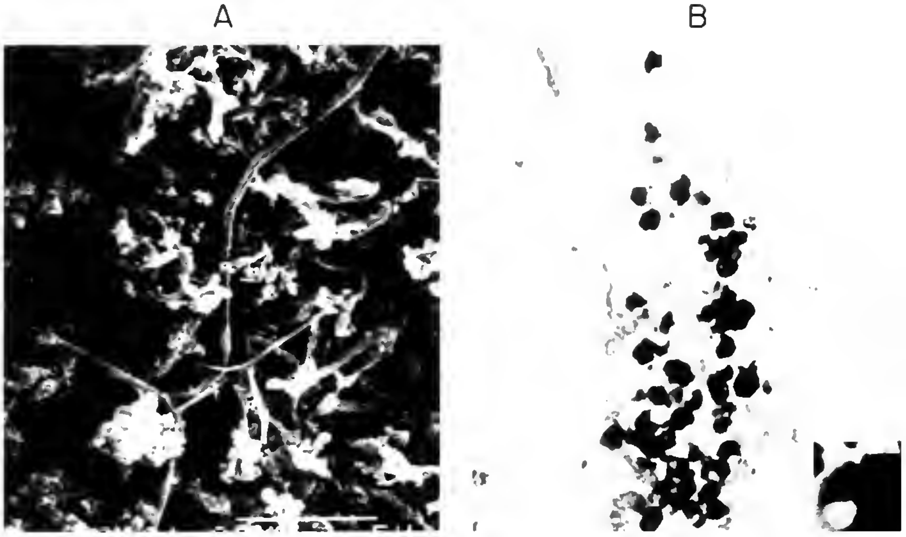


FIGURE 7. *A*. Scanning electron micrograph of cerebral cortex. Remains of neuropil made up of intermingling tubular processes can be seen. Occasional structures representing neuronal remains can be identified at lower left. Bar = 5 μm. *B*. Electron micrograph of pyramidal neuron of cerebral cortex. Cell shape can be inferred from included lipofuscin granules (Luna 1968). These electron dense granules show characteristic peripheral vacuole *(inset)*. Remainder of cell shows finely granular, moderately electron dense material with a clear region in center (also seen by light microscopy), which may represent nucleus (Luna 1968). Two circular profiles at upper right represent remains of neuritic processes. (×8000, inset ×22,000).

Scanning electron microscopy revealed a background of processes and presumptive neurons observable as accumulations of granular structures with an outer membranelike covering (Figure 7A). Transmission electron microscopy indicated the processes have a pattern reminiscent of myelinated structures; however, myelin lamellae were not identified. No organelles associated with neurons or their processes were present. The most striking finding was an accumulation of electron dense bodies which corresponded to the yellow, granular pigment seen by light microscopy (Figure 7B). These granules resemble lipofuscin pigment (Taubold et al. 1975; Adams and Lee 1982:234–237). Consistent with this interpretation was the finding of more pigment in the older brain (female) than in the younger brain (male).

ISOLATION AND DEMONSTRATION OF HUMAN DNA

Nucleic acids were extracted and purified from 15g of relatively peat-free cortex by solubilizing, chloroform-phenol extracting, and centrifuging in a CsCl-ethidium bromide density gradient. Material banding at a density of 1.55 g/cc was collected and identified as DNA by DNase sensitivity and RNase resistance. High molecular weight DNA of 8–20 kilobases was clearly present in an ethidium bromide stained gel of this DNA (Figure 8, left).

To determine whether this DNA was of human origin, a gel was blotted and hybridized to a probe specific for human mitochondrial DNA (mtDNA) (Chang and Clayton 1985). The probe hybridized to appropriate sized species in undigested brain DNA demonstrating that human mtDNA was present (Figure 8, right). To confirm the presence of human DNA a dot blot of 8000-year-old DNA was probed with an Alu repeat sequence (Figure 8, right). The Alu sequence hybridized to old human DNA but not to a peat sample from the same level. The experiment was repeated several times using DNA samples from different old brains with similar results.

The total yield of DNA was about 1 μg/g tissue, or 1% of that normally isolated from fresh tissue. Also, the amount of mtDNA present in the old DNA sample appears low relative to total isolated DNA. A comparison of hybridization suggests that about 0.05% of the total old DNA was mtDNA; DNA isolated from fresh brain tissue yields 0.5%–1% mtDNA. Quantitation of Alu sequences on dot blots allowed an independent estimate of the fraction of human DNA sequences in the old DNA samples (data not shown). We estimated that Alu sequences were present at 1% of the level of that from an equivalent amount of human placental DNA. The low yield of human mtDNA sequences could have several potential causes: preferential loss of mitochondrial sequences may occur during extraction; preferential degradation of mitochondrial sequences may occur during 8000 years or during the immediate postmortem period; and significant amounts of nucleic acids from the surrounding plant material may be present in the old-brain cortex sample. If the latter situation is the case, the apparent fraction of any specific human sequences would be diluted by plant DNA sequences. Although the surrounding peat does contain about the same amount (on a per weight basis) of DNA as brain tissue, this DNA does not hybridize to the human mtDNA probe (Figure 9).

When the mtDNA was digested with Eco R1, the expected (Anderson et al. 1981) 8kb fragment which should appear after hybridization with the probe was not present (Figure 8, right). However, partial conversion of open-circular to linear molecules did take place, as would be expected if only a fraction of the Eco R1 recognition sequences were present.

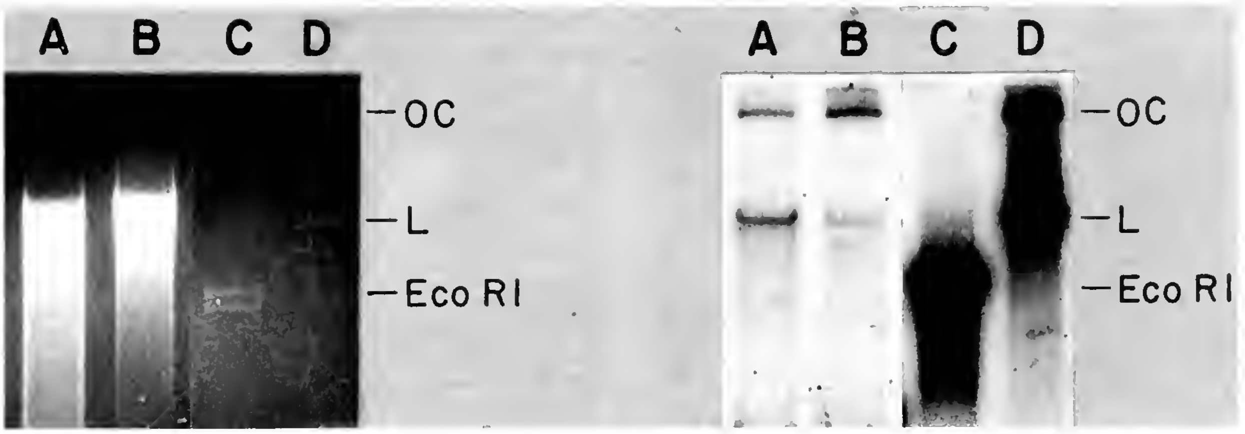


FIGURE 8. Identification of human mtDNA sequences in DNA from 8000-year-old adult male brain. Total DNA isolated and purified as described in text. An aliquot was digested with EcoRI and electrophoresed on a 1% agarose gel along with an undigested aliquot and samples of authentic human KB cell mtDNA treated similarly. After staining and photography, gel was blotted and hybridized to a human mtDNA-specific probe (Chang and Clayton 1985), washed and autoradiographed. Central lanes containing enzymes, buffers, and carrier tRNA used in extraction, and enzyme digestion showed no hybridization to probe. *Lane A*, undigested KB cell mtDNA; *lane B*, EcoRI-digested KB cell mtDNA; *lane C*, undigested ancient DNA; *lane D*, EcoRI-digested ancient DNA. *OC*, *L*, and *EcoRI* refer to positions of open- (nicked) circular, linear, and 8050-base pair (bp) EcoRI (D-loop-containing) fragment of KB cell mtDNA, respectively (Anderson et al., 1981; Houck et al., 1979). *Left:* Ethidium-stained gel. *Right:* Autoradiograph of blotted gel. Lanes A and B were exposed for 3 days, lanes C and D for 7 days.

Resistance to enzyme digestion is an intrinsic property of the old DNA because a bacterial plasmid DNA mixed with this DNA sample did digest to completion under the same conditions. The inability to completely digest the DNA may be due to base modification leading to a loss of restriction endonuclease site recognition.

The old DNA lacked supercoiled, covalently closed circles (Figure 8, right). The reason for the absence of supercoiled molecules has not been investigated further at present, but many spontaneous processes can lead to single-strand nicks in DNA, converting covalently closed molecules to open-circular forms (Vinograd et al. 1965). Multiple single-stranded nicks or damage resulting in a double-stranded scission would lead to linear, full-length molecules, as was seen.

High molecular weight and a surprisingly high fraction of intact open-circular mtDNA was observed in these DNA samples (Figure 8). Both of these observations may be due to DNA damage caused by depurination leading to interstrand-crosslinking of DNA (Goffin et al. 1984). Crosslinking would raise the apparent double-stranded molecular weight of the DNA, greatly increase the lifetime of circular DNA forms, interfere with restriction digestions, and perhaps also interfere with DNA hybridization experiments by preventing strand separation. This type of damage has been shown to be enhanced in aqueous solution (Goffin et al. 1984).

The presence and condition of nuclear DNA was also investigated. Restriction endonuclease-digested DNA was probed with radiolabeled nuclear DNA and RNA sequences present as multiple copies in the human genome (an Alu sequence, a large and small ribosomal RNA) (Houck et al. 1979; Long and Dawid 1980). None of these probes hybridized to restriction fragments of a defined size (data not shown). If nuclear DNA was damaged in a similar manner as mtDNA and largely resistant to restriction endonuclease digestion, discrete restriction fragment bands of multicopy or single copy genes would not occur in hybridization experiments. In contrast, mtDNA occurs as a small 16 kb circular molecule and migrates as a discrete species without depending upon recognition of specific undamaged sequences by restriction endonuclease; therefore, it can be detected in hybridization experiments. Alkaline cleavage of the 8000-year-old DNA also reveals a significant amount of DNA damage (data not shown). It is estimated that these alkali-sensitive sites, many of which may represent apurinic nucleotides, are present at the 1% level.

An attempt was made to clone and compare DNA sequences from several brains with nucleotide sequences of genes or other DNA sequences already known. Thus far, old brain nucleotide sequences have not corresponded to any known DNA sequence. A small library of DNA fragments was constructed using a partial Alu I digest and an M13 cloning vector. Approximately 1000 clones containing small inserts (50–1000 bp) were isolated and 90 were screened for homology to human mtDNA, human Alu repeat sequences,

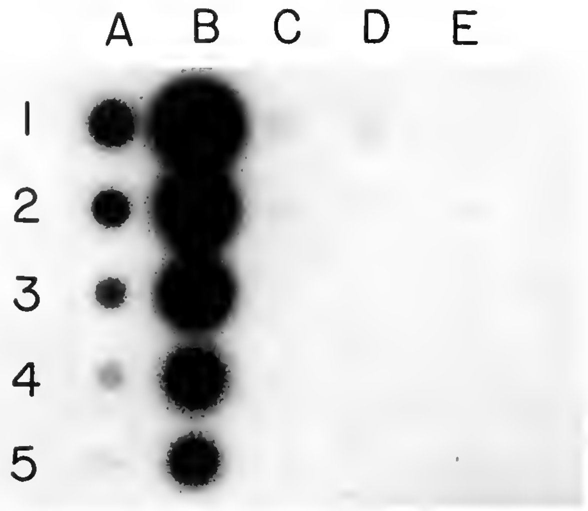


FIGURE 9. DNA dot blot comparing amount of human Alu sequences (plus pBR322 plasmid DNA) in 8000-year-old DNA, nearby peat DNA, and contemporary human DNA. Cloned Alu sequence (in pBR322) was nick translated and hybridized to dot blot containing: *lanes A and B*, pBR322 DNA 100 ng (B-1) to 0.2 ng (A-5); *lane C*, 100 ng of human placental DNA; *lane D*, 10 μg of 8000-year-old DNA from Skull #57-77; *lane E*, 10 μg of 8000-year-old DNA from peat near bone deposits. Rows 2 to 5 contain serial 2-fold dilutions of sample in row 1.

or human ribosomal genes. The inserts from three clones exhibiting weakly positive hybridization signals were sequenced. None showed significant (> 70%) homology to any of the three classes of target genes nor did these inserts (392 bp total) possess homology to any known DNA sequence by computer analysis of the Genetic Sequence Data Bank maintained by the NIH. This may not be surprising since only a small fraction (< 1%) of the human genome has been sequenced.

An alternative approach to demonstrating the potential human origin of some cloned fragments involved hybridizing Southern blots of modern human brain DNA with probes made from the cloned Alu inserts described above. Twelve randomly selected probes were made and hybridized. In two instances discrete bands of modern human DNA hybridized suggesting that at least a portion of the old brain DNA is of human origin. Again, the precise identity of the sequences remains obscure.

Discussion

TABLE 4. Sites where brain tissues have been found

Locations	*Dates*	*Conditions*[a]	*Reference*
Fort St. Marks, FL	1818 – 1819 A.D.	4	Dailey et al. 1972
Kagamill Island, AK	1740 A.D.	2	Zimmerman et al. 1971
Denmark	1236 – 1540 A.D.	4	Tkocz et al. 1979
Chihuahua, Mexico	1040 – 1260 A.D.	2	Luibel – Hulen 1985
Denmark (Grauballe Man)	210 – 410 A.D.	3	Glob 1969
Lindow Moss, England	211 ±80 A.D.	–	Stead and Turner 1985
Droitwich, England	200 A.D.	4	Oakley 1960
Holland (Windeby Girl)	100 A.D.	3	Glob 1969
Egypt (PUMIV)	100 B.C. – 100 A.D.	1	Reyman and Peck 1980
Denmark (Tolland Man)	210 B.C. ±40	3	Glob 1969; Fischer 1980
Egypt (#1981/575)	332 – 30 B.C.	1	David and Tapp 1984
Borremosse, Denmark	430 B.C. ±100	3	Glob 1969
Borremosse, Denmark	650 B.C. ±80	3	Glob 1969
Egypt (POM III)	835 B.C.	1	Riddle 1980
Egypt (PUM III)	835 B.C.	1	Reyman and Peck 1980
Egypt (ROM I)	1000 B.C.	1	Millet et al. 1980
Egypt (Tetisheri)	1600 – 1570 B.C.	1	Harris and Weeks 1973
Egypt (Salford head)	–	1	David and Tapp 1984
Egypt (#22940)	–	1	David and Tapp 1984
Egypt	3000 B.C. – 600 A.D.	1, 2	Smith 1902
Peru	–	–	Allison and Gersztén 1982
Bay West, FL	6400 – 6900 B.P.	4	Beriault et al. 1981
Little Salt Spring, FL	8000 B.P.	4	Clausen et al. 1979
Windover, FL	8000 B.P.	4	Doran et al. 1986
Warm Mineral Springs, FL	10000 B.P. (est.)	4	Royal and Clark 1960

a. 1, artificial mummy; 2, natural mummy; 3, peat bog; 4, damp or water-saturate environment

The Windover site yielded preserved brain tissue of human origin dating to the Early Archaic period. It is the oldest human soft tissue yet analyzed at a molecular level. An interdisciplinary approach demonstrated the presence of human DNA as well as remnant cellular and anatomical structure. These observations raise questions in two broad categories: first, what factors resulted in the preservation at Windover and second, what type of information does the presence of ancient DNA make available to anthropology, molecular biology, and other disciplines?

Examples of human soft tissue preservation are prevalent but discovery of preserved brain material at first appeared unique, since the brain can undergo rapid autolysis (self-disintegration of cellular components) (Becker and Barron 1961). However, brain material may remain as a recognizable mass after all other soft tissue has disappeared (Bass 1984) and several instances of preserved brain material in an archeological setting have been reported (Table 4). Soft tissue preservation can occur in a variety of environments ranging from water-saturated to extremely dry or cold. Instances of preserved human tissue as old as the Windover material are less common. Most examples of human soft tissue preservation are from hot, dry environments. Some of these have been subjected to elaborate postmortem treatment (Allison 1985; Peck 1980; Pretty and Calder 1980). Egyptian mummies (2686 B.C. to A.D. 641) are the best known examples of complex postmortem treatment. The postmortem process generally involved dehydrating the body chemically with dry natron, a naturally occurring mixture of Na_2CO_3, $NaHCO_3$, and NaCl or Na_2SO_4 and then coating the body with resins (Harris and Weeks 1973:81–92; Tapp 1984; Peck 1980).

Bodies, however, can also be preserved in hot, dry environments through accidental or intentional exploitation of natural desiccation. In these instances, the climate must be dry enough that dehydration of the bodies is rapid. Furthermore, dry conditions must persist up to the time of discovery. Examples of this type of preservation are found in predynastic Egypt (before 2686 B.C.) (Smith 1902), Peru (Vreeland and Cockburn 1980), the American Southwest (El-Najjar and Mulinski 1980), Australia (Pretty and Calder 1980) and other areas (Ascenzi et al. 1980).

Other mummified or frozen remains have been found in cold environments ranging from the high-altitude desert environments of Peru and northern Asia to the arctic areas of Alaska and Greenland (Artamonov 1965; Zimmerman and Smith 1975; Hansen et al. 1985; Dekin 1987). Under these conditions dehydration may have taken place by sublimation of body moisture (Vreeland and Cockburn 1980). The bone and soft tissue in these samples are frequently in an excellent state of preservation (Vreeland and Cockburn 1980; Hansen et al. 1985).

A variety of aqueous environments have yielded preserved tissue. The highly acidic (pH < 4) peat bogs of northern Europe have produced human remains with a remarkable amount of tissue preservation (Glob 1969; Fischer 1980). Skin and hair are intact (essentially tanned), internal organ preservation is less predictable, and bone is usually highly demineralized; less acidic conditions yield better preserved bone (Glob 1969).

Ancient human remains from damp environments or nonacidic, water-saturated environments like Windover are rarer, but have occurred. Human skulls containing the apparent remnants of brain tissue were found earlier at several Florida sites (Royal and Clark 1960; Dailey et al. 1972; Clausen et al. 1979; Beriault et al. 1981). A Danish medieval cemetery yielded 56 of 74 skulls with brain material; like the Windover site no other soft tissue was preserved (Tkocz et al. 1979). Most of the bog bodies of northern Europe are from acidic environments but some are found under less acidic conditions (pH 5–7.5), and in these cases the body is found as skeleton or adipocere (Fischer 1980).

Upon comparing descriptions of preserved tissue found at other archeological or forensic sites with the material found at Windover, we note that specimens and locations most similar to Windover came from sites where adipocere formation occurred. Adipocere (also known as "grave wax") is a mixture of free fatty acids, primarily palmitic acid, and soaps resulting from the postmortem hydrolysis and hydrogenation of fats present in naturally occurring fat tissue in the body (Mant 1957; Zivanovic 1982:18–19). Damp conditions, fatty tissue, electrolytes (which may come from body fluids), and some putrification (to initiate hydrolysis) must be present for adipocere formation to occur (Mant 1957). Apparently at Windover, burial practices and physical and chemical conditions allowed putrification to begin but the process was halted before complete decomposition of brain tissue occurred.

The conditions at Windover that appear most likely to enhance tissue preservation by inhibiting bacterial growth are the high sulfur levels in water and peat, the high amounts of minerals present in the water, and the anaerobic conditions which begin 30 cm below the peat surface. DNA within the tissue was preserved owing to at least two other factors: first, water at Windover is nearly neutral (pH 5.3–6.8), particularly in the red-brown peat stratum; second, the anaerobic property of the water limits oxidative DNA damage. Thus, DNA alteration due to acid depurination, deamination, and oxidation was minimized. Interestingly, a low temperature may not have been a factor in DNA preservation since modern subsurface ground water temperatures are around 23°C. Although DNA has been isolated from tissues preserved through rapid drying (Pääbo 1985; Higuchi et al. 1984; Rogers and Bendich 1985; Johnson et al. 1985), the present results show that tissues recovered from water-saturated environments under conditions of anaerobia, neutral pH, and high ion levels also yield preserved DNA.

In an archeological setting, DNA survival not only includes chemical factors but also ethnological practices that may act to influence burial conditions and, thus, rate of tissue decomposition and DNA survival. Individual differences in tissue integrity may reflect variations in either burial environment or the interval and conditions which prevailed between death and interment, or both. Windover skeletal material buried along the deeper edge of the pond was well preserved, but was disarticulated and appears to have been slowly transported down slope. Burials in the more shallow edge of the pond, found in the third and final field season, were articulated but poorly preserved. There is no indication of secondary burial at Windover, although the practice was widespread in later New World populations (Ubelaker 1974; O'Shea 1984; Churcher and Kenyon 1960). The interment pattern resembles Floridian aquatic burial practices occurring both earlier and later than the Windover site, possibly reflecting a long-term or recurring religious theme in Florida (Royal and Clark 1960; Clausen et al. 1975; Beriault et al. 1981; Wharton et al. 1981; Sears 1982). It is clear that rapid interment is a necessary factor in tissue preservation in environments promoting rapid decomposition. Preservation may also reflect sex- and age-specific burial patterns. However, at Windover, brain material has been recovered from infants, adolescents, young and older adults representing both sexes, thus indicating little status distinction in burial patterns. Agreement between the ages of the peat surrounding the skeletal material and the bone itself also suggests primary burials in shallow graves. There are some indications that the bodies may have been deposited in water deep enough to require pointed "hold down" stakes and stakes of unmodified wood. We suggest, therefore, that in temperate latitudes of the New World, rapid, simple burial practices in an anaerobic, water-saturated matrix may be an important factor in soft tissue and DNA preservation.

Nucleic acids (especially mtDNA) recovered from prehistoric populations could prove enormously useful in anthropological studies of population genetics. Several researchers maintain reservations about the reliability of mtDNA for cladistic studies (branching relationships) (Jones and Rouhani 1986; Slatkin 1987; Vawter and Brown 1986; Wainscoat 1987; Wainscoat et al. 1986). However, several teams have suggested that restriction maps of mtDNA may be useful for the study of cladistic relationships, timing of divergence, and investigations of multiple vs. single origins of populations. The characteristics that make mtDNA studies attractive are its high mutation rate (estimated 10 times that of nuclear DNA) (Cann et al. 1984; Cann and Wilson 1983; Wainscoat 1987) and its uniparental and haploid pattern of inheritance (Cann et al. 1987; Greenberg et al. 1983; Johnson et al. 1983; Whittam et al. 1986; Wilson et al. 1985). Several pilot studies have used mtDNA data to investigate the place and timing of the origin of modern *Homo sapiens sapiens* (Cann et al. 1987; Denaro et al. 1981; Greenberg et al. 1983; Johnson et al. 1983). Other anthropological studies have investigated southwestern United States Amerind and Asian diversity and relationships (Wallace et al. 1985) and genetic homogeneity of worldwide Jewish populations (Meyers 1985).

While there is reason for caution in the interpretation of mtDNA data (e.g., see Wainscoat 1987), there is little doubt that prehistoric mtDNA from ancient Amerind and other populations would be an extraordinary find if intact enough for investigating gene flow, bottle necks and population divergence. Although it remains uncertain whether the Windover material will provide the preservation needed for these types of studies, the preservation of mtDNA in this soft tissue suggests mtDNA may also be found in tissues from other archeological sites.

Literature cited

Adams, R.D., and J.C. Lee. 1982. In W. Haymaker and R.D. Adams, eds., *Histology and Histopathy of the Nervous System*, 234–237. Springfield, Ill.: Charles C Thomas.

Allison, M.J. 1985. Chile's Ancient Mummies. *Natural History*, 94:75–80.

Allison, M.J., and E. Gerszten. 1982. *Paleopathology in South American Mummies: Application of Modern Techniques*. Richmond: Medical College of Virginia.

Anderson, S., A.T. Bankier, B.G. Barrell, M.H.L. deBruijn, A.R. Coulson, J. Drouin, I.C. Eperon, D.P. Nierlich, B.A. Roe, F. Sanger, P.H. Schreier, A.J.H. Smith, R. Staden, and I.G. Young. 1981. Sequence and Organization of the Human Mitochondrial Genome. *Nature*, 290:457–465.

Andrews, R.L., and J.M. Adovasio. 1988. Textile and Related Perishable Remains from the Windover Site. Paper presented at the 53d annual meeting of the Society of American Archaeologists, Phoenix, Arizona.

Artamonov, M.I. 1965. Frozen Tombs of the Scythians. *Scientific American*, 212:101–109.

Ascenzi, A., A. Cockburn, and E. Kleiss. 1980. Miscellaneous Mummies. In A.C. Cockburn and E. Cockburn, eds., *Mummies, Disease and Ancient Cultures*, 224–238. Cambridge, U.K.: Cambridge University Press.

Bada, J.L. 1985. Aspartic Acid Racemization Ages of California Paleoindian Skeletons. *American Antiquity*, 50:645–647.

Bada, J.L., and P.M. Helfman. 1975. Amino Acid Racemization Dating of Fossil Bones. *World Archaeology*, 7:160–173.

Bass, W.M. 1984. Time Interval Since Death: A Difficult Decision. In T.A. Rathbun and J.E. Buikstra, eds., *Human Identification: Case Studies in Forensic Anthropology*, 136–147. Springfield, Ill.: Charles C Thomas.

Basset, E. 1982. Osteological Analysis of Carrier Mills Burial. In R. Jeffries and B. Butler, eds., *The Carrier Mills Archaeological Project*. Carbondale: Southern Illinois University Press.

Becker, N.H., and K.D. Barron. 1961. The Cytochemistry of Anoxic and AnoxicIschemic Encephalopathy in Rats: I. Alterations in Neuronal Lysosomes Identified by Acid Phosphatase Activity. *American Journal of Pathology*, 38:161–175.

Berger, R., R. Protsch, R. Reynolds, R. Rozaire, and J.R. Sackett. 1971. New Radiocarbon Dates Based on Bone Collagen of California Paleoindians. *Contributions of the University of California Archaeological Research Facility*, 12:43–49.

Beriault, J., R. Carr, J. Stipp, R. Johnson, and J. Meeder. 1981. The Archaeological Salvage of the Bay West Site, Collier County, Florida. *Florida Anthropologist*, 34:39–58.

Beukens, R.P. 1986. *Radiocarbon Analysis Report*. Toronto: Isotrace Laboratory, University of Toronto.

Black, M., and C.E. Eyman. 1963. The Union Lake Skull, a Possible Early Indian Find in Michigan. *American Journal of Physical Anthropology*, 29:39–48.

Breternitz, D.A., A.C. Swedlund, and D.C. Anderson. 1971. An Early Burial from Gordon Creek, Colorado. *American Antiquity*, 36:170–182.

Cann, R.L., W.M. Brown, and A.C. Wilson. 1984. Polymorphic Sites and the Mechanism of Evolution in Human Mitochondrial DNA. *Genetics*, 106:479–499.

Cann, R.L., M. Stoneking, and A.C. Wilson. 1987. Mitochondrial DNA and Human Evolution. *Nature*, 235:31–36.

Cann, R.L., and A.C. Wilson. 1983. Length Mutations in Human Mitochondrial DNA. *Genetics*, 104:699–711.

Chang, D.D., and D.A. Clayton. 1985. Priming of Human Mitochondrial DNA Replication Occurs at the Light Strand Promoter. *Proceedings of the National Academy of Science*, 82:351–355.

Churcher, C.S., and W.A. Kenyon. 1960. The Tabor Hill Ossuaries: A Study in Iroquois Demography. *Human Biology*, 32:249–273.

Clausen, C.J., H.K. Brooks, and A.B. Wesolowsky. 1975. The Early Man Site at Warm Mineral Springs, Florida. *Journal of Field Archaeology*, 2:191–213.

Clausen, C.J., A.D. Cohen, C. Emiliani, J.A. Holman, and J.J. Stipp. 1979. Little Salt Spring, Florida: A Unique Underwater Site. *Science*, 203:609–614.

Conrad, N., D.L. Asch, N.B. Asch, D. Elmore, H. Gove, M. Rubins, J.A. Brown, M.D. Wiant, K.B. Farnsworth, and T.G. Cook. 1984. Accelerator Radiocarbon Dating of Evidence for Prehistoric Horticulture in Illinois. *Nature*, 308:443–446.

Dailey, R.C., L.R. Morrell, and W.A. Cockrell. 1972. The St. Marks Cemetery (8WA108). *Bureau of Historic Sites and Properties Special Reports*, 2:1–24.

Dekin, A.A., Jr. 1987. When Ice Entombed an Eskimo Family. *National Geographic*, 171:824–836.

Denaro, M., H. Blanc, M.J. Johnson, K.H. Chen, E. Wilmsen, L.L. CavalliSforza, and D.C. Wallace. 1981. Ethnic Variation of Hpa I Endonuclease Cleavage Patterns of Human Mitochondrial DNA. *Genetics*, 78:5768–5772.

Dickel, D.N. 1986. Osteological Remains from the Windover Early Archaic Site, Florida. Paper presented at the annual meeting of the American Association of Physical Anthropologists, Albuquerque, New Mexico.

———. 1987. A Case of Neural Tube Defects and Associated Skeletal Pathologies from Windover. Paper presented at the annual meeting of the American Association of Physical Anthropologists, New York.

———. In press. Interproximal Grooves in an Early Archaic Population. *American Journal of Physical Anthropology*.

Dickel, D.N., and G.H. Doran. 1989. Severe Neural Tube Defect Syndrome from the Early Archaic of Florida (8BR246). *American Journal of Physical Anthropology*, 80:325–334.

Doran, G.H., D.N. Dickel, W.E. Ballinger, Jr., G.F. Agee, P.J. Laipis, and W.W. Hauswirth. 1986. Anatomical, Cellular and Molecular Analysis of 8,000 Year Old Human Brain Tissue from the Windover Archaeological Site. *Nature*, 323:803–806.

El-Najjar, M.Y., and T.M. Mulinski. 1980. Mummies and Mummification Practices in the Southwestern and Southern United States. In A.C. Cockburn and E. Cockburn, eds., *Mummies, Disease and Ancient Cultures*, 103–117. Cambridge, U.K.: Cambridge University Press.

Fischer, C. 1980. Bog Bodies of Denmark. In A.C. Cockburn and E. Cockburn, eds., *Mummies, Disease and Ancient Cultures*, 177–193. Cambridge, U.K.: Cambridge University Press.

Glob, P.V. 1969. *The Bog People: Iron-age Man Preserved*. London: Faber and Faber.

Goffin, C., S. Bricteux-Gregoire, and W.G. Verly. 1984. Some Properties of the Interstrand Crosslinks in Depurinated DNA. *Biochemica and Biophysica Acta*, 783:1–5.

Greenberg, B.D., J.E. Newbold, and A. Sugino. 1983 Intraspecific Nucleotide Sequence Variability Surrounding the Origin of Replication in Human Mitochondrial DNA. *Gene*, 21:33–49.

Hansen, J.P.H., J. Meldgaard, and J. Nordqvist. 1985. The Mummies of Qilakitsoq. *National Geographic*, 167:191–207.

Harris, J.E., and K.R. Weeks. 1973. *X-raying the Pharaohs*. New York: Charles Scribner's Sons.

Higuchi, R., B. Bowman, M. Freiberger, O.A. Ryder, and A.C. Wilson. 1984. DNA Sequences from the Quagga, an Extinct Member of the Horse Family. *Nature*, 312:282–284.

Houck, C.M., F.P. Rinehart, and C.W. Schmid 1979 A Ubiquitous Family of Repeated DNA Sequences in the Human Genome. *Journal of Molecular Biology*, 132:289–306.

Ike, D., J.L. Bada, P.M. Masters, G.K. Vogel, and J.C. Vogel. 1979. Aspartic Acid Racemization and Radiocarbon Dating of an Early Milling Stone Horizon Burial in California. *American Antiquity*, 44:524–530.

Johnson, M.J., D.C. Wallace, S.D. Ferris, M.C. Rattazzi, and L.L. CavalliSforza. 1983. Radiation of Human Mitochondria DNA Types Analyzed by Restriction Endonuclease Cleavage Patterns. *Journal of Molecular Evolution*, 19:255–271.

Johnson, P.H., C.B. Olson, and M. Goodman. 1985. Isolation and Characterization of Deoxyribonucleic Acid from Tissue of the Wooly Mammoth *(Mammus primigenius)*. *Comparative Biochemical Physiology*, 81B:1045–1051.

Jones, J.S., and S. Rouhani. 1986. How Small was the Bottleneck? *Nature*, 319:449–450.

Kaufman, T.S. 1980. Early Prehistory of the Clean Lake Area, Lake County, California. Ph.D. dissertation, Department of Anthropology, University of California, Los Angeles.

Klein, J., J.C. Lerman, P.E. Damon, and E.K. Ralph. 1982. Calibration of Radiocarbon Dates: Tables Based on Consensus Data of the Workshop on Calibrating the Radiocarbon Time Scale. *Radiocarbon*, 24:103–150.

Lewis, T.M.N., and M.K. Lewis. 1961. *Eva: An Archaic Site*. Knoxville: University of Tennessee Press.

Lien, P.M. 1983. Amino Acid Racemization Dates from Paleo-Indian Sites in Florida. *Florida Anthropologist*, 36:106–107.

Long, E.O., and I.B. Dawid. 1980. Repeated Genes in Eucaryotes. *Annual Review of Biochemistry*, 49:727–764.

Luibel-Hulen, A.M. 1985. Use of Computer Assisted Tomography in the Study of a Female Mummy from Chihuahua, Mexico. *San Diego Museum Papers*, 19:34–36.

Luna, L.G. 1968. *Manual of Histological Staining Methods of the Armed Forces Institute of Pathology*. 3d edition. New York: McGraw-Hill.

Mant, A.K. 1957. Adipocere—A Review. *Journal of Forensic Medicine*, 4:18–35.

Mason, R.J., and C. Irwin. 1960. An Eden-Scottsbluff Burial in Northeastern Wisconsin. *American Antiquity*, 26:43–57.

Meyers, N. 1985. Genetic Links for Scattered Jews. *Nature*, 314:208.

Milanich, J.T., and C.W. Fairbanks. 1980. *Florida Archaeology*. New York: Academic Press.

Millet, N.B., G.D. Hart, T.A. Reyman, M.R. Zimmerman, and P.K. Lewin. 1980. ROM I: Mummification for the Common People. In A.C. Cockburn and E. Cockburn, eds., *Mummies, Disease and Ancient Cultures*, 71–84. Cambridge, U.K.: Cambridge University Press.

Neumann, H.W. 1967. The Paleopathology of the Archaic Modoc Rock Shelter Inhabitants. *Reports of Investigations*, No. 11. Springfield, Ill.: Illinois State Museum.

Oakley, K.P. 1960. Ancient Preserved Brains. *Man*, 60:122–123.

O'Shea, J.M. 1984. *Mortuary Variability: An Archeological Investigation*. New York: Academic Press.

Owen, R.C. 1964. Early Milling Stone Horizon (Oak Grove), Santa Barbara County, California: Radiocarbon Dates. *American Antiquity*, 30:210–213.

Pääbo, S. 1985. Molecular Cloning of Ancient Egyptian Mummy DNA. *Nature*, 314:644–645.

Peck, W.H. 1980. Mummies of Ancient Egypt. In A.C. Cockburn and E. Cockburn, eds., *Mummies, Disease and Ancient Cultures*, 11–28. Cambridge, U.K.: Cambridge University Press. Pretty, G.L., and A. Calder. 1980. Mummification in Australia and Melanesia. In A.C. Cockburn and E. Cockburn, eds., *Mummies, Disease and Ancient Cultures*, 194–210. Cambridge, U.K.: Cambridge University Press.

Protsch, R.R. 1978. *Catalogue of Fossil Hominids of North America*. New York: Gustav Fisher.

Reyman, T.A., and W.H. Peck. 1980. Egyptian Mummification with Evisceration per Ano. In A.C. Cockburn and E. Cockburn, eds., *Mummies, Disease and Ancient Cultures,* 85–100. Cambridge, U.K.: Cambridge University Press.

Riddle, J.M. 1980. A Survey of Ancient Specimens by Electron Microscopy. In A.C. Cockburn and E. Cockburn, eds., *Mummies, Disease and Ancient Cultures,* 274–286. Cambridge, U.K.: Cambridge University Press.

Rogers, S.O., and A.J. Bendich. 1985. Extraction of DNA from Milligram Amounts of Fresh, Herbarium and Mummified Plant Tissues. *Plant Molecular Biology,* 5:69–76.

Royal, W., and E. Clark. 1960. Natural Preservation of Human Brain, Warm Mineral Springs, Florida. *American Antiquity,* 26:285–287.

Sears, W. 1982. Fort Center, an Archaeological Site in the Lake Okeechobee Basin. *Ripley P. Bulletin Monographs in Anthropology and History,* 4. Florida State Museum, Gainesville: University Presses of Florida.

Sheppard, J.C., P.E. Wigand, C.E. Gustafson, and M. Rubin. 1987. A Reevaluation of the Marmes Rockshelter Radiocarbon Chronology. *American Antiquity,* 52:118–125.

Slatkin, M. 1987. Gene Flow and the Geographic Structure of Natural Populations. *Science,* 236:787–792.

Smith, G.E. 1902. On the Natural Preservation of the Brain in the Ancient Egyptians. *Journal of Anatomy and Physiology,* 36:375–381.

Stead, I., and R.C. Turner. 1985. Lindow Man. *Antiquity,* 225:25–29.

Tapp, E. 1984. In R. David and E. Tapp, eds., *Evidence Embalmed: Modern Medicine and the Mummies of Ancient Egypt,* 78–95. Manchester, U.K.: Manchester University Press.

Taubold, R.D., A.N. Siakotos, and E.G. Perkins. 1975. Studies on the Chemical Nature of Lipofuscin (Age Pigment) Isolated from Normal Human Brain. *Lipids,* 10:383–390.

Taylor, D.C. 1969. The Wilsall Excavations: An Exercise in Frustration. *Proceedings of the Montana Academy of Science,* 29:147–150.

Taylor, R.E., L.A. Payen, C.A. Prior, P.J. Slota Jr., R. Gillespie, J.A. Gowlett, R.E.M. Hedges, A.J.T. Juli, T.H. Zabel, D.J. Donahue, and R. Berger. 1985. Major Revisions in the Pleistocene Age Assignments for North American Human Skeletons by C–14 Accelerator Mass Spectrometry: None Older than 11,000 C–14 Years BP. *American Antiquity,* 50:136–140.

Tkocz, I., P. Bytzer, and F. Bierring. 1979. Preserved Brains in Medieval Skulls. *American Journal of Physical Anthropology,* 51:197–202.

Ubelaker, D.H. 1974. Reconstruction of Demographic Profiles from Ossuary Skeletal Samples: A Case Study from the Tidewater Potomac. *Smithsonian Contributions to Anthropology,* 18. Washington D.C.: Smithsonian Institution Press.

Vawter, L., and W.M. Brown. 1986. Nuclear and Mitochondrial DNA Comparisons Reveal Extreme Rate Variation in the Molecular Clock. *Science,* 234:194–196.

Vinograd, J., J. Lebowitz, R. Radloff, R. Watson, and P. Laipis. 1965. The Twisted Circular Form of Polyoma Viral DNA. *Proceedings of the National Academy of Sciences,* 53:1104–1111.

Vreeland, J.M., and A. Cockburn. 1980. Mummies of Peru. In A.C. Cockburn and E. Cockburn, eds., *Mummies, Disease and Ancient Cultures,* 135–176. Cambridge, U.K.: Cambridge University Press.

Wainscoat, J.S. 1987. Out of the Garden of Eden. *Nature,* 325:13.

Wainscoat, J.S., A.V.S. Hill, A.L. Boyce, J. Flint, M. Hernandez, S.L. Thein, J.M. Old, J.R. Lynch, A.G. Falusi, D.J. Weatherall, and J.B. Clegg. 1986. Evolutionary Relationships of Human Populations from an Analysis of Nuclear DNA Polymorphisms. *Nature,* 319:491–493.

Wallace, D.C., K. Garrison, and W.C. Knowler. 1985. Dramatic Founder Effects in Amerindian Mitochondrial DNAs. *American Journal of Physical Anthropology,* 68:146–155.

Waters, M.R. 1985. Early Holocene Occupation Along White Water Draw, Southeastern Arizona: The Sulphur Spring Stage. *Current Research in the Pleistocene,* 2:31–33.

______. 1986. Sulphur Springs Woman: An Early Human Skeleton from Southeastern Arizona. *American Antiquity,* 51:361–365.

Weir, F.A. 1985. An Early Holocene Burial at the Wilson-Leonard Site in Central Texas. *Mammoth Trumpet,* 2(1):2–3.

Wendorf, F., A.D. Krieger, and C.E. Albritton. 1955. *The Midland Discovery: A Report of the Pleistocene Human Remains from Midland, Texas.* Austin: University of Texas Press.

Wharton, B.R., G.R. Ballo, and M.E. Hope. 1981. The Republic Groves Site, Hardee County, Florida. *Florida Anthropologist,* 34:59–80.

Whittam, T.S., A.G. Clark, M. Stoneking, R.L. Cann, and A.C. Wilson. 1986. Allelic Variation in Human Mitochondrial Genes Based on Patterns of Restriction Site Polymorphism. *Proceedings of the National Academy of Sciences,* 83:9611–9615.

Wilson, A.C., R.L. Cann, S.M. Carr, M. George, U.B. Gyllensten, K.M. Helm-Bychowski, R.G. Higuchi, S.R. Palumbi, E.M. Prager, R.D. Sage, and M. Stoneking. 1985. Mitochondrial DNA and Two Perspectives on Evolutionary Genetics. *Biological Journal of the Linnean Society,* 26:375–400.

Young, D.E. 1985. The Paleoindian Skeletal Material from Horn Rock Shelter in Central Texas. *Current Research in the Pleistocene,* 2:39–40.

______. 1986. The Paleoindian Skeletal Material from Horn Shelter, Number 2 in Central Texas, an Analysis and Perspective. Master's thesis, Texas A & M University, College Station.

Zimmerman, M.R., and G.S. Smith. 1975. A Probable Case of Accidental Inhumation of 1,600 Years Ago. *Bulletin of the New York Academy of Medicine,* 51:828–837.

Zimmerman, M.R., G.W. Yeatman, and H. Sprinz. 1971. Examination of an Aleutian Mummy. *Bulletin of the New York Academy of Medicine,* 47:80–103.

Zivanovic, S. 1982. *Ancient Diseases: The Elements of Paleopathology.* London: Methuen.

SUMMARY OF AUDIENCE DISCUSSION: The specimens were retrieved from peat moss in water at neutral pH. Tissue sections demonstrated no paleopathology. Immunoglobulin may be present and will be sought. Blood types and most any genetic parameter may be predictable.

Diagnosis of thalassemia in ancient bones: Problems and prospects in pathology

Antonio Ascenzi, A. Bellelli, M. Brunori, G. Citro, R. Ippoliti,
E. Lendaro, and R. Zito

"Porotic hyperostosis" is a generic term applied to a type of bone lesion characterized by a symmetrically distributed increase in the volume of the skeleton, associated with a reduction of the bone texture. Such a type of lesion was recognized for the first time by Cooley and Lee (1925) as a feature peculiar to thalassemia. Subsequent investigations (Moseley 1963; Ascenzi 1976,1979) deeply modified the original view, so that nowadays it is quite obvious that porotic hyperostosis can be induced by any disease that leads to an increase in bone marrow volume, causing the skeleton to adapt its capacity to contain the excess of hemopoietic marrow. The most profound changes of porotic hyperostosis are seen in children, and they diminish as the individual approaches adult life (Caffey 1951). This agrees with Neumann's law in which half of the bone marrow is adipose in the adult, while the entire long bone marrow is hemopoietic in the child. Because of this, the adult can double the volume of hemopoietic marrow without any change to the skeleton; on the other hand, even a limited hyperplasia of the hemopoietic marrow induces in the subadult a volumetric increase in the bone marrow. A list of conditions inducing porotic hyperostosis has been compiled by Moseley (1965), and the subject has been reviewed by Ascenzi (1976,1979). In theory, any condition that abnormally increases the rate of blood cell turnover can produce porotic hyperostosis. Diseases known to do this include: congenital hemolytic anemias (thalassemia, sickle cell disease, hereditary spherocytosis, hereditary elliptocytosis, hereditary nonspherocytic hemolytic anemia), iron deficiency anemia, cyanotic congenital heart disease, polycythemia vera in childhood.

From what is reported above it may be inferred that, in paleopathologic terms, porotic hyperostosis is the only suitable evidence for the existence of medullary hyperplasia, either primary or secondary, when the skeleton is the only tissue which time has preserved. However, skeletal remains showing porotic hyperostosis have been unable so far to provide unequivocal information on the specific disease syndrome which led to bone lesions.

Starting with these premises, we attempted to remove ambiguities for the diagnosis of thalassemia in skeletal remains by examining the possibility that hemoglobin arising from postmortem lysis of the erythrocytes may remain adsorbed to the bone and be specifically detected. In a previous paper (Ascenzi et al. 1985) we provided evidence that hemoglobin is indeed measurable in skeletal remains dating back to Eneolytic age using an immunochemical technique. This discovery encouraged further investigations, and in this paper we present additional progress toward an unequivocal diagnosis of thalassemia in skeletal remains with porotic hyperostosis.

Materials and methods

Samples (lumbar vertebrae, skulls and other bones) were obtained from the Verano cemetery of Rome (samples buried for 15 to 30 years), from the S. Senatore Catacumbae in Albano Laziale, Rome (samples dating back from the first to third century of this era), from the Necropolis of Porto (Isola Sacra, Rome, second century B.C.) and of Castiglione (Rome, 1000 B.C.).

Bones were frozen in liquid nitrogen and crushed into powder with a hydraulic press. The powder was extracted with 6M urea for 4 hours at room temperature and neutral pH. The extract was filtered and urea was removed by means of gel permeation chromatography on Sephadex G25 (Pharmacia, Sweden).

Antiserum against human apohemoglobin (globin) was raised in adult male rabbits with three subcutaneous inoculations of 0.8 mg of the antigen in complete Freund adjuvant. Hemoglobin constituent alpha and beta chains were prepared as by Bucci and Fronticelli (1965) and antisera against the two purified chains were obtained following the same procedure. The content of hemoglobin remnants in bone extracts was determined with the immunoblot technique employing a Bio Dot apparatus (BioRad, U.S.A.); solutions of apohemoglobin, alpha and beta chains were employed as stan-

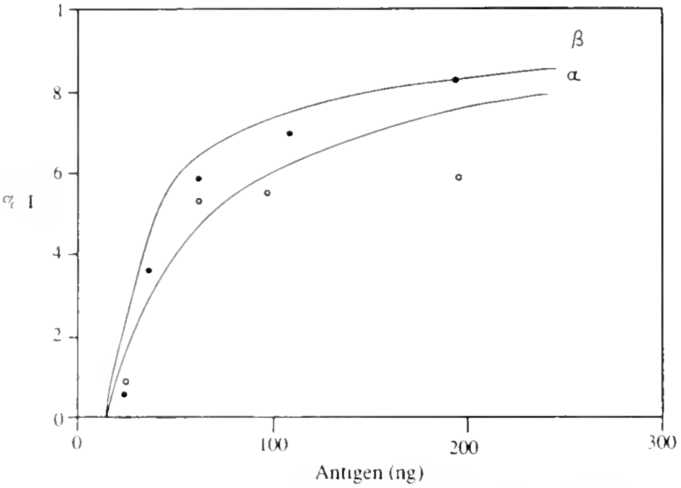


FIGURE 1. Reaction of antiglobin rabbit antiserum against human alpha and beta chains.

dards. Quantification was achieved by integration of dot intensity using a LKB Laser Scanner densitometer and digital integration.

Results

Apohemoglobin (globin), alpha chains and beta chains were used to standardize rabbit antisera raised against human globin. The results of one experiment illustrating this procedure are shown in Figure 1.

Lumbar vertebrae extracts were tested with the same antiserum and the content of hemoglobin in the skeletal remains estimated by comparison with the standard curves. As already shown qualitatively (Ascenzi et al. 1985), hemoglobin or hemoglobin fragments can be determined quantitatively in bone extracts with this technique. The average hemoglobin content of lumbar vertebrae from adults buried for 15 years is 0.7 to 1 μg/100 gram of dry powder (Table 1).

A larger amount of titratable material is extracted from the lumbar vertebrae of younger individuals. As a general rule, the younger the individual, the higher the hemoglobin content of the bone extracts, as may have been anticipated.

Skull and other bones were used to determine the correlation between hemoglobin content and anatomy and physiology of the skeleton. It was found that, for the same individual, the absolute amount of hemoglobin detected from bone powder varies markedly with the type of bone examined. Highly vascularized bones, whose marrow exhibits high erythropoietic activity, are (as expected) much richer in titratable hemoglobin than bones with low or absent erythropoietic activity. Data in Table 1 report one example by comparing the hemoglobin content of the lumbar vertebrae and the skull of an infant.

TABLE 1. Content of hemoglobin (or hemoglobin fragments) in lumbar vertebrae extracts (except D2 = skull); quantitation with antiglobin rabbit antiserum

Sample	*Age of subject*	*Time elapsed from death*	*Hb μg/100 g bone () = No. of tests*
A	senescent	15 years	0.16–0.2 (3)
B	adult	15 years	0.9–1.0 (3)
C	adult	15 years	0.7–1.0 (3)
D1	infant	15 years	1.1–1.6 (3)
D2	infant	15 years	0.2–0.4 (3)

TABLE 2. Content of hemoglobin (or hemoglobin fragments) in lumbar vertebrae of adults at different times after death (in years)

Sample	*Time elapsed from death*	*Hb μg/100 g bone () = No. of tests*
A	15 years	0.7–1.0 (3)
B	15 years	0.9–1.0 (3)
C	30 years	0.6–1.0 (3)
D	2000 years	0.6–0.9 (3)

These results are in complete agreement with the known correlation between the age of the subject and erythropoietic activity, since in very young individuals all the skeleton houses active marrow. Surprisingly, the amount of hemoglobin detected in lumbar vertebrae of adults seems to be essentially independent of the time elapsed from burial, at least for the homogeneous set of data reported in Table 2. Because very ancient samples are rare and some bones are more easily destroyed, the oldest specimens consisted only of lumbar vertebrae and skulls; nevertheless similar dependence of hemoglobin content on age at death and anatomy was demonstrated (data not shown).

The reactivity of antialpha and antibeta rabbit antisera was standardized as described above, and the content of alpha chains in bone extracts was determined. Due to some residual cross-reactivity, only a semiquantitative estimate is reported; however, it was clearly observed that both chains can be detected in ancient skeletal remains, and are present in significant amount.

This is particularly important in the case of the alpha chains, in view of the known sensitivity of these polypeptides to proteolytic degradation (Chalevelakis et al. 1975), which had raised concern that it might be difficult to determine quantitatively the content of each chain in ancient skeletal specimens.

Discussion

The results described above demonstrate that the immunochemical technique employed in this work is sufficiently sensitive and reliable to allow quantitative titration of hemoglobin and its constituent polypeptide chains in bone extracts, even some thousands of years after burial of the individual. It is important that, as expected, the results indicate that bones with higher erythropoietic activity contain greater amounts of titratable hemoglobin or hemoglobin fragments than those characterized by reduced erythropoiesis. Moreover it is clear from data in Table 2 that, within the accuracy of the methodology, no significant decrease of hemoglobin content is observed with the archeological age of the specimen, within a range of 15–2000 years. Previous results (Ascenzi et al. 1985) indicated a loss of immunochemically titratable hemoglobin with time of burial, but it may be that characteristics of the soil, humidity, or other interferences may be more important than age after death in determining the final content of detectable protein. It should moreover be emphasized that some uncertainties about the quantitative estimate of hemoglobin in bone extracts remain, owing to the interferences by false positive immunochemical reactions.

As an example, it may be mentioned that two samples of lumbar vertebrae of adult humans from different burial sites (both approximately 2000 years old) were tested against an affinity chromatography purified antialpha chain Ig fraction of the rabbit antiserum as well as against the antialpha depleted fraction of the same serum. The hemoglobin content in the two tests was comparable (1–3 μg/100 g), while the nonhemoglobin reactivity was zero in one specimen and almost eight times higher than that of hemoglobin in the other.

Up to now, the immunochemical technique has been applied only to morphologically normal bones, but it may be possible to determine the content of hemoglobin remnants even in bones whose morphology indicates hyperactive erythropoiesis. As reported above, differential diagnosis of chronic anemias with increased but insufficient compensatory erythropoiesis is usually impossible on the mere anatomical evidence of porotic hyperostosis. In this respect, the immunochemical technique employed here represents a possible tool for an unequivocal diagnosis of alpha and beta thalassemias (and possibly other hemoglobinopathies) in very old skeletal remains. Further work along these lines is in progress.

Literature cited

Ascenzi, A. 1976. Physiological Relationship and Pathological Interferences Between Bone Tissue and Marrow. In G.H. Bourne, ed., *The Biochemistry and Physiology of Bone*, 403–444. New York: Academic Press.

———. 1979. A Problem in Paleopathology: The Origin of Thalassemia in Italy. *Virchows Archiv A Pathological Anatomy and Histology*, 384:121–130.

Ascenzi, A., M. Brunori, G. Citro, and R. Zito. 1985. Immunological Detection of Hemoglobin in Bones of Ancient Roman Times and of Iron and Eneolithic Ages. *Proceedings of the National Academy of Sciences*, 82:7170–7172.

Bucci, E., and C. Fronticelli. 1965. A New Method for the Preparation of Alpha and Beta Chains of Human Hemoglobin. *Journal of Biological Chemistry*, 240:PC 551.

Caffey, J. 1951. Cooley's Erythroblastic Anemia. Some Skeletal Findings in Adolescent and Young Adults. *American Journal of Roentgenology and Radium Therapy*, 65:547–560.

Chalevelakis, C., J.B. Clegg, and D.J. Weatherall. 1975. Imbalanced Globin Chain Synthesis in Heterozygous β-Thalassemic Bone Marrow. *Proceedings of the National Academy of Sciences*, 72:3853–3857.

Cooley, T.B., and P. Lee. 1925. Series of Cases of Splenomegaly in Children with Anemia and Peculiar Bone Changes. *Transactions of the American Pediatric Society*, 37:29–30.

Moseley, J.E. 1963. *Bone Changes in Hematologic Disorders (Roentgen Aspects)*. Orlando, Fla.: Grune and Stratton.

———. 1965. The Paleopathologic Riddle of "Symmetrical Osteoporosis." *American Journal of Roentgenology, Radium Therapy and Nuclear Medicine*, 95:135–142.

Summary of audience discussion: Before one can attempt to correlate porotic hyperostosis with the chemical findings of globin patterns common to thalassemia, one must develop laboratory methods not only for alpha and beta chains but also delta and gamma. The work presented in this study used trabecular bone because of its hematopoietic marrow and therefore because hemoglobin was present there in vivo. Unfortunately during interment its porosity also invites diagenetic changes which might alter protein structure. Use of ribs as source material might minimize the diagenetic hazard. Dental pulp is even more sheltered from the environment. Conceivably the problem of antibodies against alpha and beta chains also cross-reacting with gamma chains might be prevented by raising antibodies in appropriate animals against an antigen composed only of a peptide, preferably one with a known amino acid sequence; even better would be production of a monoclonal antibody in the usual manner. Considering the small quantity of protein present in fossil material, the use of radioimmunoassay methodology would appear to be desirable.

Trends and perspectives in paleoparasitological research

U.E.C. Confalonieri, L.F. Ferreira, A.J.G. Araújo, M. Chame, and B.M. Ribeiro Filho

This report presents some methodological questions involved with our research on parasites from archeological material in Brazil. Our investigations deal mainly with parasitological findings in human and animal coprolites from South American archeological sites and rarely with mummies, since, for paleoecological and paleoanthropological reasons, mummies are not common in Brazil. A review of helminths in mummified human remains has been presented recently (Horne 1986:4–5).

The first finding of parasites in archeological material was in 1910, when Ruffer found *Schistosoma haematobium* eggs in renal tissue of Egyptian mummies, and paleoparasitology has been growing ever since as a scientific discipline. The term "paleoparasitology" was first used by Jean Baer (1971:317), although he mentioned it only parenthetically, commenting on the study of the coevolution of hosts and parasites. It acquired its definitive meaning after the first paper of Ferreira et al. (1979) and is widely used today to characterize the study of parasitic forms in archeological material. After some decades of research, interesting findings have been obtained, and new questions arise concerning the interpretation of these findings. In this report we relate our experience regarding the use of some methods in paleoparasitological investigation as well as difficulties in the interpretation of the data.

In a recent review, Reinhard et al. (1988) discussed the principal techniques for isolation of parasitic forms from coprolites in soil and fecal deposits from archeological sites, and thus these will not be commented upon here.

In paleoparasitology as well as in paleopathology sensu stricto, the main methodological question is the reliability of the diagnosis of the material. Our experience primarily involves the study of eggs and larvae of intestinal parasitic helminths found in archeological material from South America. These differ to some extent from those in the Old World material (see below).

The methodological issues with which we deal involve three main aspects: (1) identification of the zoological origin of the material found (human or animal?); (2) recognition of the possible morphological alterations in the parasitic forms resulting from the desiccation process in archeological deposits or from other physical and biological events during many centuries; (3) better techniques for studying parasite morphology, aimed toward their specific identification.

It is necessary to stress that the approach to these questions is based on knowledge from zoological and morphological sciences, biometrics, electron microscopy, and biochemistry. We will comment only on analysis of helminths, since the other common intestinal parasites, the protozoans, are poorly preserved and can rarely be found.

The first problem faced by paleoparasitologists is the identification of the origins of coprolite material found free in archeological sites, that is, outside mummified bodies. It must be stressed there is an important difference regarding the contents of parasite-containing archeological sediments from the New World and the Old World. Because the latter sites are mostly historical and urban, the possibility of misdiagnosis lies between human coprolites and fecal material produced by domestic animals. In the American sites, at least those from South America, human coprolites have been found in places which could have been also occupied only by wild animals, since the South American Paleo-Indian did not domesticate animals. Therefore we are doing surveys at the archeological sites in the semi-arid regions of Brazil to increase our knowledge of the morphological aspects and contents of the feces of recent local fauna, basically the same animals as from the prehistoric Holocene. This approach was initiated by Fry (1977:7) and is being used to describe the size, form and contents of fresh animal feces as well as feces naturally and artificially desiccated. With this we intend to prepare a catalogue to serve as a guide for the identification of coprolites. So far the results are encouraging because of the peculiarities of South American fauna in general, particularly at Brazilian excavation sites where there are few large omnivorous or carnivorous animals whose feces could be more easily misidentified as those of human origin.

However, for the assessment of the origin of coprolites we must not forget other parameters, such as their food content as well as parasite composition known to be typical for the human host. Such parameters also include biochemical studies which, although still in their infancy, certainly became an important option after the identification of steroids from 2000-year-old North American coprolites (Lin et al. 1978).

A second problem relates to the possible structural modifications found in parasites contained in fecal material from archeological sites. Under the influence of environmental factors, parasite remains could undergo deformities and/or size modifications making their identification difficult or even impossible. Such physicochemical processes could destroy parasitic forms in the fecal mass; these phenomena seem to be responsible for the scarcity of findings of protozoan cysts in coprolites. The first attempt to solve this problem came from the experimental approach inaugurated by Adamson (1976) when he assessed the persistence of eggs of *Schistosoma* sp. in artificially desiccated tissues in order to evaluate the actual frequency of these findings in Egyptian mummies. This approach was then extended to several normal tissues as well as to soft tissue lesions (tumors, for example) by Zimmerman (1972,1977,1978) in an effort to assess the possibilities of histopathological diagnosis in mummified bodies. More recently we started to study the morphological modifications which occurred in helminth eggs and larvae after artificial desiccation and rehydration of fresh feces using different techniques. We have tested whether helminth eggs, such as those of the nematode genus *Trichuris* sp. (Confalonieri et al. 1985) and ancylostomids (Araújo 1987), whose diagnosis depends not only on qualitative morphological characteristics but also on size variations, would undergo significant alterations in their dimensions.

So far these experiments have demonstrated that the desiccation process does not cause deformities in these biological structures that would hinder an adequate identification.

Finally, detailed morphologic study of parasites found in ancient material should be considered in qualitative as well as in quantitative aspects. In the former case, the differential diagnosis rests on detectable microscopic differences of closely related taxa. For this purpose the best technique is scanning electron microscopy, which can reveal variations in the surface relief of eggs and larvae of helminths. A comparative morphological study using this technique is presently underway in our laboratory. It focuses on larvae of *Ancylostoma duodenale* and *Necator americanus*, the most common human hookworms whose desiccated forms cannot be easily separated with the light microscope.

Diagnosis of some parasites depends on biometric evaluation. This includes the ova of *Trichuris*, the helminth most commonly found in South American coprolites, but also very common in European archeological deposits. These are being studied with some taxonomic techniques, such as the Student t-test, for small samples (Sokal and Rohlf 1969:223). The test was applied to the identification of eggs of this genus from small fragments of coprolites from South American sites (Confalonieri 1988) under circumstances in which morphological criteria to the identification of the fecal material cannot be used. In such cases in which several species of *Trichuris* have overlapping size ranges, including the human *T. trichiura*, the statistical procedure is useful since it indicates in probabilistic terms the possibility for human origin of the material.

We are using, again with *Trichuris* eggs, a new biometric parameter for a better discrimination of the different species. This is the linear regression coefficient between length and width of the eggs, already used by Joyner and Norton (1980) in the specific diagnosis of protozoan oocysts. Thus we can add another variable for a more complete morphological evaluation of the egg of each species. This parameter was shown to be especially useful for the differential diagnosis of eggs of *T. trichiura* and *T.* suis, two sister species commonly associated in archeological material from Europe (Jones 1982).

In summary, advances in techniques and methods applied to paleoparasitological investigation are the result of new approaches from biomedical and zoological sciences. In the future these will provide a greater reliability for identification of parasitic diseases in pre- and protohistorical populations.

Literature cited

Adamson, P.B. 1976. Schistosomiasis in Antiquity. *Medical History*, 20:176–188.

Araújo, A.J.G. 1987. Paleoepidemiologia da Ancilostomose. D.Sc. dissertation, Ensp, Fiocruz, Rio de Janeiro.

Baer, J.G. 1971. *Animal Parasites*. New York: McGraw-Hill.

Confalonieri, U.E.C. 1988. The Use of Statistical Test for the Identification of Helminth Eggs in Coprolites. *Paleopathology Newsletter*, 62:7–8.

Confalonieri, U.E.C., B.M. Ribeiro-Filho, L.F. Ferreira, and A.J.G. Araújo. 1985. The Experimental Approach to Paleoparasitology: Desiccation of *Trichuris trichiura* Eggs. *Paleopathology Newsletter*, 51:9–11.

Ferreira, L.F., A.J.G. Araújo, U.E.C. Confalonieri. 1979. Subsídios para a Paleoparasitologia do Brasil I. Parasitos Encontrados em Coprólitos no Município de Unaí, MG. *Resumos IV Congresso Sociedade Brasileira Parasitologia, Campinas*.

Fry, G.F. 1977. Analysis of Prehistoric Coprolites from Utah. *Anthropological Papers*, vol. 97. University of Utah.

Horne, P.D. 1986. Helminthiasis and Mummified Human Remains. Abstract. 13th annual meeting of the Paleopathology Association, Albuquerque, New Mexico.

Jones, A.K.G. 1982. Human Parasite Remains: Prospects for a Quantitative Approach. In A.R. Hall and H.K. Kenward, eds., Environmental Archeology in the Urban Context. *Council for British Archeology Research Report*, 43:66–70.

Joyner, L.P., and C.C. Norton. 1980. The *Eimeiria acervulina* Complex: Problems of Differentiation of *Eimeria acervulina*, *E. mitis* and *E. mivati*. *Protozoological Abstracts*, 4:45–52.

Lin, D.S., W.E. Connor, L.K. Napton, and R.F. Heizer. 1978. The Steroids of 2000-Year-Old Human Coprolites. *Journal of Lipid Research*, 19:215–221.

Reinhard, K.J., U.E.C. Confalonieri, B. Herrmann, L.F. Ferreira, and A.J.G. Araújo. 1988. Aspects of Paleoparasitological Technique: Recovery of Parasite Eggs from Coprolites and Latrines. *Homo*, 37:217–239.

Ruffer, M.A. 1910. Note on the Presence of "Bilharzia haematobia" in Egyptian Mummies of the Twentieth Dynasty (1250–1000 B.C.). *British Medical Journal*, 1:16.

Sokal, R.R., and F.J. Rohlf. 1969. *Biometry: The Principles and Practice of Statistics in Biological Research*. San Francisco: W.H. Freeman.

Zimmerman, M.R. 1972. Histological Examination of Experimentally Mummified Tissue. *American Journal of Physical Anthropology*, 37:271–280.

______. 1977. An Experimental Study of Mummification Pertinent to the Antiquity of Cancer. *Cancer*, 40:1358–1362.

______. 1978. An Experimental Base for Paleopathologic Diagnosis. *Transactions and Studies, College of Physicians of Philadelphia*, 45:299–305.

Taphonomy of spontaneous ("natural") mummification with applications to the mummies of Venzone, Italy

Arthur C. Aufderheide and Mary L. Aufderheide

Soft tissue preservation ("mummification") of human remains is of more than curious interest. Information of interpretive value applicable to epidemiology, parasitology, anthropology, archeology, and many other fields has been extracted by appropriate laboratory technology applied to such tissues (Cockburn and Cockburn 1980:1–8). In most instances postmortem preservation of soft tissues is the result of specific, anthropogenic efforts directed at such an outcome. Methods have varied from evisceration and desiccation by heat in the Chinchorro culture of northern Chile 8000 years ago (Allison et al. 1984) to the modern techniques using intra-arterial injection of protein-denaturing chemicals.

While the effectiveness of the different methods varies, in most cases the principle of the technique employed is self-evident. However, in certain human mummies no evidence of anthropogenic, conservational effort is apparent, nor are the mechanisms involved in such apparently spontaneous mummification processes obvious (Fornaciari 1982). Some of these are desert burials where the combination of heat and the capillary action of sand are probably the principal elements causing moisture removal from the body before soft tissue dissolution is completed (Cockburn and Cockburn 1980:140). Many of the other cases, however, deal with human bodies interred under conditions not normally expected to conserve soft tissues, such as subterranean tombs frequently carved out of rock, commonly beneath a church or other religious structure (Kleiss 1967:208), hence their collective appellation "catacomb mummies."

This communication identifies the principal mechanisms involved in postmortem soft tissue lysis and the factors potentially available to cause "natural" mummification by retarding or arresting these processes. As an example, we examine these possibilities in the case of spontaneous mummification of a group of human mummies in Venzone, Italy.

The report concludes with identification of specific deficits in our knowledge of such processes, and the research required to provide the information base necessary to understand the biological process of spontaneous mummification.

Mechanisms inhibiting postmortem soft tissue lysis

Normally, dissolution of soft tissues after death is achieved by enzymatic action. These enzymes are derived from three sources: (1) body tissues themselves, principally intracellular enzymes, mostly of lysosomal origin; (2) bacteria, commonly from the colon; and (3) insects from the environment. Properties shared by most enzymes include considerable specificity in the molecular structure of their substrate as well as a high degree of sensitivity to environmental changes of temperature or acidity (pH) and to the presence of heavy metal ions. Furthermore all enzymes need a liquid medium in which to operate. Prevention of postmortem soft tissue lysis ("mummification") in any given situation can therefore be expected to involve one or more of these areas of vulnerability resulting in partial or total inhibition of enzyme action. For example, one of the mentioned factors may produce partial suppression of enzyme action, retarding the dissolution rate sufficiently to permit an arid environment to desiccate the soft tissue and prevent further enzyme activity (Evans 1963:3–22).

THERMAL ACTION

COOLING and FREEZING to preserve perishable foods are part of our everyday experience. Not only the well-publicized mammoths of Siberia (Orslanov et al. 1980:1–5) but also human bodies have been preserved by this method. The latter include Greenland Eskimos (Hansen and Gurtler 1983), the "Prince of el Plomo" mummy from Chile—a nine-year-old sacrificial victim entombed above the frost line on an Andean mountain at an altitude of 5400 m (Mostny 1957; Schobinger 1966), Scythian tribal chiefs (Artamonov 1965) and Arctic expedition members (Beattie 1983; Paddock et al. 1970) buried in tombs dug into permafrost areas. Although most of these were probably continuously frozen, cooling near but not necessarily below the freezing point (as in Arctic summer thaw periods) may inhibit enzyme action at least to the point of profound retardation, as it did in several 650-year-old Inuit bodies from Alaska (Zimmerman and Aufderheide 1984). Of interest here are Micozzi's experimental taphonomy studies demonstrating delayed soft tissue putrefaction at summer outdoor temperatures following a brief period of body freezing immediately after death (although the mechanical effects of freezing actually hastened disarticulation). Micozzi (1986) felt this was the result of substantial intestinal bacterial mortality during the period of freezing.

WARMING the body may also retard putrefactive chemical reactions. Since intestinal bacterial growth in vitro frequently ceases at incubation temperatures only a few degrees above body temperature, the delaying effect of warming may be operative both at the bacterial level as well as creating an environmental temperature substantially deviant from the optimum for some enzymes. Heat generated by intestinal bacterial activity in a living individual is normally dissipated by intestinal wall blood flow. When this cooling flow of blood ceases after death, intra-abdominal temperatures have been shown to rise. Native Aleuts exploited this preserving effect by first heating their deceased tribal leaders' corpses over a fire and then placing them in a cave continuously warmed by a natural volcanic heat source (Alexander 1949; Zimmerman et al. 1981:640) In the southwestern United States, bodies of pre-Columbian North American natives were sometimes buried in stone-lined cists exposed to the hot summer sun. Conceivably, elevated ambient temperature may have been the principal factor in delaying putrefactive enzyme activity until the corpse became very dry (El-Najjar et al. 1985). Such effects probably require substantial temperature elevations since only mild rises, although perhaps inhibiting bacterial growth, may accelerate the proteolytic activity of certain enzymes.

CHEMICAL ACTION

There are few well-documented instances of spontaneous mummification largely due to an environmental ALTERATION OF PH, although this possibility is seldom pursued vigorously by investigators. The well-known tissue-preserving effect of encasing a corpse in highly alkaline, powdered lime testifies to its potential effectiveness. It is conceivable that water percolating through a limestone soil may become sufficiently basic to paralyze enzymatic activity when it saturates a body buried therein.

HEAVY METALS are powerful enzyme poisons. This is, for example, the principal mechanism of lead toxicity in living individuals. Arsenic is so effective that it was used commonly as an intra-arterial injection method of embalming by American morticians until the early part of this century (Snow and Reyman 1977). Arsenic was accumulated during life in the bodies of pre-Columbian natives of northern Chile's Camarones Valley as a result of drinking water from the valley's arsenic-contaminated river. This may have contributed to the excellent state of preservation present in these bodies (M.J. Allison, pers. comm.).

The ABSENCE OF OXYGEN is commonly invoked as an explanation for postmortem soft tissue preservation, though it is difficult to identify a well-controlled, laboratory study establishing this conviction. The astonishing quality of soft tissue preservation in the body of a Chinese noblewoman from 100 B.C. has been attributed to the assumed anoxic tomb environment, though the assignment of oxygen absence as the principal factor in that case was done by exclusion of other apparent possibilities (Wu et al. 1980).

In addition to the action of heavy metals and the chemical methods listed below, occasionally specific ANTIMICROBIAL SUBSTANCES may be present which delay degenerative processes by inhibiting bacterial proliferation. Probably the best known of these is the production of tetracycline by the mold-like bacteria *Streptomyces*. Ingestion of this antibiotic by a living individual for infection control has been found to cause a specific, fluorescent staining of bone collagen. Detection of a similar staining pattern in archeological (Nubian) bones (believed to be the result of eating *Streptomyces*-contaminated grain) demonstrated that accidental antibiotic ingestion occurred during antiquity (Bassett et al. 1980). *Aspergillus flavus*, which flourishes in grain, also produces a chemical with antibacterial action: aflatoxin. Theoretically such a mechanism might contribute to soft tissue preservation after death.

One reason living cells survive the frequently complex chemical milieu commonly present within intracellular environments of living biological systems is that most enzymes are designed to respond to only a very narrow range of molecular structures. This high degree of *specificity* for enzymes' intended substrates makes possible the success of tissue preservation by the use of "fixing" substances like formaldehyde or certain alcohols which so radically rearrange proteins' molecular contour that proteases which are commonly present post mortem no longer react with them. Tannic acid is a fixing agent commonly employed today by taxidermists to preserve animal skins. The presence of tannic acid in many northern European swamps is believed to be responsible for the frequently excellent preservation of the "bog people" mummies found within them (Glob 1965:1–45).

ADIPOCERE FORMATION is initiated in the form of neutral fat hydrolysis by endogenous lipases subsequently modified by bacterial enzymes (usually of clostridial origin) resulting in the formation of a different group of fatty acids which are relatively insoluble and chemically poorly reactive. These may form a shell around the body surface, physically shielding the enclosed viscera from external influence and paralyzing further bacterial growth and enzymatic action internally by lowering the pH. These changes were thought to have been responsible for the preservation of two bodies submerged in water of known temperature for five years (Cotton et al. 1987).

DESSICATION

All enzymes can exert their action only in a fluid environment. Hence, enzymatic tissue destruction can be prevented if sufficient water is removed from the tissue (desiccation), a principle employed commercially for preservation of fruit and other foods. In order to be the sole operating mechanism in natural mummification such water removal would need to occur quite rapidly. There may be occasional situations in which this could occur (prolonged exposure to the summer sun in Cairo, for example), but they must be exceptional, and most naturally mummified bodies are not found in circumstances where this could have been exclusively operative. Burial conditions which would encourage removal of body fluids by conduction (capillary action) could be expected to accelerate the dehydration process in comparison to circumstances dependent only on surface evaporation and convection. For example, wrapping a corpse snugly in a wool blanket and interring it in sand may increase the rate of water removal from the body through the "wicking" effect of the textiles and sand. Furthermore, if the body position is vertical, enzyme-laden small-intestinal fluids will drain out through the perineum, sparing the viscera of the upper abdomen and chest. In spite of such enhancing conditions, however, it is probable that in most cases at least partial suppression of enzymatic action by one or more of the other, previously discussed mechanisms may need to operate in order to provide the time necessary for sufficient water removal from the tissues to prevent further enzymatic action by dehydration alone.

The mummies at Venzone, Italy

Resting at the junction of the Venzonassa Valley with that of the larger Tagliamento River, the small community of Venzone was strategically located to permit control of the flow of men and arms in ancient times to and from the Fruilian Plain through this narrow gap in the mountains of northeastern Italy near Trieste. Romans exploited its military virtue, but it was not until A.D. 1258 that the first stone of the wall presently enclosing the village was laid. While feudal lords vied for her possession, Venzone's inhabitants were more concerned with the commercial and social activities of their neighboring (and competing) community, Gemona. When the latter erected a majestic cathedral, Venzonians expanded their small church of St. Andrew into a grand cathedral, consecrating it in 1338. Burial vaults constructed beneath its stone floor served as the final resting place for clerics and important citizens during the subsequent five centuries. One of these bodies, relocated during construction work in 1679, was found to be mummified and was hastily hidden protectively in the underground tombs. Later remodeling expanded the cathedral further. By the first quarter of the 19th century so many of the bodies in the subterranean church vaults had become mummified that they attracted the attention of a physician from the nearby community of Udine. F.M. Marcolini, a medical staff member of the Udine municipal hospital, reviewed these mummies in 1829, carried out an autopsy on one of them, and published his findings, providing detailed descriptions of 17 of them, including the names and death dates of many (1831:42–121). By 1850, 27 mummies had been removed from the burial vaults, and eventually these were placed in glass-fronted display cabinets in the baptistery adjacent to the cathedral where they attracted the curiosity of many a visitor (Galassi 1950).

Several mummies from this collection have been lost. One or two were transferred to Vienna shortly after Napoleon conquered the area and annexed it to Austria. Two are believed to have been transported to a New York museum and two others to Rome or Padua. None of these have been located in recent times. In addition one was destroyed by Marcolini's autopsy in 1829 (Galassi 1950).

On 6 August 1950 Dr. A. Gallassi from the University of Bologna inspected the displayed mummies, compared them with Marcolini's descriptions and found little change other than the loss of hair from a red beard in mummy No.5 (Saccerdote Mistrozzi). He also added his own descriptions of an additional five mummies exhumed since Marcolini's visit (Galassi 1950).

A total of 21 mummies continued to be displayed in the cathedral's baptistery until the tragedy of 1976. On May 6 of that year a vigorous earthquake centered near Venzone caused considerable damage to the area, but a second one on September 15 was of catastrophic violence and nearly leveled most of the buildings in this unfortunate community. Not only much of the cathedral but also the entire baptistery collapsed, partially or completely burying many of the enclosed mummies. A local naturalist led several volunteers in a mummy recovery effort. About half of the 22 bodies were found reasonably intact. The remainder were disrupted, sometimes extensively. Some of the dismembered mummies were reassembled with the aid of pre-earthquake photos, but it was possible to salvage only a total of 15 of the 22 damaged bodies. Exposure to the elements had resulted in some apparent dampness of the skin in many. Fearing bacterial or fungal growth, the restoring party washed the bodies with a mixture of formalin and phenol, storing them in a room whose walls they washed with a similar solution. Further suppression of microorganismal growth was achieved by lining the floor and part of the room with formalin-soaked newspapers. Subsequently the mummies were transferred to a metal hut where they have been displayed during the period of community reconstruction until a building to house them permanently has been completed (Mainardis 1983).

When the cathedral was reconstructed and expanded in the 14th century the new floor level was raised 1.5 m. Most of the tombs 11 through 17 are constructed between the original floor level and that of the new, current one; the bottom of these tombs, therefore, is sealed by the original stone floor (Anonymous 1971:96). Tombs 1 through 10, however, were constructed under the floor level of the new, expanded portion and the bottom of these tombs consists only of soil. Interestingly, while skeletonized bodies were present in all tombs, mummies were found only in tomb numbers 1 through 10.

During periods of heavy rainfall and high stream levels, standing water has been observed in the tombs containing mummies, and such an occasion led the residents to remove the mummies from the tombs when they found some of the wood coffins flooded and a white fungus partially covering the surface of many of the mummified bodies within them. About 1829, B. Biasoletto, director of the Botanical Garden at Trieste, examined the mummies and identified the fungus as *Hypha bombycina* Pers., suggesting that the fungal growth, by extracting water from the bodies, may have been responsible for the process of natural mummification in these cadavers. Except for Marcolini's (1831) suggestion of the action of an acid gas from the soil and some adipocere formation, Biasoletto's suggestions have remained the locally accepted explanation of their mummification.

MATERIALS AND METHODS

Having obtained permission to view the mummies from the proper local authorities, the authors visited Venzone, Italy on 13 December 1983. No permission to biopsy or dissect the bodies was requested and no such procedures were carried out. Local archeologists, naturalists, historians, and residents with knowledge about the mummies and their tombs were interviewed. The mummies were inspected and two (numbers 1 and 8) were cultured for fungi by swabbing their facial skin and exposed leg muscle surface with sterile cotton and streaking these on slants of Sabouraud's agar tubes. These were incubated at room temperature for up to several months. Material from growth was transplanted into individual Sabouraud's agar tubes and onto slide cultures. Microscopic examination of slide cultures and wet slide preparations, together with gross culture characteristics, provided evidence for identification. A sample of light-gray sand and fine gravel soil from a recent building excavation adjacent to the cathedral was procured after scraping away 5 cm of soil from the vertical face of the excavated pit wall at a depth of 22 cm from the ground surface level.

The roofs of many of the tombs in the front of the church had collapsed during the earthquake and were sealed with cement slabs for safety reasons, but the cavity of tomb 9 was filled with debris, exposing only a part of its intact roof. Samples of wood and brick were removed from the exposed roof of this tomb in the cathedral. Access to tomb 15 was possible by removing its temporary wooden cover and descending cement steps leading to its interior. This was occupied by a jumble of disintegrated and collapsed wooden coffins. Individual human bones were scattered throughout this conglomeration, mixed with a damp, powdery material which also lined the floor under the coffins. Only soil at the foot of the steps, away from the coffins, was dry and light grey, similar to the soil in the excavation pit adjacent to the cathedral. No stone floor was identifiable. Coffin wood and floor soil were sampled from tomb 15. These samples were also weighed, then dried to constant weight and their moisture content calculated. Soil specimens were analyzed for lead content by graphite furnace atomic absorption spectrometry (Wittmers et al. 1981), and for mercury, arsenic, copper and cobalt by neutron activation analysis (the latter by Technical Services Laboratory, Mississauga, Ontario, Canada). Soil pH was determined by suspending the soil in distilled water and measuring the pH of the mixture with a Corning pH meter (McLean 1980). Swabs of the coffin boards in tomb 15 and of the soil samples were also prepared for fungus cultures as described above.

Analytical results are itemized in Table 1.

DISCUSSION

The geological structure of the Venzone area is almost entirely that of calcareous rocks (Mainardis 1976:16). The high carbonate content of the soil adjacent to the church and that composing the floor of tomb 15 (Table 1) is consistent with limestone. Surface and ground waters of such areas normally are quite alkaline. The pH values of samples listed in Table 1, however, range from neutrality to 6.0. This is probably a local phenomenon secondary to industrial and other anthropogenic acidic products affecting the soil sampled outside of and adjacent to the church, while that of the tomb floor was undoubtedly contaminated with acidic tissue products from multiple degenerating bodies. The neutral or slightly acidic values in these particular samples do not exclude an alkaline pH effect on tissue enzymes resulting from periodic tomb flooding by ground water seeping through limestone.

Similarly, moisture content of the "dark" soil sample from the tomb floor is high. Since the normal soil color of this area is light grey, the dark color of this soil sample from beneath the collapsed coffins undoubtedly reflects substantial contamination with degenerating human tissue. The relatively low moisture content of the boards and that of the bricks in the tomb roof probably more accurately reflect the normal state of humidity in an empty tomb lined by normally hydrophilic dolomite rock and soil, as suggested by the nearly

TABLE 1. Analytical results of studies performed on samples relating to mummies at Venzone, Italy

Sample	*Fungus culture*	*Moisture content %*	*pH*	*Heavy metals (ppm)* *Pb*	*As*	*Hg*	*Cu*	*Co*	*Carbonate*
Skin, mummy #1	*Penicillium* sp.	-	-	-	-	-	-	-	-
Skin, mummy #8	*Bacillus* (strep.)	-	-	-	-	-	-	-	-
Soil, excavation	*Microascus* sp.	1	6.5	-	.76	.161	19	16	Moderate
Soil D, floor, Tomb 15	*Trichoderma* sp.	47	6.0	460	1.2	1.64	346	.62	-
Soil L, floor, Tomb 15	No growth	14	6.5	94	.90	.608	26	13	High
Coffin board, Tomb 15	*Scedosporium* sp.	7	-	-	-	-	-	-	-
Brick roof, Tomb 9	*Trichoderma* sp. *Penicillium* sp.	9	6.8	17	-	-	-	-	High

absent moisture in the soil sample from adjacent to the church. This could be a factor contributing to the dehydration of a body whose postmortem, enzymatic tissue digestion had been delayed by some other factor.

Contamination of Venzone area soils by common heavy metals is clearly absent, as seen in the listed analytical values of the sample from adjacent to the church. The dark soil of the floor from tomb 15 immediately below the coffins reveals contamination by lead (460 ppm) and copper (346 ppm). The latter is a known, powerful, enzymatic poison, and it is a common archeological experience to find human soft tissue preserved adjacent to a copper ornament, such as a bracelet, on an otherwise skeletonized body. While our observation circumstances did not permit a thorough search of the tomb content, both lead and copper items were common components of coffins or their contained artifacts since medieval times, and thus are the most likely source of these contaminating elements.

The fungus cultures are of special interest. Many of the mummies were partially covered with a white fungus at the time of their exhumation. Biasoletto's concept (1829?), attributing the desiccated state of the mummies' tissues to the dehydrating effect of growth of the fungus "Hypha bombacina," has become a permanent part of local folklore. Since this terminology is no longer used, it was necessary to peruse 19th century mycological taxonomy texts to obtain a description of the organism to which this label was applied. An 1822 publication (see Appendix) suggests this name was created by Persoon (1822:64) and persisted at least until 1899 (Saccardo 1899:1192). It was used to describe a white, fluffy, cottony or silky mycelial growth without conidia. Unfortunately, this description is too nonspecific to permit its assignment within the modern system of mycological taxonomy.

The fungi cultured from the various sources listed in Table 1 do not include any which demonstrate the growth characteristics listed under the rubric "Hypha bombycina" in the older texts. Indeed, they represent common household and soil fungi. The formaldehyde and phenol treatment of the bodies following the earthquake as described above can be expected to have destroyed fungi and probably also their spores previously present in or on the bodies, but if fungi meeting the description of "Hypha bombycina" had played a significant role in the mummification of the bodies in the various tombs, it would seem reasonable to anticipate their abundant presence in the soil samples and on the tomb contents. Of the organisms recovered from our sample cultures, only *Penicillium* is known to produce an antibiotic substance. While penicillin is effective against many gram-positive bacteria, including the clostridia commonly present in feces, it does not inhibit the growth of most gram-negative organisms including *Escherichia coli*, the most numerous stool inhabitant. It is also common hospital experience that therapeutic tissue concentrations of penicillin at the time of death do not prevent postmortem tissue degeneration. Postmortem fungal growth is extremely common on tissues of exposed, degenerating bodies after autogenous, bacterial, and insect enzymatic action has acidified the tissues (Evans 1963:4). Such growth, however, almost invariably restricts itself to exposed surfaces and is thus unable to exert any significant preserving effect on deeper tissues. It appears improbable to us that fungal growth contributed significantly to the desiccated state of the Venzone mummies.

The mummy bodies were examined (limited to inspection) for gross evidence of molecular alteration of proteins and/or fat to a chemically inert state. Facial features were frequently reasonably intact. The skin over the thorax was riddled with insect holes but was generally otherwise intact in many mummies. The anterior abdominal wall was frequently partially or completely absent; in these the exposed abdominal cavity rarely contained recognizable organs. Tissues comprising the perineum, gluteal areas, medial aspect of the thighs and posterior trunk were absent in most of the mummies—a pattern commonly present in naturally mummified bodies placed in a supine position post mortem and reflecting the gravitational distribution of enzyme-rich, intestinal digestive fluids. While admittedly adipocere formation is not always obvious, only a few, focal areas (primarily facial) could be identified which conceivably could represent such an alteration of fat. Certainly we could not confirm that the bulk of the tissue preservation was the consequence of a shell of protective adipocere formation, nor was there any recognizable evidence of chemical protein fixation. The general condition of the mummies surviving the earthquake was similar to that described by Gallassi in 1950.

It appears unlikely that tomb temperatures ever reach the freezing point. Midwinter temperature measurement (made by the authors) of a sealed, nearly identical tomb beneath the stone floor of the church in a nearby community of Urbania was 18°C.

In summary, consideration of factors possibly contributing to the natural mummification of these bodies suggests it is conceivable, though not established, that highly alkaline ground water seeping through the surrounding area limestone intermittently may gain access through the soil floors to these bodies' tombs (though not as readily to the tombs with stone floors in the rear of the church), transiently immersing the corpses and raising their tissue pH at least temporarily to a degree sufficient to retard enzymatic soft tissue destruction for a period long enough to permit subsequent tissue desiccation by the hydrophilic effect of the dolomite soil. Alternatively, postmortem enzymatic action on tissues could have been paralyzed by the presence of heavy metals or by acidic action from degenerating textiles or other artifacts like copper, probably from coffin components or included contents (since area soils do not reveal excess quantities of at least the more common heavy metals). Cool but not frigid ambient temperatures may have enhanced retardation of soft tissue degeneration during winter burials, but it appears highly unlikely that natural preservation of these bodies was achieved through extreme thermal action (freezing or very high temperatures), by the mechanism of anoxia, by protein fixation or significant adipocere formation, nor by the dehydrating or antibiotic action of any fungus, including that of the organism termed "Hypha bombycina."

In brief, while application of our current information (regarding spontaneous mummification) to the Venzone mummies makes certain possibilities more conceivable than others, it is not possible to establish the locally operative mechanisms with a desirable degree of certainty. Clearly, more precise predictions of such events will require a much more detailed understanding of postmortem changes, made possible by a program of laboratory investigation designed for that purpose.

Research needs

Presently, bodies preserved by spontaneous mummification represent the most valuable form of human remains for biomedical and anthropological study, since they retain the organs bearing the anatomical, biochemical, immunological or microbiological evidence of the disease afflicting them, including the final, fatal episode. Postmortem degenerative processes place constraints on the ability of our modern laboratory methods to extract the desired information from these tissues. Detailed knowledge of these postmortem changes would permit us to adapt our technology so as to maximize the informational return.

The broad generalizations necessary in the previous discussion as well as our inability to identify unequivocally the mechanisms producing the Venzone mummies reflect the relatively barren state of our knowledge about postmortem human tissue changes. In a few, restricted areas isolated reports permit the synthesis of at least a proposed chemical sequence leading to a specific preservational product (adipocere) (Cotton et al. 1987), but every mechanism discussed here needs to be studied in detail by developing an appropriate operational model in which the individual variables of interest can be controlled. Measurement methods for the various reactions studied need to be created. Effects of the individual factors impeding or enhancing these reactions need to be identified and quantified. Following this the results obtained in isolated tissue under controlled circumstances then need to be evaluated under the infinitely more complex, field situations of the entire organism. While the "chemistry of death" (Evans 1963:1–87) is surely a complicated matter, it may be no more so than many which have been well defined in living organisms.

Our understanding of postmortem tissue changes and the factors affecting them will only be achieved by such orderly investigations. Field observations unsupported by the controlled studies described above rarely provide information applicable to situations occurring under different circumstances. Funding necessary to carry out such studies may be justified by the obvious applications of the derived data to everyday forensic problems of enormous medico-legal importance.

Appendix

The authors are very grateful to John Rippon, Ph.D., Section of Dermatology, Department of Medicine, University of Chicago and Pritzker School of Medicine, for the following information.

In *Mycologia Europaea*, Vol. 1, by Persoon, published in 1822, the following entry is found on page 65:

> Classis prima, Ordo primus:
>
> XXXV. Hypha. Flocciformis, mollissima ad tactum diffluens, fugax. Obs. Fungi sunt subterranei, aut in locis suffocatis et cryptis vegetant, colore plerumque albi.
>
> 1. bombycina, subrotunda indeterminata, humida floccos gossypinos referens Hyphasma floccosum. *Rebentisch, Flor.* p.396. Dill. Hist. Musc. p.5,t.1.f.9. Dematium bombycinum. Syn. fung. 696. Hab in fodinis et cellis, lignis et lapidibus adhaerens. Colore nivea . . .

In *Sylloge Fungorum*, Vol.14, by P.A. Saccardo, published in Pavia, 1899, by the author, the following entry is found on page 1192:

> Mycelia sterilia, Hypha:
>
> HYPHA Pers. M.E.I, p.63, *Hyphasma* Rebent. *Dematium* Pers. *Byssus* L. pr. pr. ex emend.
>
> Humb.,Dub. (Etym. *hyphe* texture).
>
> —Rhabdi arachnoidei hyalini simplices v. ramosi, decumbentes, fistulosi, continui, laxe intertexti, fugaces (aeris contactu saltem) contabescentes v. confluentes, sporis destituti. Mycetes subterranei in cryptis locisque suffocatis, vegeti albi, aliorum procul dubie initia, ex loco lucisque defectu pessumdati, hinc dubii.
>
> 1. Hypha bombycina Pers. M.E.I, p.63, *Byssus floccosa* Schreb. Spic. 144, *B. bombycina* Nees Syst. f. 73, *Hyphasma floccosum* Rebent. Neom. p.396, *Dematium bombycinum* Pers. Syn. p.696, Dill. Hist. t.1.f.9—Rhabdis hyphoideis, simplicissimis, subparallelis, niveis, primum laxe intertextis, hypham veluti gossypinam, elatam subrotundam, mundissimam, dein collapsis, membranam compactam seu corium tenacellum mentientibus. *Hab.* ad ligna et alpides in caveis udis et fodinis minime rara in Germania.—18–20 cm. alta et lata, ocissime gliscens.

Literature cited

Alexander, F. 1949. A Medical Survey of the Aleutian Islands. *New England Journal of Medicine*, 240:1035–1040.

Allison, M.J., G. Focacci, B. Arriaza, V. Standen, M. Rivera, and J.M. Lowenstein. 1984. Chinchorro, Momias de Preparación Complicada: Métodos de Momificación. *Chungara*, 13:155–174.

Anonymous. 1971. *Venzone*. Udine, Italy: Societat Filologiche Furlane.

Artamonov, M.I. 1965. Frozen Tombs of the Scythians. *Scientific American*, 212:101–110.

Bassett, E.J., M.S. Keith, G.J. Armelagos, and D.L. Martin. 1980. Tetracycline-Labelled Human Bone from Ancient Sudanese Nubia (A.D. 350). *Science*, 209:1532–1534.

Beattie, O.B. 1983. A Report on Newly Discovered Human Skeletal Remains from the Last Sir John Franklin Expedition. *The Musk-Ox*, 33:68–77.

Biasoletto, B. 1829(?) *In Cella Subterranea Venzonense ad Corpora Humana Emortua. Exiccataque Lectus.* Quoted in F.M. Marcolini, 1831. *Sulle Mummie di Venzone*, 58, 148. Milan: Dalla Societa Tipografica de'Classici Italiani.

Cheng, T.O. 1984. Glimpses from the Past from the Recently Unearthed Ancient Corpses in China. *Annals of Internal Medicine*, 101:714–715.

Cockburn, A., and E. Cockburn, eds. 1980. *Mummies, Disease and Ancient Cultures.* Cambridge, U.K.: Cambridge University Press.

Cotton, G.E., A.C. Aufderheide, and V.G. Goldschmidt. 1987. Preservation of Human Tissue Immersed for Five Years in Fresh Water of Known Temperature. *Journal of Forensic Sciences*, 32:1125–1130.

El-Najjar, M., A.C. Aufderheide, and D.J. Ortner. 1985. Preserved Human Remains from the Southern Region of the North American Continent. *Human Pathology*, 16:273–276.

Evans, W.E.D. 1963. *The Chemistry of Death.* Springfield, Ill.: Charles C Thomas.

Fornaciari, G. 1982. Natural Mummies in Central Italy. *Paleopathology Newsletter*, 40:11–13.

Gallassi, A. 1950. Le Mummie Naturali di Venzone. *Rivista di Storia Delle Scienze Mediche e Naturali*, 41:194–199.

Glob, P.V. 1965. *Mosefolket.* Copenhagen: Gyldendal.

Hansen, H.E., and H. Gurtler. 1983. HLA Types of Mummified Eskimo Bodies from the 15th Century. *American Journal of Physical Anthropology*, 61:447–452.

Kleiss, E. 1967. Zum Problem der Naturlichen Mummifikation und Konzervierung. *Zeitschrift fur Morphologie und Anthropologie*, 59:204–213.

Mainardis, G. 1976. *Venzone: Studi Geologici Sul Terreno.* Udine, Italy: Arti Grafiche Friulane.

Marcolini, F.M. 1831. *Sulle Mummie di Venzone.* Milan: Dalla Societa Tipografica de'Classici Italiani.

McLean, E.O. 1980. Recommended pH and Lime Requirement Tests. Recommended Chemical Soil Test Procedures for the North Central Region, North Dakota Agricultural Experiment Station, North Dakota State University, Fargo. *North Central Regional Publication No. 221*, Bulletin 499:5–6.

Micozzi, M.S. 1986. Experimental Study of Post-Mortem Change Under Field Conditions: Effects of Freezing, Thawing and Mechanical Injury. *Journal of Forensic Sciences*, 31(3):953–961.

Mostny, G., ed. 1957. La Momia del Cerro el Plomo. *Boletin del Museo Nacional de Historia Natural*, 27:1–118.

Orslanov, Kh.A., V.V. Lyadov, and T.V. Tertychnaya. 1980. In N.K. Vereshchagin, ed., *The Magadan Baby Mammoth, Collection of Articles*. Leningrad: Academy of Sciences USSR, Section of Chemical-Technological and Biological Sciences. Committee for the Study of Mammoths and Mammoth Fauna.

Paddock, F.K., C.C. Loomis Jr., and A.K. Perkons. 1970. An Inquest on the Death of Charles Francis Hall. *New England Journal of Medicine*, 282:784–786.

Persoon. 1822. *Mycologia Europaea*, vol. 1.

Saccardo, P.A. 1899. *Sylloge Fungorum*, vol. 14. Pavia, Italy: published by the author.

Schobinger, J. 1966. *La "Momia" del Cerro el Toro.* Mendoza, Argentina: Taller Grafico.

Snow, C., and T.A. Reyman. 1977. The Life and Afterlife of Elmer J. McCurdy. *Paleopathology Newsletter,* 19. Special supplement.

Wittmers, L.E., A. Alich, and A.C. Aufderheide. 1981. Lead in Bone I. Direct Analysis for Lead in Milligram Quantities of Bone Ash by Graphite Furnace Atomic Absorption Spectrometry. *American Journal of Clinical Pathology,* 75:80–85.

Wu, Z.B., H.S. Tian, and Y.E. Zeng. 1980. Study of the Ancient Corpse of the Western Han Dynasty Unearthed from Tomb No. 168 on Phoenix Hill at Jiangling County (A Comprehensive Report). *Medical Bulletin Wuhan Medical College,* 1:1–8.

Zimmerman, M.R., and A.C. Aufderheide. 1984. The Frozen Family of Utqiagvik: The Autopsy Findings. *Arctic Anthropology,* 21:53–64.

Zimmerman, M.R., E. Trinkhaus, M. LeMay, A.C. Aufderheide, T.A. Reyman, G.A. Marrocco, W. Ortel, J.T. Benitez, W.S. Laughlin, P.D. Horne, R.E. Schultes, and E.A. Coughlin. 1981. The Paleopathology of an Aleutian Mummy. *Archives of Pathology and Laboratory Medicine,* 105:638–641.

SUMMARY OF AUDIENCE DISCUSSION: In Israel a monk buried with intact clothing has been disinterred and found to be marvelously preserved. Other areas where spontaneous mummification has occurred in subterranean chambers include Urbania (northern Italy), Palermo (Sicily) and Guanamoto (northwestern Mexico). All of these occur in limestone areas but the mechanisms are speculative. Recently in a medieval cemetery in northern England the very well-preserved body of a knight was found wrapped in a shroud and rolled up in a lead sheet. Clearly we need to study the phenomenon of spontaneous mummification by experimental archeological means.

Soft tissue calcifications in paleopathology

C.-A. Baud and Christiane Kramar

Calcified masses are rarely described in paleopathological literature (because they are perhaps not always found). As their origin (Table 1) and their interest may be varied, it is important, in the presence of extraskeletal calcifications, to distinguish ectopic ossifications, tissue calcifications, and calculi.

The diagnosis of some calcifications, owing to their attachment to the skeleton, is easy: for example, the stylohyoid ligament (O'Carroll 1984), costal cartilages (McCormick 1980) and myositis ossificans (Lagier and Baud 1978). When calcifications are isolated, but identifiable owing to their shape, diagnosis is also easy: the laryngeal cartilages (Jurik 1984), for example. When calcifications are not attached to the skeleton and are without anatomical shape, the diagnosis is harder. As such examples we present pleural plaques, leiomyomas of the uterus, a tuberculous lymph node, and a hydatid cyst.

Material and methods

Pleural plaques were found with the osseous remains of men from three different medieval cemeteries: Collonge, Geneva, Switzerland, 10-11c, grave 45, man of more than 60 years old (Baud 1972; Bonnet 1972); St. Matthieu's Church, Bernex, Geneva, Switzerland, 13-14c, grave 44, man of 60 years old (Kramar 1984), and St. Gervais, Geneva, Switzerland, grave 27.

The first leiomyoma of the uterus was found among human skeletal remains from a Middle Neolithic population, Corseaux-sur-Vevey, Vaud, Switzerland, 4700–3490 B.C. (Kramar 1982; Kramar et al. 1983). The two others are from Early Middle Ages cemeteries: Sion Sous-le-Scex, Valais, Switzerland, 5-10c, grave 37 and Rances, Vaud, Switzerland, 5-7c.

The lymph node was found in a collective burial from the Chalcolithic period, Dolmen des Peirieres, Villedubert, Aude, France (Roudil 1976).

The hydatid cyst was found with the remains of a medieval child 2–4 years old.

All concretions were first macroscopically and radiologically observed. Fragments of concretions were embedded in methyl methacrylate and sectioned for microscopic examination in normal and polarized light. Ground sections 10 μ thick were decalcified in formaldehyde-formic acid and stained with van Gieson's picrofuchsine.

Microradiographs were made from sections 100 μ thick, according to the technique of Boivin and Baud (1984). Fragments from the 100-μ sections were reembedded in epoxy resin (Epon) for electron microscopic examination. Ultrathin sections were decalcified and stained with phosphotungstic acid.

An x-ray powder diffraction analysis was performed with a Guinier camera to determine the crystalline species, and an x-ray diffractometric recording, following the technique of Jacquet et al. (1980), for crystal size and/or perfection estimate.

Results

PLEURAL PLAQUES

Macroscopically the pleural plaques were hard, of variable size (14.5 × 13 cm the biggest; 7.5 × 4.5 cm the smallest; 4–5 mm thick), with an irregular surface structure. Radiographs reflected these differences with a disparity of absorption.

TABLE 1. Examples and etiology of some soft tissue calcification

Etiology	*Examples*
Developmental	Stylohyoid ligament Crowned odontoid
Aging	Laryngeal cartilages Costal cartilages
Immobilization	Articular tissues
Trauma	Myositis ossificans
Inflammatory	Pleural plaques Lymph node Hydatid cysts
Tumoral	Leiomyoma

The microradiographs showed large areas with a high and uniform degree of mineralization, and others characterized by a lower degree of mineralization and the presence of some lens-shaped cavities of the size of the bone osteocytic lacunae. Examination of the decalcified sections showed areas stained red with van Gieson's method, fibrillar texture, and positive birefringence. Electron microscopic study indicated the presence of collagen fibrils with characteristic striation; these fibrils were scattered in highly mineralized zones and packed close in a parallel direction in the less mineralized ones.

X-ray diffraction patterns were characteristic of apatite, with large crystals in the more mineralized zones (fine lines) and small crystals in the others (broad lines).

After dissolution of apatite, we looked at the presence of minerals known to provoke fibroses (Le Bouffant 1974): we found nothing to support the hypothesis that minerals in the environment of these individuals are agents in this etiology (Constantopoulos et al. 1985).

LEIOMYOMAS OF THE UTERUS

Macroscopically the Neolithic leiomyoma was a spherical mass (56 × 52 × 45 mm) with a smooth but irregular surface. X-rays confirmed its mineralized nature. Examination of the decalcified sections showed the presence of collagen fibers; van Gieson's method stained them in red, and birefringence was positive; no bone structure was observed. Microradiographs showed a high and uniform mineralization. Electron microscopy confirmed the presence of collagen fibrils with the characteristic striation. The mineral material was apatite; crystal size and/or perfection were good.

The dimensions of the two others were 46 × 32 × 25 mm (Sion Sous-le-Scex) and 30 × 26 × 18 mm (Rances). The section of these calcifications showed the characteristic whorllike pattern of the leiomyoma (Bartholomew et al. 1961).

A similar case of calcified uterine leiomyoma was reported by Strouhal and Jungwirth (1977).

LYMPH NODE

The lymph node was a reniform mass (12 × 8 mm) with a lamellar capsule with numerous perforations enclosing two rounded nodules. Microradiographs of the sections showed that both capsule and nodules were highly mineralized. Histological study of decalcified sections showed fibrillar structure with a positive birefringence and a red van Gieson's staining, particularly in the surface layers of the node. X-ray diffraction revealed two mineral components: apatite in the periphery, and apatite together with whitlockite in the center, as we observe in calcified tuberculous lesions (Lindgren 1961:81–89; Lagier et al. 1966; Sakae and Yamamoto 1987). The shape, size, and fibrous capsule with numerous perforations suggest a lymph node; calcified foci formed of apatite and whitlockite suggest calcification of tuberculous origin.

HYDATID CYST

The cyst was an ovoid, hollow concretion 1 cm in diameter, with a smooth internal surface and an irregular external surface. The observation of a section with polarized light showed tangled collagen fibrils. All these facts characterize a cyst wall (Weiss and Møller-Christensen 1971; Price 1975:366–367; Wells and Dallas 1976; Ortner and Putschar 1981).

The mineral component was apatite only; this is compatible with an hydatid cyst (Lagier et al. 1966:158).

Differential diagnosis of soft tissue calcifications

It is important to distinguish between ossifications, calcifications, and calculi.

We have first to differentiate tissue calcifications from calculi. Concretions in the body cavities (gastroliths, enteroliths, bezoars, etc.) and calculi in excretory ducts (salivary, biliary, urinary) also contain an organic matrix (Kahn and Hackett 1984), but in small quantity and not of collagenous nature (not stained with van Gieson's picrofuchsine).

Among tissue calcifications we have to distinguish between a calcification and an ossification: calcification corresponds to a deposit of mineral material in a connective tissue, more or less altered, which shows the presence of scattered collagen fibrils and a very high degree of mineralization; ossification has a characteristic texture with an oriented disposition of collagen fibrils and osteocytic lacunae. We have to note that a tissue calcification tends to be replaced by an ossification (Kuhlmann 1934), which explains the coexistence, in pleural plaques, of ossified zones and calcified zones.

The study of the mineral component of a calcification can permit the substantiation of an etiological diagnosis: most of the soft tissue calcifications are formed of apatite only, and they correspond to a broad spectrum of pathological conditions (Lagier et al. 1966:158). Mixed crystal deposits, with apatite and whitlockite, are found predominantly in lesions of tuberculous or parasitic origin (Lagier et al. 1966:159).

Literature cited

Bartholomew, L.G., J.C. Cain, G.D. Davis, and A.H. Bulbulian. 1961. Misleading Calcific Shadows in the Abdomen. *Postgraduate Medicine*, 30:51–52.

Baud, C.-A. 1972. Une Plaque Pleurale Calcifiee: Etude Ultrastructurale et Cristallographique. *Genava*, 20:196–199.

Boivin, G., and C.-A. Baud. 1984. Microradiographic Methods for Calcified Tissues. In G.R. Dickson, ed., *Methods of Calcified*

Tissue Preparation, 391–412. New York: Elsevier Science Publishers, B.V.

Bonnet, C. 1972. L'Ancienne Eglise de Collonge (Collonge-Bellerive, Geneve). *Geneva*, 20:131–203.

Constantopoulos, S.H., J.A. Goudevenos, N. Saratzis, A.M. Langer, I.J. Selikoff, and H.M. Moutsopoulos. 1985. Metsovo Lung: Pleural Calcification and Restrictive Lung Function in Northwestern Greece. Environmental Exposure to Mineral Fiber as Etiology. *Environmental Research*, 38:319–331.

Jacquet, J., J.M. Very, and H.D. Flack. 1980. The 2 Determination of Diffraction Peaks from "Poor" Powder Samples: Application to Biological Apatite. *Journal of Applied Crystallography*, 13:380–384.

Jurik, A.G. 1984. Ossification and Calcification of the Laryngeal Skeleton. *Acta Radiologica*, 25:17–22.

Kahn, S.R., and R.L. Hackett. 1984. Microstructure of Decalcified Human Calcium Oxalate Urinary Stones. *Scanning Electron Microscopy*, 2:935–941.

Kramar, C. 1982. La Nécropole de Corseaux-sur-Vevey: Etude Anthropologique et Description Archéologique. Thèse 2041, Genève.

———. 1984. Plaques Pleurales Chez un Homme du Moyen Age: Etude Radiologique, Microscopique et Cristallographique. *Fifth European Meeting of the Paleopathology Association*, 199–210. Siena, Italy.

Kramar, C., C.-A. Baud, and R. Lagier. 1983. Presumed Calcified Leiomyoma of the Uterus. *Archives of Pathology and Laboratory Medicine*, 107:91–93.

Kuhlmann, K. 1934. Zur Atiologie, Entstehung und Bedeutung der Pleuraverkalkungen bzw. Pleuraverknöcherungen. *Fortschritte auf dem Gebiete der Röntgenstrahlen*, 49:147–154.

Lagier, R., and C.-A. Baud. 1978. Some Comments on Paleopathology Suggested by a Case of Myositis Ossificans Circumscripta Observed on a Medieval Skeleton. *Journal of Human Evolution*, 9:9–13.

Lagier, R., C.-A. Baud, and M. Buchs. 1966. Crystallographic Identification of Calcium Deposits as Regards Their Pathological Nature, with Special Reference to Chondrocalcinosis. In H. Fleisch, H.J.J. Blackwood, and M. Owen, eds., *Third European Symposium on Calcified Tissues*, 158–162. New York: Springer-Verlag.

Le Bouffant, L. 1974. Investigation and Analysis of Asbestos Fibers and Accompanying Minerals in Biological Materials. *Environmental Health Perspectives*, 9:149–153.

Lindgren, I. 1961. Anatomical and Roentgenologic Studies of Tuberculous Infections in BCG-Vaccinated and Non-Vaccinated Subjects. With Biophysical Investigations of Calcified Foci. *Acta Radiologica* (supplementum) 209:1–101. Stockholm.

McCormick, W.F. 1980. Mineralization of the Costal Cartilages as an Indicator of Age: Preliminary Observations. *Journal of Forensic Sciences*, 25:736–741.

O'Carroll, M.K. 1984. Calcification in the Stylohyoid Ligament. *Oral Surgery*, 58:617–621.

Ortner, D.J., and W.G.J. Putschar. 1981. Identification of Pathological Conditions in Human Skeletal Remains. *Smithsonian Contributions to Anthropology*, 28:229–233. Washington, D.C.: Smithsonian Institution Press.

Price, J.L. 1975. The Radiology of Excavated Saxon and Medieval Human Remains from Winchester. *Clinical Radiology*, 26:363–370.

Roudil, J.-L. 1976. Villedubert, Les Peirières. Informations Archéologiques. *Gallia Préhistoire*, 19:553–555.

Sakae, T., and H. Yamamoto. 1987. Crystals and Calcification Patterns in Two Lymph Node Calcifications. *Journal of Oral Pathology*, 16:456–462.

Strouhal, E., and J. Jungwirth. 1977. Ein verkalktes Myoma uteri aus der späten Römerzeit in Aegyptisch-Nubien. *Mitteilungen der Anthropologischen Gesellschaft*, 107:215–221. Vienna.

Weiss, D.L., and V. Møller-Christensen. 1971. Leprosy, Echinococcosis and Amulets: A Study of a Medieval Danish Inhumation. *Medical History*, 15:260–267.

Wells, C., and C. Dallas. 1976. Romano-British Pathology. *Antiquity*, 50:53–55.

Summary of audience discussion: Interpretation of crystal characteristics of excavated calcified material is most useful if its location within the body is known precisely. In the thorax, for example, it becomes more informative if it can be determined whether the calcified mass represents simple exudate or a pleural plaque and whether costal periostitis was present. Archeologists need to be made aware of the importance inherent in such detailed observations. Since Dr. Brothwell has observed bacteria in dental plaque specimens viewed by scanning electron microscopy, a similar search for mycobacteria in calcified material suspected to be of tuberculous origin appears desirable, as well as diagnostic immunological studies on protein included within calcified material.

Technological innovations and discoveries in the investigation of ancient preserved man

Peter K. Lewin

Stereoscan imaging from composite "CT scans"

Late in 1976 when the first computerized axial x-ray tomographic machine (CT scan) was installed at the Hospital for Sick Children in Toronto, Dr. Harwood-Nash and I performed the first CT scan on an archeological specimen (Lewin and Harwood-Nash 1977). The specimen was the desiccated brain from Nakht, an adolescent weaver from the funerary chapel of Setnakht, who had lived in Thebes about 3000 years ago. The images from Nakht's brain demonstrated intact ventricular cavities and partial differentiation of the grey and white areas of the brain together with small postmortem cavities.

The brain scan was followed by the first total body scan of the mummy of the priestess Djema 'Etes' Ankh, dating from about ninth century B.C. Tomographic sections of the head showed prosthetic artificial eyes set in the eye sockets. Sections through the pelvis showed normal hip joints, and within the pelvis the remains of the uterus could be visualized (Lewin 1978).

In the last two years in collaboration with John Stevens and Judy Trogadis at Toronto Western Hospital, who developed the computer software for this project (Stevens and Trogadis 1986), we have extended our noninvasive imaging techniques by constructing three-dimensional images from sequential data obtained by the two-dimensional CT scan.

A beautifully preserved head from the Greco-Roman period is shown in Figure 1. Composite spacial see-through reconstructions of ancient archeological specimens, using computed axial tomograms, are presented in Figures 2 and 3.

This technique can also be applied to other imaging methods and enhanced with newer, digital processors. These new, nondestructive imaging methods would be invaluable in the three-dimensional examination of mummified remains and their internal structures including archeological objects, keeping these valuable specimens intact for posterity.

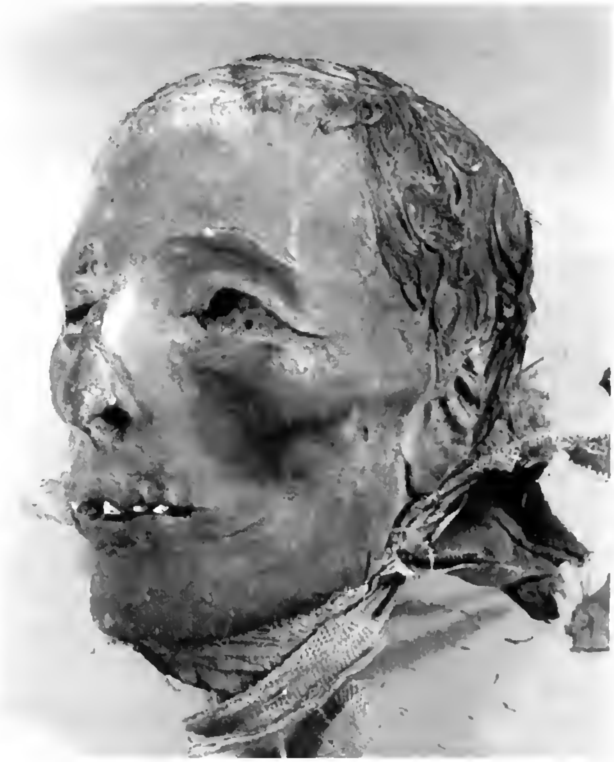


FIGURE 1. Ancient Egyptian mummified head from Greco-Roman Period.

Electron microscopy of mummified tissues to demonstrate viral agents and their possible viability

Ancient Egyptian mummified material was first examined at the ultrastructural level in 1966 (Lewin 1967) and demonstrated reasonable preservation of cellular organelles. Since then numerous tissues have been examined by electron microscopy, the best preserved tissues often being skin, blood, muscles, and vessels.

Viral agents have also been demonstrated, including smallpox-like particles from the mummy of Ramses the Fifth

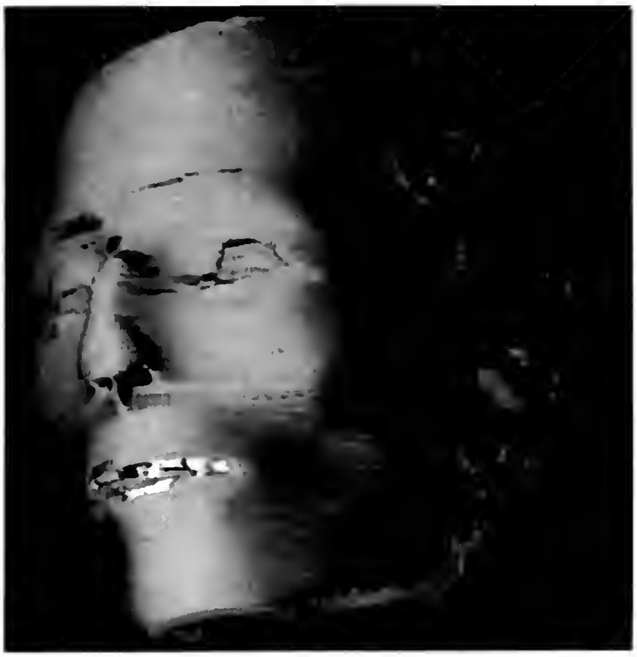


FIGURE 2. Computer-generated image of mummified head. (Photograph courtesy of P.K. Lewin, J.K. Stevens and J. Trogadis)

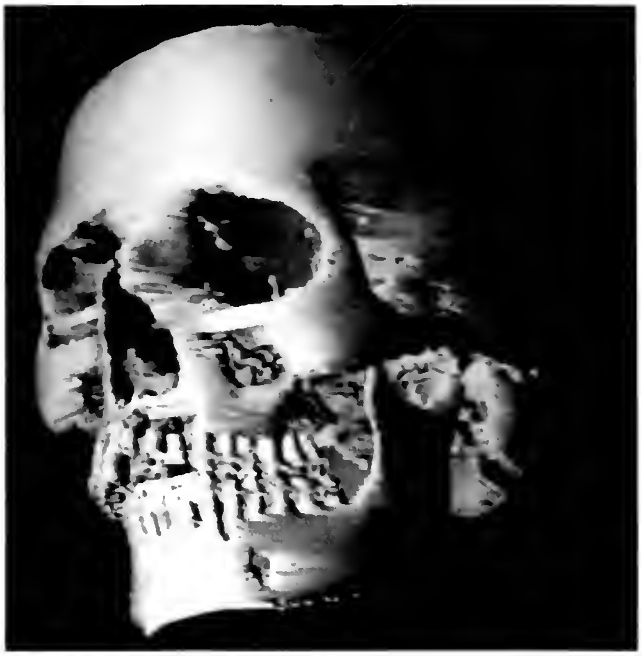


FIGURE 3. Computer-generated image of skull from mummified head. (Photograph courtesy of P.K. Lewin, J.K. Stevens and J. Trogadis)

(Figure 4) (Lewin 1984) and from a naturally mummified two-year-old infant from Naples (Fornaciari and Marchetti 1986).

The above methods and the recent use of DNA hybridization techniques are being used to determine the viability of at least some of the genetic DNA content of ancient biological remains. It is possible that infectious agents are still viable, particularly viruses in ancient human and animal specimens preserved in northern areas by permafrost.

Literature cited

Fornaciari, G., and A. Marchetti. 1986. Intact Smallpox Virus Particles in an Italian Mummy of the XVI Century: An Immuno-Electron Microscopic Study. *Paleopathology Newsletter*, 56:7–12.

Lewin, P.K. 1967. Palaeo-Electron Microscopy of Mummified Tissue. *Nature*, 213:416–417.

______. 1978. Whole Body Scan of an Egyptian Mummy Using X-Ray Computed Axial Tomography. Abstract of a paper presented to Paleopathology Association Meeting, April 1978. *Paleopathology Newsletter*, 22:T7–8.

______. 1984. "Mummy" Riddles Unraveled. *Microscopy Society of Canada Bulletin*, 12:4–8.

Lewin, P.K., and D.C. Harwood-Nash. 1977. X-Ray Computed Axial Tomography of an Egyptian Brain. *International Research Communication System, Medical Science*, 5:78.

Stevens, J.K., and J. Trogadis. 1986. Reconstructive Three Dimensional Electron Microscopy. *Analytical and Quantitative Cytology and Histology*, 8:102–107.

FIGURE 4. Smallpox-like viral particle found on the mummy of Ramses the Fifth (× 200,000) (Lewin 1984).

SUMMARY OF AUDIENCE DISCUSSION: The three-dimensional image generated by computerized radiology is sufficiently accurate to be usable for forensic purposes especially in cases of only partial preservation of cranial bones. The value of preserving such images of bones scheduled for interment is potentially enormous. The present resolution of 1.5 mm is expected to be reduced soon to 0.25 mm or even lower, which would permit evaluation of such areas as the pubic symphysis which are uniquely useful in paleopathology. A good deal of ethnic variation remains to be included into the programs, after which it may be possible to create a reconstructed image of the soft tissue on the basis of a "skull-scan."

Harris' lines in adults: An open problem

Lubos Vyhnanek and Milan Stloukal

More than 30 years ago Harris drew attention to the characteristic metaphyseal transverse lines found in the x-ray pictures of the long bones, especially in children. He rightly concluded that these lines represented the consequences of the temporary arrest of growth which could be caused by different factors (Harris 1933). The term "lines" refers to the radiological pictures in reality; they constitute zones of lamellar bone (Figure 1) which appear as lines when tangentially projected in the x-ray picture. They develop during the growth of the bones as the result of a temporary growth mechanism disturbance of cartilaginous cells and of osteoblasts (Goodman et al. 1984). These zones can be best demonstrated in the long bones, although the temporary arrest of growth of course involves the entire skeleton (Steinbock 1976). With renewed growth and during its further course, the zones remain in place and appear therefore successively more and more distant from the epiphyseal level in the diaphyseal direction. They usually—and in some cases very early—undergo resorption and disappear; in other cases they remain apparent up to an advanced age (Garn and Schwager 1967). In tribute to the author who described them the lines are called Harris' lines.

Even this very schematic presentation of Harris' lines includes the features which it is necessary to remember with respect to their application in anthropological research. In the first place, these lines *never* represent the full registration of all periods of temporary arrest of growth. Their absence never means that the individual did not suffer from any disease or hunger period in his past history. The laws governing the persistence of Harris' lines up to adult age are unknown. It cannot be assumed that the most prominent or most recent Harris' lines are preserved because clinical studies prove this is not so.

A broad variety of factors provoke Harris' lines (Cohen and Armelagos 1984). Harris' lines are found after inflammation of the upper respiratory passages as well as after other infectious diseases, in diabetes, chronic anemias, chronic metal and other poisoning, and after surgical interventions. Animal experiments demonstrated that they are induced by protein and vitamin A deficiency, and by fasting in general (Acheson and MacIntyre 1958). During long-time follow-up of children it was found that the number of Harris' lines does not necessarily equal the number of illness periods (Gindhart 1969). In some cases the lines appeared even without any proved cause.

In clinical practice, Harris' lines in adults are not considered as clinically important and usually they are not mentioned in interpretation of radiological pictures. In recent years a new interest in Harris' lines was raised by studies in which the authors tried to use them for paleopathological purposes (Allison et al. 1974). Among studies which dealt

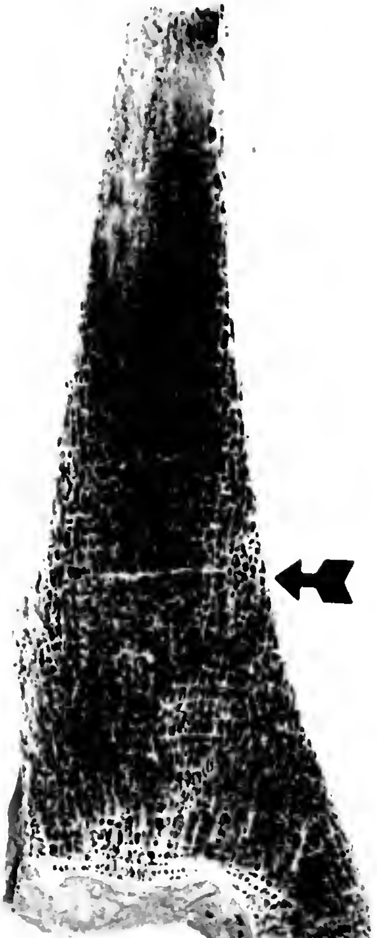


FIGURE 1. Prominent Harris' lines at a distance of 45 mm from distal articular surface of tibia. Photograph of section of bone.

very thoroughly from different points of view with the incidence of Harris' lines in ancient bone materials, we mention especially the studies of Wells (1961,1967) and Kuhl (1977). Calvin Wells introduced the Index of Morbidity, as the means to characterize a population group. The best preserved long bone of the skeleton is always selected for study, and the number of Harris' lines in the entire population studied is divided by the number of observed skeletons. Unfortunately, studies applying Wells' Morbidity Index do not mention if the number of Harris' lines counted includes only those which completely traverse the full width of the bone or those which are partially preserved. In our opinion, these incomplete remnants of Harris' lines signal the period of temporary growth arrest in the individuals past as importantly as the complete ones. In addition it is necessary to take x-ray pictures of the evaluated bones in two views; the discrete shadow of Harris' lines may be seen only in one projection.

As an example of the incidence of Harris' lines in a recent population we present the findings in a group of 160 men with x-ray pictures of the lower extremities examined for other than anthropological reasons in one region of Czechoslovakia. We followed the Harris' lines especially in the tibiae, where these lines appeared most prominently and where they could be recognized most securely. The average age of the men examined was 67.3 years, ranging from 49 to 88 years. With five exceptions only, the childhood of all these men occurred during the period of World War I, or the years close to it. It could be supposed that apart from the usual childhood infectious diseases, they were exposed to other similar provoking factors during their growth, especially dietary deficiency. Nevertheless, this study was not made to estimate the influence of different factors in the frequency of Harris' lines but to follow the Harris' lines preserved in a group of adult men at a certain individual age and with a similar childhood history.

In this group of 160 adult men, Harris' lines were found in only 35 (21.9%) cases. This would imply that nearly 80% of these men did not suffer from any serious illness or hunger period during World War I, which is hardly probable. Harris' lines were bilateral in 27 (77.1%) of the 35 men, on the right side only in 3 (8.6%) and on the left side in 5 (14.3%). The average age of the men with the Harris' lines was 68.5 years (ranging from 62 to 84 years). It did not differ significantly from the average age of men without Harris' lines. In most cases more than two Harris' lines were present. Lines nearest to the epiphysis were mostly complete; lines situated nearer to the center of the bone were often represented by their dorsally and tibially preserved parts only.

The distance of the Harris' line to the distal tibial articular surface was different in individual cases. In most cases it was 2.5–3.5 cm, but in several cases it measured 6 cm and exceptionally 10 cm. In bilateral findings the distance on the right and left side was equal. Only in 2.8% of men—quite sporadic—was it possible to identify Harris' lines not only in the distal parts of the tibiae but in the proximal ends, too. In one of these findings the distance between the first proximal Harris' line and the proximal articular surface of the tibia corresponded with the distance between the first distal Harris' line and the distal tibial articular surface. In all cases of proximal tibial Harris' lines, distal Harris' lines were present also. In only two cases did Harris' lines in the distal parts of the fibulae accompany those in the tibiae. Considering the total number of tibiae with preserved Harris' lines, these two fibula cases are really exceptional.

We studied the coincidence of Harris' lines and the persistence of the epiphyseal line in the distal end of the tibia. The result in our group showed that no less than 31 (88.6%) of the men with Harris' lines aged 62 to 84 also had preservation of the epiphyseal line at the same time. Only 4 men (11.4%) with Harris' lines failed to demonstrate a recognizable epiphyseal line. In men without Harris' lines, the epiphyseal line was visible in only 41 (32.8%). The association of simultaneous persistence of Harris' lines and of the epiphyseal line was statistically significant.

In conclusion, we summarize our opinions on the possibility of using Harris' lines in the paleopathological analysis of adults:

1. It is necessary to consider Harris' lines in the skeleton of an adult individual as an expression of a strikingly individual feature. The absence of Harris' lines can hardly represent secure evidence of the general state of health of the population because there are no identified rules of their preservation up to adult age.

2. Asymmetry of the occurrence of Harris' lines on the left and right side of long bones is so exceptional that it is possible to use either tibia for their demonstration and study.

3. In the evaluation of Harris' lines, it is necessary to base the result on x-ray pictures in two views to demonstrate incomplete lines.

4. It is possible to use the frequency of Harris' lines to characterize a certain population group if it is remembered that Harris' lines are polyetiological and that the problems of their persistence up to adult age are not solved.

5. The statistical significance of the coincidence of persistent Harris' lines with the epiphyseal lines in our group suggests very interesting relationships.

Literature cited

Acheson, R.M., and M.N. MacIntyre. 1958. The Effects of Acute Infection and Acute Starvation on Skeletal Development. A Study of Young Rats. *British Journal of Experimental Pathology*, 39:37–45.

Allison, M.J., D. Mendoza, and A. Pezzia. 1974. A Radiographic Approach to Childhood Illness in Precolumbian Inhabitants of Southern Peru. *American Journal of Physical Anthropology*, 40:409–416.

Cohen, M.N., and G.J. Armelagos. 1984. Paleopathology at the Origins of Agriculture: Editors' Summation. In M.N. Cohen and G.J. Armelagos, eds., *Paleopathology at the Origins of Agriculture*, 585–601. New York: Academic Press.

Garn, S.M., and P.M. Schwager. 1967. Age Dynamics of Persistent Transverse Lines in the Tibia. *American Journal of Physical Anthropology*, 27:375–377.

Gindhart, P.S. 1969. The Frequency of Appearance of Transverse Lines in the Tibia in Relation to Childhood Illness. *American Journal of Physical Anthropology*, 31:17–22.

Goodman, A.H., D.L. Martin, and G.J. Armelagos. 1984. Indications of Stress from Bone and Teeth. In M.N. Cohen and G.J. Armelagos, eds., *Paleopathology at the Origins of Agriculture*, 13–49. New York: Academic Press.

Harris, H.A. 1933. *Bone Growth in Health and Disease*. London: Oxford University Press.

Kuhl, I. 1977. *Die Leichenbrande vom Brandgraberfeld auf der Dune Wissing, Gem. Halden*, 213. Kiel, Germany: Kreis Wesel.

Steinbock, R.T. 1976. *Paleopathological Diagnosis and Interpretation*, 46–55. Springfield, Ill.: Charles C Thomas.

Wells, C. 1961. A New Approach to Ancient Disease. *Discovery*, 22:526–531.

———. 1967. A New Approach to Paleopathology: Harris' Lines. In D. Brothwell and A.T. Sandison, eds., *Diseases of Antiquity*, 390–404. Springfield, Ill.: Charles C Thomas.

SUMMARY OF AUDIENCE DISCUSSION: The "disappearance" of Harris' lines in adults may be an illusion, resulting from their obscuration by the thickening of the growing diaphyseal cortex; they may become apparent again ("reappear") following demineralization of the bone in the osteoporotic patient. It is also important to remember that only those who suffer a transient pulse of illness and recover promptly will develop them; the chronically ill and the very healthy individuals do not develop Harris' lines. Correlation between development of Harris' lines and dental hypoplasia is often not high because each responds to different stimuli or at least to different degrees, but if one compares *all* microscopic dental defects the correlation is good.

Medical ceramic representation of nasal leishmaniasis and surgical amputation in ancient Peruvian civilization

Oscar Urteaga-Ballon

From 3000 years ago until the Spanish conquest, the central part of the mighty Andean Mountains of South America was the center of some of the most splendid, ancient American civilizations. The Inca empire was the last of those civilizations. It began in the Peruvian lands and spread throughout parts of Ecuador, Colombia, Bolivia, Argentina and Chile (Larco-Hoyle 1938–1939; Mason 1957; Tello and Mejia 1960; Bird 1962; Sawyer 1966). The Incas considered themselves the direct descendants of the sun and moon, which they worshipped as their gods.

The ancient Peruvians did not leave a written language, but in their metal art, pottery and textiles, they did leave a graphic sculptural language, which, if one is prepared to do so, can be read like the pages of a book. In the north coast valleys of Peru began one of the most splendid civilizations of ancient Peruvians. The Mochica culture had left beautiful, realistic ceramic sculptures, which covered broad aspects of Mochica life, including special medical collections. In 1968 we published a complete review of some chapters of the sexual behavior of ancient Peruvians as shown in their ceramic art (Urteaga-Ballon 1968). These ancient people were powerful warriors with large conquering armies, but during periods of peace they developed an outstanding civilization rich in artistic culture.

Much of what we know about the remarkable medical history of pre-Columbian Peru is the result of research on the ceramic pieces of the ancient Peruvian civilizations. Many infectious diseases have been portrayed in ceramic art. As a result of chronic warfare, many people suffered serious lesions which demanded different types of surgical operations. Mutilation and amputation of the limbs were frequent. The most dramatic representations of the medical pottery are the cranial operations (trephination), which were conducted by members of the Paracas civilization beginning more than 2000 years ago.

Two different types of ceramic medical art serve to illustrate this rich and important source of information on ancient medical traditions in the New World. We first present some examples of ceramic art showing evidence of leishmaniasis, a condition well known in modern Peru and often seen by me during my career as a pathologist in Peru. We follow this with a brief review of a few cases of ceramic art exhibiting amputated limbs and the prosthetic devices that were often made to assist the patient with at least some use of the limb following healing of the amputation stump.

Nasal leishmaniasis

Mucocutaneous leishmaniasis is a chronic infectious disease caused by the protozoan *Leishmania braziliensis*. The disease was endemic in the ancient Peruvian lands, as it is found in the ceramics of those cultures.

Between 1962 and 1974 in the Museum of Paleopathology of Lima, Peru, I studied 67 ceramic pieces in which the pre-Columbian Peruvian craftsmen represented different lesions of infectious and parasitic diseases. In this paper I show four ceramic representations of mucocutaneous leishmaniasis representing different stages of lesions affecting the nasal and oral cavities. As a comparative reference I include two other paleopathological ceramic representations which prove the skill and knowledge of the ancient Peruvian physicians.

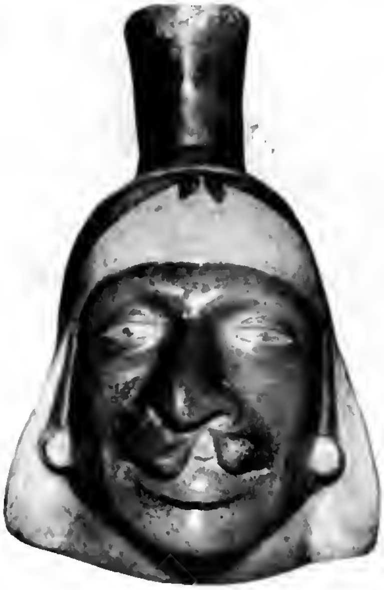
Figure 1 corresponds to the famous portrait-head vessel of the Mochica ceramic described by Larco-Hoyle (1938–1939) and Sawyer (1966). It represents a congenital cleft of the upper palate and lip. Figure 2 represents a punitive case with surgical mutilation of the nose and lips. Both pieces are examples of the graphic realism of the ancient Peruvian craftsmen.

Figure 3 is also a Mochica portrait-head vessel which shows the first stage of mucocutaneous leishmaniasis. A large ulcer has destroyed the left wall of the nose. This is a typical lesion of the disease. Figure 4 corresponds to an advanced stage of the infection. The necrotic lesions have destroyed both sides of the nares walls and the infection has also invaded the left side of the upper lip. If you compare the graphic representations of these four cases you cannot miss the diagnosis. In our time the microscopic finding of the protozoan organism makes the diagnosis definitive, but the gross lesion is extremely clear in the endemic zones of this disease. Figure 5 corresponds to the third stage of the leishmaniasis infection. The necrotic lesion has destroyed not only the subcutaneous tissues of the nose, but also completely destroyed the nasal septum, rounding and widening the nasal cavity. The upper lip has also disappeared but there are no lesions in the dental alveolar process.

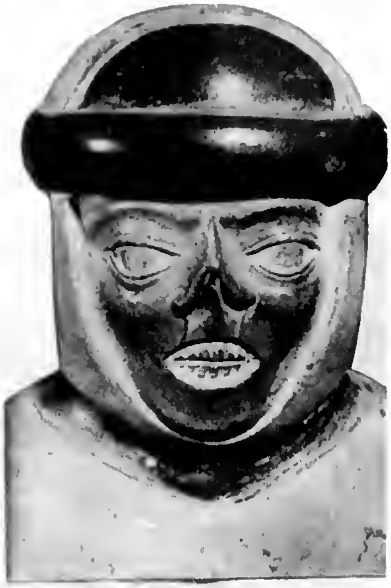
The last stage of the disease is characterized by a complete destruction of the nasal cavity with partial resorption of the hard palate bone, which produced a direct opening between the nasal and the oral cavities. Figure 6 shows one of these cases. The destruction of the frontal part of the palate bone also produces the loss of the upper incisor teeth.

We have found similar lesions in the skull of some ancient Peruvian mummies. Figure 7 corresponds to one of these skulls and shows a complete destruction of the nasal septum and the resorption of the lateral border lines of the nasal cavity, which appear unusually round and wide. The bone resorption also included the anterior part of the palate bone. Figure 8 corresponds to a Mochica ceramic which represents the frontal view of the face of a normal skull. The Peruvian craftsmen have represented the anatomical proportion of size and shape of the nasal and oral cavities, including the nasal septal bone.

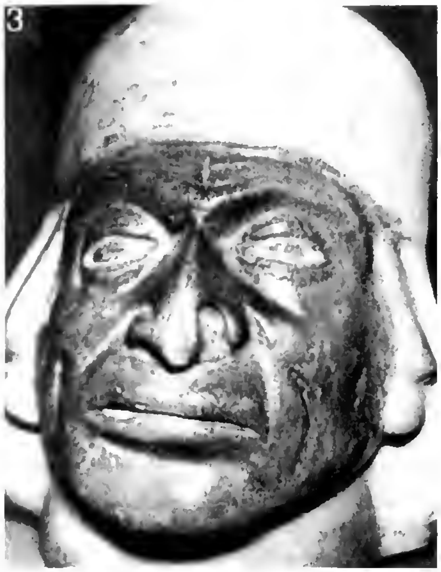
1



2



3



4

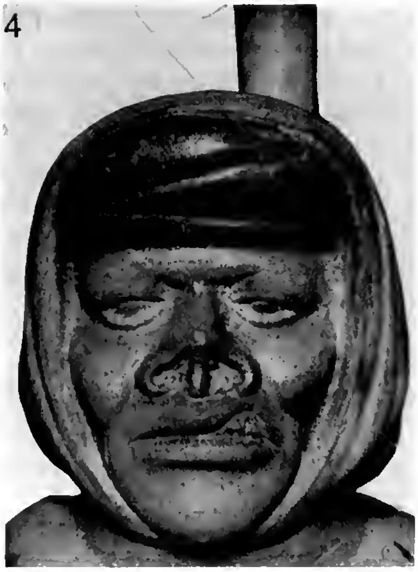


FIGURE 1. Ceramic representation of a case of cleft palate and lip. Peruvian Mochica culture. Lima, Peru.

FIGURE 2. Ceramic representation of a punitive mutilation of nose and lips. Peruvian Mochica culture. Lima, Peru.

FIGURE 3. Ceramic representation of a mucocutaneous leishmaniasis. Mochica portrait-head vessel shows an ulcerative lesion of left wall of nose. Peruvian Mochica culture. Lima, Peru.

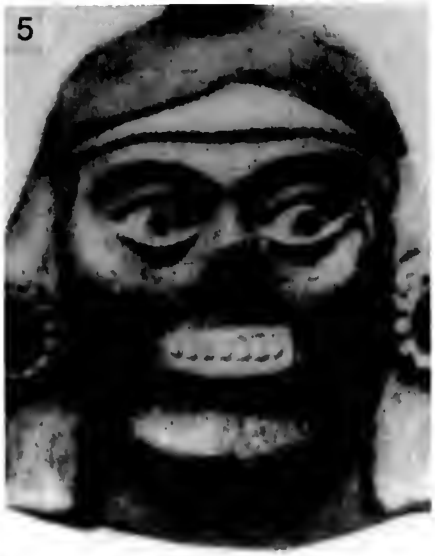
FIGURE 4. Ceramic representation of a case of mucocutaneous leishmaniasis. Second stage. Necrotic lesions have destroyed both sides of nose and upper lip. Peruvian Mochica culture. Lima, Peru.

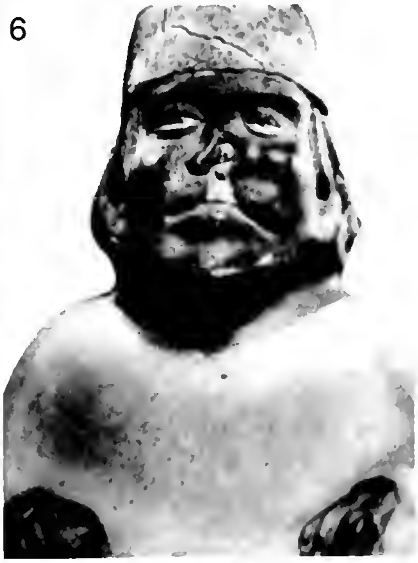
Some paleopathologists have described these nasal and oral lesions as pathognomonic of a form of leprosy. Ortner and Putschar (1981), in their classic paleopathology book, have commented on the findings of different investigators in skulls found in a medieval Danish leprosy cemetery in 1953. All of them think that these nasal and oral lesions are characteristic of leprosy. However, they did not discard the possibility that similar lesions could be found in tertiary syphilis and in lupus vulgaris.

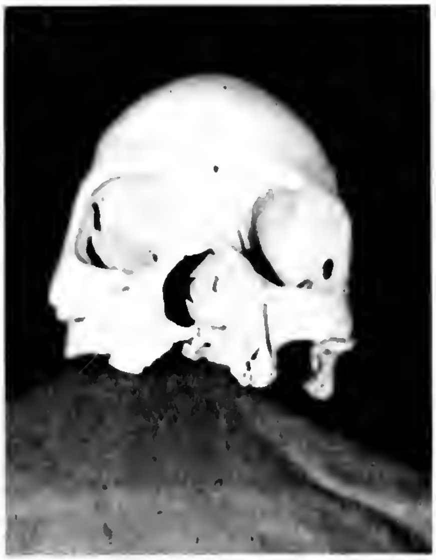
My experience in more than thirty years as a pathologist of tropical infectious diseases is different. Today, in the jungle of the Amazon River, leprosy is endemic. We have seen more than 300 patients with leprosy there. Many of them were in the acute lepromatous stage and others had chronic, advanced lesions. Most of those patients had microconfluent, nodular lesions of the nose. Some of them also had granulomatous lesions in the nasal septum with ulceration and perforation of the septum, but we did not find one case in which the nasal septum and the palate bone were totally destroyed giving the classic appearance of a round, wide and large nasal cavity with nasal-oral communication.

However, in the jungle of the Amazon River, mucocutaneous leishmaniasis is also endemic. We have studied almost 100 cases of these patients. More than 20 were in an advanced stage, showing the typical destructive lesions of the nasal septum and the palate bones, exactly like the graphic representations in the ceramics of the ancient Peruvians.

Mitsuda and Ogawa (1937) reviewed 150 autopsies in the Aisein National Leprosarium in Japan. On this subject they said, "Some leprotic periostitis and involvement of the osseous structure are found in various bones, such as the tibia and the phalanges." However, they did not describe any special naso-







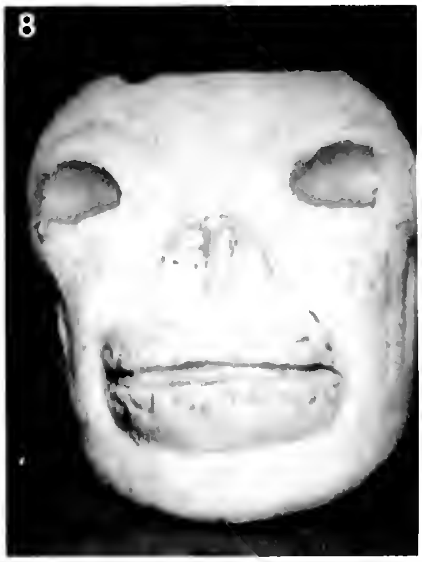


FIGURE 5. Ceramic representation of a case of mucocutaneous leishmaniasis. Third stage. Infection has destroyed nasal septum, rounding and widening nasal cavity. Peruvian Mochica culture. Lima, Peru.

FIGURE 6. Ceramic representation of a case of mucocutaneous leishmaniasis. Last stage. Complete destruction of nasal cavity with partial resorption of hard palate bone. Peruvian Mochica culture. Lima, Peru.

FIGURE 7. Ancient Peruvian skull with a case of mucocutaneous leishmaniasis, showing classical nasal and oral lesions of this disease. Nose is round and wide for total destruction of nasal bones. Part of palate bone also destroyed. Skull of Peruvian mummy. Lima, Peru.

FIGURE 8. Ceramic representation of face of a normal skull showing anatomical proportion of size and shape of nasal and oral cavities, including septum. Peruvian Mochica culture. Lima, Peru.

oral destructive lesions. Kean and Childrees (1942) made a summary of 103 autopsies of leprosy cases at the Gorgas Hospital, Canal Zone, Panama. They described the leprosy osseous lesions as follows: "Amputation of one toe 12, one or more fingers 14, leg 9, foot 1; absorptions of fingers 19, toes 18, feet 3; gangrene of the toe 2, miscellaneous lesions 7." They also did not find the naso-oral destructive lesions. Desikan and Job (1968) in the General Hospital in Vellore, India, reviewed 37 autopsies of leprosy. They did not describe one case of naso-oral destructive lesions.

Enna (1968), in Ryukyu Island in Okinawa, studied 996 leprosy patients with lesions showing advanced deformity; 15.2% had nasal deformities, but not one had the osseous destruction of the oral and nasal cavities. Bernard and Vazquez (1973) studied 60 necropsies with similar results.

However, other leprologists have found some degree of destructive lesions in the nasal cavity of leprosy patients. Powell and Swan (1955), in the National Leprosarium at Carville, Louisiana, in advanced cases of lepromatous leprosy found that ulceration and perforation of the nasal septum were common, with destruction of nasal cartilage and bone resulting in varying degrees of "saddle" deformity. Kumar et al. (1979), in the Leprosy Clinic in Chandigarh, India, described 25 selected patients. Of these 88% had nasal obstruction, while 48% and 32% had ulceration and perforation of nasal septum. Barton et al. (1982), in the Victoria Hospital in Dichpalli, India, studied 62 patients of lepromatous leprosy; one of them showed a completely perforated septum.

Furthermore, in a North American textbook of pathology, Marcial Rojas and Kissane (1985) made more radical affirmations. They said that mucocutaneous leishmaniasis produces "extensive destruction of the soft and underlying hard tissues of the nose and pharynx producing severe mutilation of the face."

9

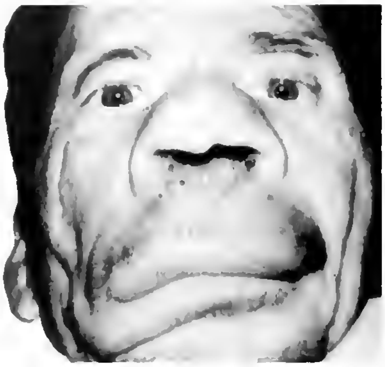


FIGURE 9. Mucocutaneous leishmaniasis. Total destruction of septum and deformation of nose and lips. Photo from Atlas of Tropical Pathology (Binford and Connor 1976:207).

10

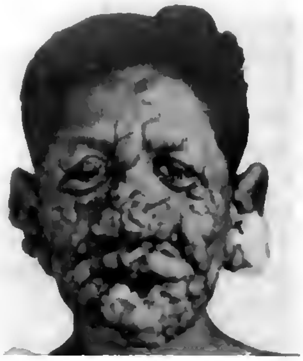


FIGURE 10. Lepromatous leprosy. Tremendous deformation of nose and lips, without destruction of nasal septum, in contrast to case of leishmaniasis in Figure 9. Photo from Atlas of Tropical Pathology (Binford and Connor 1976:261).

Finally, Binford and Connor (1976) make the most complete revision of leprosy and leishmaniasis. In Section 6 of this atlas, Binford and Meyers published 76 photographs of gross and microscopic lesions of all types of leprosy. Despite the fact that some of their cases show tremendous deformation of the nose and the lips, none of these patients show the destructive bone lesions in their nasal-oral cavities. However, in Section 7 of the same atlas Connor and Neafie show one case of mucocutaneous leishmaniasis with tremendous destructive lesions. The authors say, "A Brazilian with mucocutaneous leishmaniasis has a destroyed nasal septum and deformed nose and lips" (F-3-B-6, page 261).

We have included as comparison two figures from this atlas. Figure 9 shows a case of mucocutaneous leishmaniasis with a massive destruction of the nasal septum and deformities of the nose and lip. Figure 10 corresponds to a case of lepromatous leprosy with tremendous deformation of the nose and the lips, but with no destruction of nasal-oral bones.

In medicine nothing is exact; we cannot say with certainty that the granulomatous nasal-oral destruction is pathognomonic of one specific disease. According to its ancient and modern incidence, mucocutaneous leishmaniasis occupied first place. Lepromatous leprosy is in second place. Tertiary syphilis and yaws are in third place. Rhinoscleroma and other infectious diseases rarely produce this anatomical lesion.

More important in this academic, historical discussion is the fact that ancient Peruvian physicians had left indisputable evidence of their knowledge of some of the most complex chapters of medicine. Their graphic ceramic representations are unquestionable.

Surgical amputation and limb prostheses

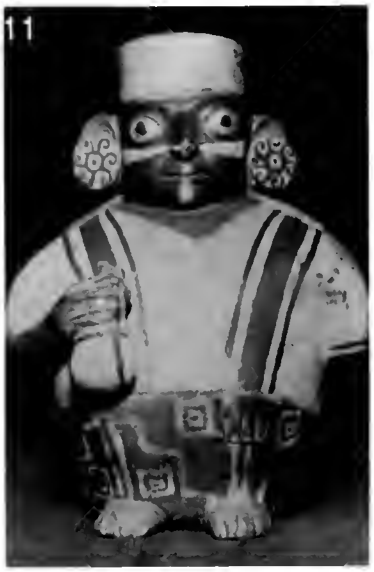
We have studied 65 ceramic pieces in which the Mochica craftsmen represented traumatic medical surgery.

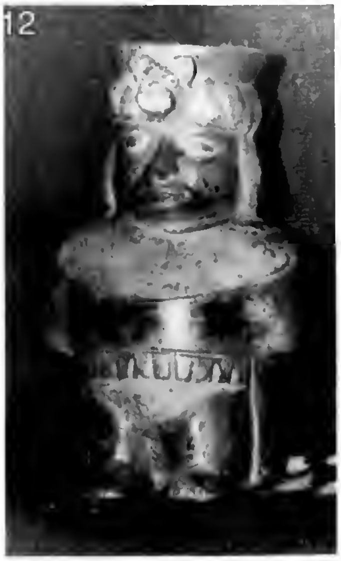
Among them were prostheses of the upper and lower extremities. We selected 12 of these cases, the first 4 representing different types of limb amputations and the rest being a complete sequence of the prostheses operations.

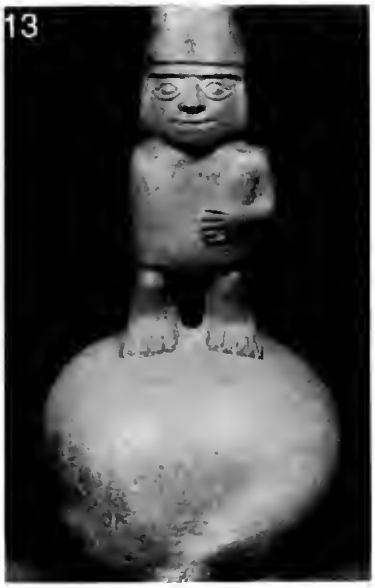
Figure 11 represents a case of an amputation of the left arm. The person uses a short cape which had a false hand at the bottom. Figure 12 shows a case of a bilateral arm amputation. Figure 13 reveals an extensive operation with the removal of the complete right arm. A more radical surgical operation appears in Figure 14: the patient has suffered bilateral arm and foot amputations; it is possible that these surgical mutilations were done as a punitive sentence because there are also mutilations of the upper lips and part of the wall of the nose.

In the next eight ceramic pieces the craftsmen represent a successful sequence of a prosthesis of the leg and arm. Figure 15 shows the traumatologist examining a patient's leg previous to the operation. Figure 16 demonstrates the second stage. The patient has been operated upon recently. The two bones of the left leg appear through the surgical wound. Figure 17 shows another patient who has been operated on some time ago. The wound appears completely healed. Figures 18 and 19 correspond to the fourth stage of the prosthesis operation. Two patients are testing the prosthesis apparatus with the hand opposite to the amputated leg. In Figure 20 another patient appears with the prosthesis attached to his left leg, and Figure 21 reveals the last stage of this traumatologic operation. The patient is walking with the help of a cane and the prosthesis is attached to his left leg.

We did not find a complete sequence of the prosthesis of the arms. However, we found two instances of patients with the prosthesis attached to the arms. Figure 22 shows one of these cases. The patient was a blind man who had a pros-







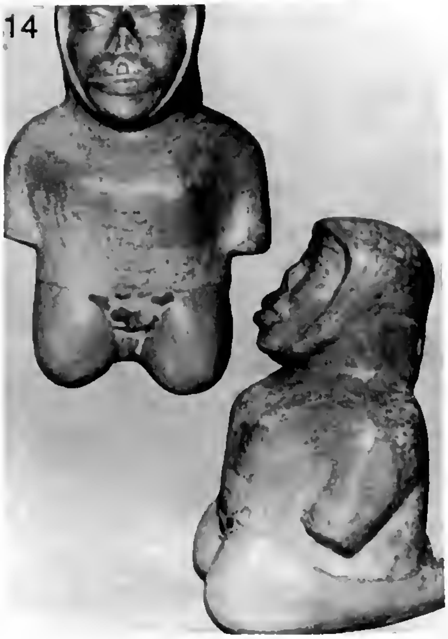


Figure 11. Amputation of left arm. Mochica culture, ceramic. Lima, Peru.

Figure 12. Bilateral arm amputation. Mochica culture, ceramic. Lima, Peru.

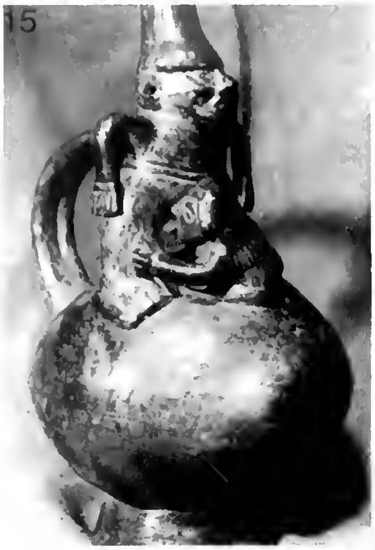
Figure 13. Extensive surgical operation of right arm. Mochica culture, ceramic. Lima, Peru.

Figure 14. Bilateral arm and foot amputations. Mochica culture, ceramic. Lima, Peru.

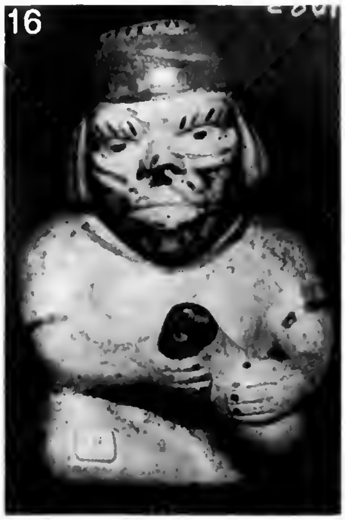
thesis attached to his right arm. Nine ceramic pieces of the same Mochica culture represent some prosthesis apparatus. They correspond to the legs, arms and hands. One of these pieces represents a surgical knife held between the fingers. The surgical knife was named "tumi" and the surgeons used it in different types of operations. Some ceramic pieces represent the surgeons with the tumi knife in their hand during a cranial trephine.

The representations of lesions and prostheses, carved in Peruvian ceramics more than 2000 years ago, are graphic medical lessons proving the knowledge and skill of pre-Columbian physicians. These ceramic sculptures illustrate an important chapter in the history of medicine.

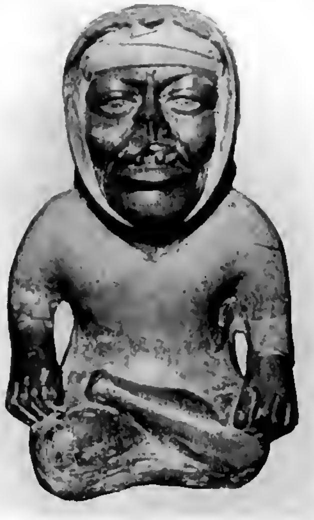
15



16



17



18

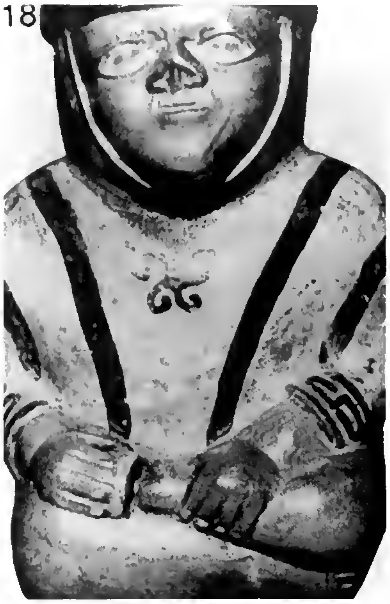


FIGURE 15. Prosthesis of the leg. Traumatologist examining patient's leg previous to operation. Chimu culture, ceramic. Lima, Peru.

FIGURE 16. Prosthesis of the leg. Second stage. Patient has been operated upon recently. Two bones of left leg appear through surgical wound. Mochica culture, ceramic. Lima, Peru.

FIGURE 17. Prosthesis of the leg. Third stage. Wound appears completely healed. Mochica culture, ceramic. Lima, Peru.

FIGURE 18. Prosthesis of the leg. Fourth stage. Patient is testing prosthesis apparatus with hand opposite to amputated leg. Mochica culture, ceramic. Lima, Peru.

Literature cited

Barton, R., J. Rees, C. McDougall, and G. Ellard. 1982. The Nose in Lepromatous Leprosy: Bacteriological and Histopathological Studies of Patients Treated with Dapsone Monotherapy. *International Journal of Leprosy,* 50:58–67.

Bernard, J.C., and C.A. Vazquez. 1973. Study of Sixty Necropsies. *International Journal of Leprosy,* 41:94–101.

Binford, C., and D. Connors. 1976. *Pathology of Tropical and Extraordinary Diseases.* Washington, D.C.: Armed Forces Institute of Pathology.

Bird J.B. 1962. *Art and Life in Ancient Peru.* An Exhibition in Curator II, New York.

Desikan, K.V., and C.K. Job. 1968. A Review of Postmortem Findings in 37 Cases of Leprosy. *International Journal of Leprosy,* 36:32–44.

Enna, C.D. 1968. A Survey of Leprous Deformities in Ryukyu Island. *International Journal of Leprosy,* 36:271–281.

Kean, B.H., and M.E. Childrees. 1942. A Summary of 103 Autopsies of Leprosy Patients on the Isthmus of Panama. *International Journal of Leprosy,* 10:51–59.

Kumar, S., K. Malik, B. Kuman, M. Singh, and R. Chakravarty. 1979. Respiratory System Involvement in Leprosy. *International Journal of Leprosy,* 47:18–25.

Larco-Hoyle, R. 1938–1939. *Los Mochicas.* Lima, Peru.

Marcial Rojas, R.A., and J.M. Kissane. 1985. Mucocutaneous Leishmaniasis. In J.M. Kissane, ed., *Anderson's Pathology,* 411–412. St. Louis: C.V. Mosby.

Mason, J.A. 1957. *The Ancient Civilization of Peru.* [Harmondsworth, Middlesex]: Penguin Books.

Mitsuda, K., and M. Ogawa. 1937. A Study of One Hundred and Fifty Autopsies on Cases of Leprosy. *International Journal of Leprosy,* 5:53–60.

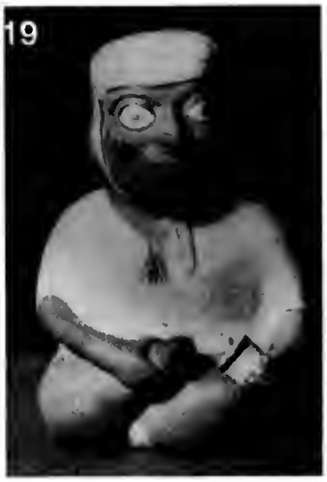
Ortner, D.J., and W.J.G. Putschar. 1981. Identification of Pathological Conditions in Human Skeletal Remains. *Smithsonian Contributions to Anthropology,* 28:176–180. Washington, D.C.: Smithsonian Institution Press.

Powell, S.C., and L.L. Swan. 1955. Leprosy: Pathologic Changes Observed in Fifty Consecutive Necropsies. *American Journal of Pathology,* 31:1131–1148.

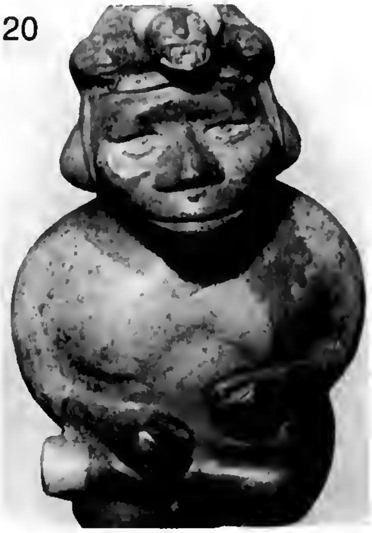
Sawyer, A.R. 1966. *Ancient Peruvian Ceramics. The Nathan Cummings Collection.* New York: The Metropolitan Museum of Art.

Tello, J.C., and X.T. Mejia. 1960. *Chavin, Cultura Matriz de al Civilizacion Andina.* Lima, Peru: Imprenta de la Universidad de San Marcos.

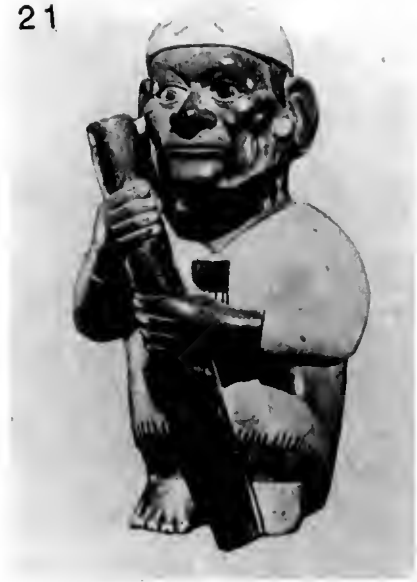
Urteaga-Ballon, O. 1968. *Interpretation of Sexuality in the Ceramic Art of Ancient Peru.* Lima, Peru: Museo de Paleopatologia, Hospital "2 de Mayo."



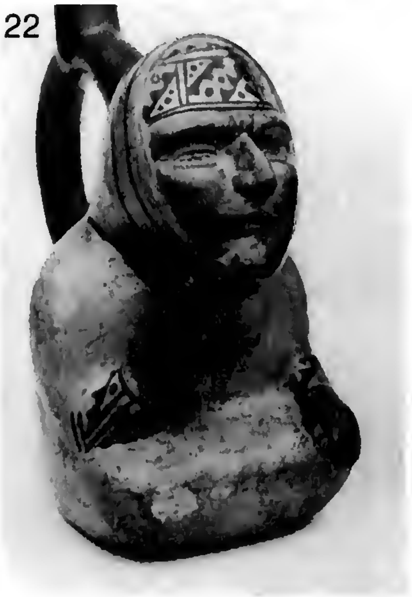
19



20



21



22

FIGURE 19. Prosthesis of the leg. Fourth stage. Patient is testing prosthesis apparatus with hand opposite to amputated leg. Mochica culture, ceramic. Lima, Peru.

FIGURE 20. Prosthesis of the leg. Fifth stage. Prosthesis apparatus attached to patient's left leg. Mochica culture, ceramic. Lima, Peru.

FIGURE 21. Prosthesis of the leg. Last stage. Patient is walking with help of a cane and prosthesis is attached to his left leg. Mochica culture, ceramic. Lima, Peru.

FIGURE 22. Prosthesis of right arm. Patient was a blind man who had a prosthesis attached to his right arm. Mochica culture, ceramic. Lima, Peru.

Population Studies

Temporal variation in femoral cortical thickness of North American Plains Indians

Douglas W. Owsley

Cortical bone growth during periods of juvenile gain and later adult loss has been documented in long-term studies of populations in Central and North America (Garn 1970). Several variables affect tubular bone cortical thickness including age, sex, and nutrition. Changes in the bone envelope are surface-specific and reflect the combined response of subperiosteal apposition and endosteal resorption or apposition.

Simple malnutrition slows the rate of bone growth and can lead to the formation of less bone (Garn 1970,1972). Subperiosteal growth depends more on caloric sufficiency; protein seems to be less of a limiting nutrient. The effect of protein-energy malnutrition on cortical thickness is seen primarily on the inner bone surface. Endosteal resorption induced by kwashiorkor or marasmus can reduce the cortical wall to a thin shell with an enlarged medullary cavity. As much as 40% of the preformed bone can be lost, even though the external bone size remains relatively unaffected (Garn 1970; Garn et al. 1964,1969). Recovery-related catch-up growth or surface repair through endosteal replacement is limited (Garn 1966).

Although most studies of cortical bone mass have focused on the living, these observations can be applied to the analysis of archeological samples. Bioarcheological interpretations of past subsistence patterns have used cortical bone growth and thickness as an indicator of nutritional status (Cashion 1987; Cook 1979; Huss-Ashmore 1978; Hummert 1983; Hummert and Van Gerven 1983; Keith 1981; Owsley 1985). Cashion (1987) and Owsley (1985) have reported age-controlled adult femoral midshaft cortical thicknesses in temporally sequential North American Arikara Indians of South Dakota. Comparison of bone cortex data for villagers representing three archeological variants of the Plains Village Coalescent Tradition (Extended, A.D. 1550–1675; Post-Contact, A.D. 1675–1780; and Disorganized, A.D. 1780–1845) revealed statistically significant differences. Relative to the earlier Extended Coalescent and later Disorganized Coalescent samples, the Post-Contact Variant sample showed more cortical bone, presumably reflecting greater success in meeting village nutritional needs.

This presentation examines the historical patterning of these temporal differences. Specific objectives are to define the surface-specific nature of the differences reported between archeological variants and to apply greater temporal control using chronological periods of shorter duration. Cortical thickness is a composite measure determined by both the medullary cavity and the total subperiosteal diameters. Are changes in cortical thickness caused by changes in only one or both of these dimensions? Greater temporal control was made possible by obtaining data for related sites and by sorting this larger data base into smaller temporal units representing Late Prehistoric (A.D. 1600–1650), Early Protohistoric (1650–1740), Late Protohistoric (1740–1795), and Historic (1795–1832) period sites. Comparison of these four samples provides a clearer representation as to the timing of cortical bone change during the Post-Contact period. An appreciation of this timing is essential for understanding the nutritional impact of contact-related historical events.

Materials and methods

Sample sizes and approximate dates of the 12 archeological sites included in this analysis are presented in Table 1. In order to avoid age-related cortical bone involution, the analysis was limited to femora of young adults aged 16–35 years, giving a total of 110 males and 134 females. The bones were x-rayed in a standardized posterior-anterior projection using a Kodak single lanex, fine screen X-omatic cassette. Only bones in good condition were measured with preference given the left side when available.

Two midpoint cross-sectional observations, total subperiosteal diameter (T) and width of the medullary cavity (M), were measured with a Helios dial caliper to 0.1 mm (cf. Garn 1970). Three composite variables were derived from these measurements: cortical thickness (C), Nordin's Index (NI), and cortical area (CA). Cortical thickness was calculated as $C = T - M$. The value C represents the combined or net thickness of the medial and lateral walls. Nordin's Index (NI) or score was determined as NI = C/T. This score is essentially two-dimensional and describes the proportion of the total width attributed to the cortex. Cross-sectional area measurements were calculated as CA = 0.785 ($T^2 - M^2$) (Garn 1970). This procedure assumes that the femur has an approximately circular cross-sectional geometry in both the endosteal and subperiosteal surfaces. The assumption of a cylindrical shape is not necessarily valid for the femur (Van Gerven et al. 1969). Nevertheless, it is useful to consider cortical area as another indicator of bone status because this value represents absolute bone mass and potential calcium reserves (J. Dequeker, pers. comm.). As judged from studies based on the second metacarpal, cortical area shows the highest correlation with the ash content of the bone relative to the other measurements and indices (Dequeker 1976). Maximum femoral lengths (ML) were measured on the radiographs using a metric ruler. Sample differences were evaluated using the general linear models analysis of variance statistic presented in SAS (1985).

Results

Sample means and standard deviations are presented in Table 2 by sex for each of the four time periods. The means for cortical thickness and Nordin's Index are illustrated in Figures 1 and 2. Results of the analysis of variance comparisons by sex and time period are given in Table 3. As expected, sex differences are highly significant for the three primary variables (i.e., M, T, ML) and also for cortical thickness and area, but not for Nordin's Index. Temporal differences are evident in each variable with the exception of maximum length. The femoral lengths of these four samples are similar. In con-

TABLE 1. Archaeological sites, sample sizes and time periods

Site	*Site number*	*N* *Males*	*Females*	*Date range*	*Time period*
Leavenworth	39CO9	13	13	1802 - 1832	Historic
Leavitt	39ST215	3	1	1784 - 1792	Late Protohistoric
Cheyenne River	39ST1	1	1	1740 - 1795	Late Protohistoric
Stony Point Village	39ST235	3	6	1740 - 1795	Late Protohistoric
Four Bear	39DW2	3	10	1758 - 1774	Late Protohistoric
Dinehart Villae	39LM33	6	2	1725 - 1760 ?	Early Protohistoric
Larson	39WW2	24	34	1679 - 1733	Early Protohistoric
Oahe Village	39HU2	6	1	1650 - 1725	Early Protohistoric
Swan Creek	39WW7	15	7	1675 - 1725	Early Protohistoric
Sully	39SL4	14	20	1650 - 1733	Early Protohistoric
Mobridge 2	39WW1	11	24	1675 - 1700	Early Protohistoric
Mobridge 1, 3	39WW1	7	7	1600 - 1650	Late Prehistoric
Rygh	39CA4	4	8	1600 - 1650	Late Prehistoric

TABLE 2. Means and standard deviations for femoral cortex and maximum length measurements by sex and time period

	Late Prehistoric			*Early Protohistoric*			*Late Protohistoric*			*Historic*		
Variable[a]	*N*	*Mean*	*S.D.*	*N*	*Mean*	*S.D.*	*N*	*Mean*	*S.D.*	*N*	*Mean*	*S.D.*
MALE												
M	11	11.79	1.37	76	12.24	1.92	10	13.50	2.20	13	13.42	1.95
T	11	25.29	2.12	76	26.94	1.55	10	28.09	1.20	13	27.64	1.51
C	11	13.5	2.15	76	14.69	1.55	10	14.59	1.67	13	14.22	2.04
NI	11	0.53	0.06	76	0.55	0.06	10	0.52	0.07	13	0.51	0.06
CA	11	394.84	79.92	76	450.94	51.85	10	473.92	44.18	13	457.27	63.98
ML	8	466.13	13.93	66	466.85	22.22	9	481.22	20.21	12	470.75	24.07
FEMALE												
M	15	11.44	1.68	88	10.57	1.60	18	11.58	2.10	13	12.95	1.53
T	15	24.01	2.32	88	24.12	1.54	18	24.84	1.94	13	25.00	1.32
C	15	12.57	1.38	88	13.55	1.51	18	13.26	1.95	13	12.03	1.62
NI	15	0.52	0.04	88	0.56	0.06	18	0.53	0.07	13	0.48	0.06
CA	15	351.79	62.59	88	368.76	49.07	18	378.51	64.17	13	358.17	51.08
ML	12	431.83	21.77	76	429.47	17.01	17	431.71	17.34	11	433.45	18.69

a. M, medullary cavity diameter; T, total subperiosteal diameter; C, cortical thickness; NI, Nordin's index; CA, cortical area; ML, maximum length.

trast, marked sample differences are present in the amounts of cortical bone, as measured by the variables C, NI and CA. The separate patterns reflected by male and female measurements reveal marked correspondence. Mean values for C and NI for the Early Protohistoric period represent the high peaks for this time series. Cortical thickness increased in both sexes during the transition from the Late Prehistoric to the Early Protohistoric period. These gains, however, were only temporary and not sustained in the more recent time periods. Later samples have reduced cortical thicknesses. Both sexes correspond in this trend, although females reveal the most dramatic loss of tubular bone.

Sample differences in C and NI reflect net changes in both the medullary cavity diameter and the total subperiosteal diameter. In females, medullary widths decreased during the Early Protohistoric period. Late Protohistoric and Historic period female values returned to the Late Prehistoric average diameter and then greatly surpassed this base, more closely approximating the larger diameters of males. Males during the Late Protohistoric and Historic periods are characterized by larger medullary cavity diameters than found decades earlier in Arikara populations. Total subperiosteal diameters also show temporal change with mean values for T increasing through time, especially in males. In terms of estimated cortical areas, these small increases in T diminish the effect of the linear increases in M, the smaller diameter. In both sexes, calculated cortical areas show the largest increase with the transition from the Late Prehistoric to the Early Protohistoric period. Because of the increase in T, cortical areas continue to increase through the Late Protohistoric period, followed by decline during the early Historic period. In the most recent period, male and female cortical areas, although approaching the early 17th century base line (especially in females) are still higher than during the Late Prehistoric Period.

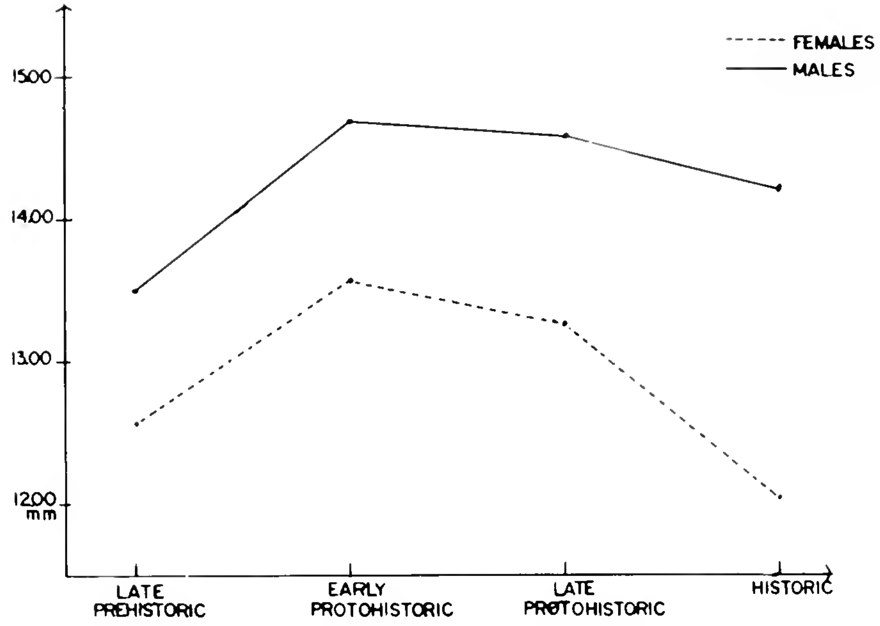


FIGURE 1. Femur midshaft cortical thickness by time period

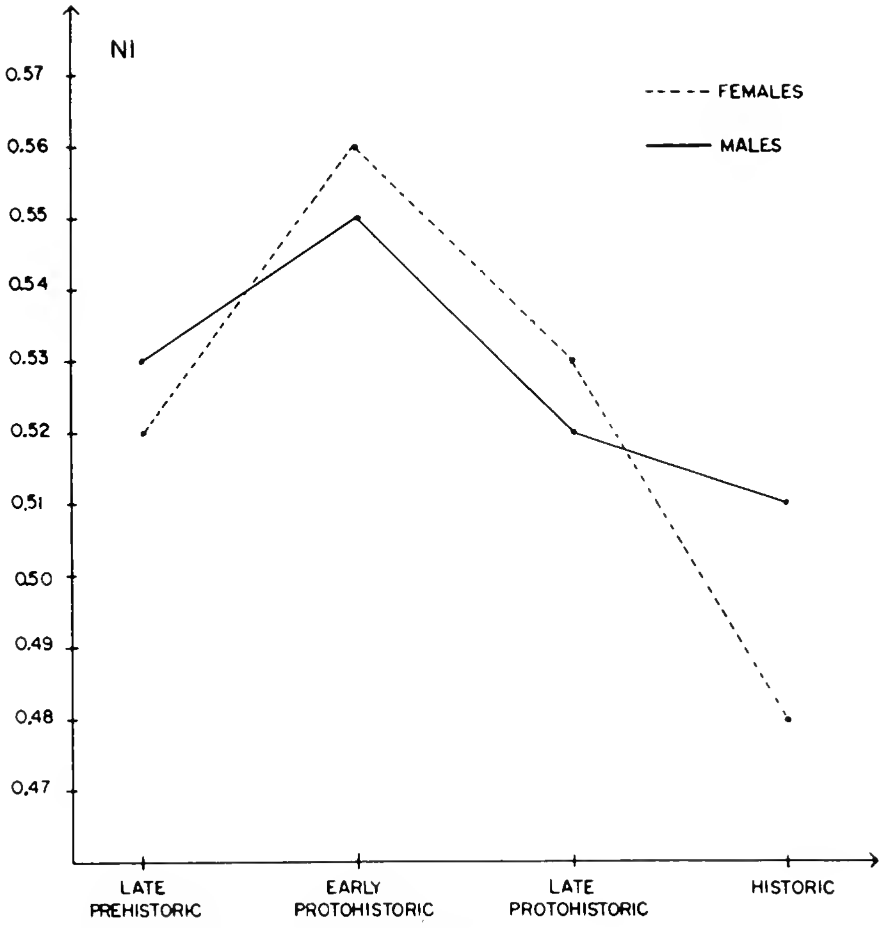


FIGURE 2. Temporal variation in Nordin's Index

TABLE 3. Two-factor analysis of variance of femur measurements by time period and sex

Variable	*Source*	*DF*	*Sum of squares*	*F*	*PR > F*
M	Model	7	226.61	10.21	0.0001
	Time	3	89.81	9.44	0.0001
	Sex	1	39.04	12.31	0.0005
	Interaction	3	17.49	1.84	0.14
	Error	236	748.57		
	TOTAL	243	975.18		
T	Model	7	495.64	26.41	0.0001
	Time	3	56.23	6.99	0.0002
	Sex	1	202.78	75.63	0.0001
	Interaction	3	15.35	1.91	0.13
	Error	236	632.76		
	TOTAL	243	1128.40		
C	Model	7	144.62	7.83	0.0001
	Time	3	42.25	5.34	0.001
	Sex	1	63.87	24.21	0.0001
	Interaction	3	6.89	0.87	0.46
	Error	236	622.45		
	TOTAL	243	767.07		
NI	Model	7	0.11	4.48	0.0001
	Time	3	0.09	8.43	0.0001
	Sex	1	0.00	0.08	0.78
	Interaction	3	0.02	1.49	0.22
	Error	236	0.80		
	TOTAL	243	0.91		
CA	Model	7	443280.83	21.42	0.0001
	Time	3	39578.65	4.46	0.0045
	Sex	1	207997.53	70.34	0.0001
	Interaction	3	12656.08	1.43	0.24
	Error	236	697817.91		
	TOTAL	243	1141098.74		
ML	Model	7	78253.13	28.98	0.0001
	Time	3	1552.64	1.34	0.26
	Sex	1	43241.22	112.08	0.0001
	Interaction	3	852.13	0.74	0.53
	Error	203	78320.04		
	TOTAL	210	156573.17		

NOTE: Sums of squares for time period, sex and interaction are Type III. For variable abbreviations see Table 2.

The interpretation of the overall patterns revealed by the variables C and NI and CA offers insight into the nutritional status of the post-contact Arikara. Specific reference to the archeological and ethnohistorical records helps clarify the historical events that affected food resources and reserves.

Discussion

The period after Euro-American contact was one of dramatic change. Arikara villages were located along the flood plain and lower terraces of the Missouri River valley (Lehmer 1971). Their mixed subsistence strategy was based on the gathering of wild plants, the cultivating of corn, beans, squash, and sunflowers, trading, and hunting, especially of bison. Initially, their warriors controlled activities on the river. Because of their ideal geographic location, the villagers actively participated in intertribal exchange networks, reaping profits as middlemen between groups bringing aboriginal and European trade materials from the eastern woodlands and others in the southern Plains and the southwest (Ewers 1955; Orser 1984; Wood 1980). This trade network had its roots in the Prehistoric period. Following contact, the traditional pattern expanded to include the movement of horses and European goods. The Arikara intensified their horticultural activities during the early contact period to produce surpluses for exchange.

The early contact period before 1750 has been described as a prosperous and stable period for the Arikara as reflected archeologically in the number and sizes of their villages and associated midden deposits. Cache pit size and number increased during this period suggesting increased horticultural productivity (Lehmer and Jones 1968). Some evidence also suggests that shifts in climatic conditions produced perceptible differences in the archeological patterns of Extended Coalescent and Post-Contact Coalescent sites:

> The Extended Coalescent settlement pattern of small villages occupied for only a short time may represent a response to marginal economic conditions, which in turn were the product of a less favorable climate. . . . Bryson has suggested (Baerreis and Bryson, 1965) that the Neo-Boreal conditions were modified somewhat during the first half of the 18th century. The larger and more permanent Post-Contact Coalescent settlements in South Dakota may represent a response to this improvement in climate. (Lehmer 1971:128)

Late 17th and early 18th century populations in general were well off. As reflected by increased cortical thickness and cortical area, the transition from the Late Prehistoric to the Early Protohistoric was marked by a positive change in nutritional status. A culmination of factors insured greater reserves and diversity of resources that buffered the villages against lean times. These factors included agricultural intensification to acquire surpluses for trade and increased availability of meat obtained through hunting and trade.

Horses, first brought to the valley from the southwest, circa 1739, were a valued commodity in the middleman trade, both as an object of trade and as pack animals. Given the lag time between their introduction, initial use as pack animals, and later use for riding, the impact of the horse on the Arikara economy would have been most significant after 1750 (D.J. Blakeslee, pers. comm.; Ewers 1955). Theoretically, horses made it easier to locate and kill wild game and to transport larger quantities of meat to the village than possible during the prehistoric period when dogs were used for this purpose (Holder 1970). The acquisition of the horse, with a corollary increase in high-quality protein in the diet, has been interpreted as a primary reason why Post-Contact Coalescent villagers show increased femoral cortical thickness relative to their late prehistoric Extended Coalescent antecedents (Cashion 1987; Owsley 1985). Yet, as apparent in this more finely graded temporal analysis, the Late Protohistoric period (1740–1795) was not accompanied by dramatic increases in femoral cortical thickness. In fact, a subtle reverse occurs. Medullary cavity diameters increased markedly and mediolateral cortical thicknesses began to drop. In contrast, cortical areas did increase slightly because of the larger values for T. Although this apparent inconsistency is not easily interpreted, it is clear that the major change (improvement) in Post-Contact period nutritional status preceded the arrival of the horse. Any potential nutritional benefits derived from use of the horse were less significant.

Later effects of Euro-American contact were negative and disruptive. Disorganized Coalescent villages suffered higher levels of morbidity, population losses from disease and warfare, and sociocultural deterioration. "Between 1738 and 1845, there was considerable instability of population marked by village abandonment and relocation" (Ramenofsky 1987:104). Catastrophic population losses followed the introduction and diffusion of acute infectious diseases, including cholera, measles, smallpox and whooping cough (Lehmer 1971; Ramenofsky 1987; Trimble 1979,1985). Moreover, intertribal conflict escalated as militant nomadic groups moved into the middle Missouri region after being pushed out of the eastern woodlands. Arikara relations were poorest with the Dakota Sioux, who arrived circa 1730–1740 (Owsley et al. 1977; Smith 1980). The Sioux placed increasing pressure on the Arikara as conflict over bottom lands, which provided protection during the harsh winters, and competition for trade booty and the yield from the Arikara gardens intensified. The period of most intense warfare with the Sioux dates to the historic period after 1790 (D.J. Blakeslee, pers. comm.). Truteau's journal for his journey of 1794–1796, for example, mentions an expected attack on an Arikara village by 500 Sioux warriors well armed with guns (Truteau 1913–1914).

The nutritional effects of these changes are clearly registered in increased medullary cavity diameters and net reductions in femoral cortical bone during the early Historic period. Females seem to have been most affected by the stresses of this turbulent era. Although the direction and pattern of change in bone mass are evident, estimated cortical areas were still higher than during the Late Prehistoric period.

In summary, cortical bone thickness has proven to be a sensitive indicator of changes in Arikara nutritional status caused by environmental change. In contrast, maximum long bone length has not shown a significant response. The difference between these variables merits further consideration. Moreover, future research must examine changes reported for the variable T. Total subperiosteal diameters increased through time, especially in males. Recent research concerning the geometric properties of lower limb bone diaphyses has shown the responsiveness of cross-sectional shape to different mechanical loadings associated with behavior such as more frequent running (Ruff 1987). Perhaps the increase in T was a structural response to changes in activity level. In the future, we plan to consider possible variations in geometric form by examining both the mediolateral and anteroposterior axes and direct measurement of cross-sectional area.

Acknowledgments

William Bass and Douglas Ubelaker kindly granted permission to examine these collections. Archeological recovery was made possible by grants from the National Science Foundation and the National Geographic Society. Donald Blakeslee and Daniel Rogers provided archeological and ethnohistorical information. Steve Symes, Terry Zobeck, and Maria Cashion were responsible for bone radiography and osteometry. Suggestions concerning analytical methodology were provided by J. Dequeker. Dana Bovee helped prepare this manuscript. The illustrations were provided by Ethan Ericksen. Data collection and analysis were supported by NSF BNS-8102650 and BNS-8510588.

Literature cited

Baerreis, D.A., and R.A. Bryson. 1965. Climate Episodes and the Dating of Mississippian Cultures. *The Wisconsin Archeologist*, 46(4).

Cashion, M.A. 1987. A Diachronic Assessment of Stress Indicators in Three Plains American Indian Populations. M.A. thesis, Department of Geography and Anthropology, Louisiana State University, Baton Rouge.

Cook, D.C. 1979. Subsistence Base and Health in Prehistoric Illinois Valley: Evidence from the Human Skeleton. *Medical Anthropology*, 4:109–124.

Dequeker, J. 1976. Quantitative Radiology: Radiogrammetry of Cortical Bone. *British Journal of Radiology*, 49:912–920.

Ewers, J.C. 1955. The Horse in Blackfoot Culture. *Bureau of American Ethnology Bulletin*, 159. Washington, D.C.: Bureau of American Ethnology.

Garn, S.M. 1966. Malnutrition and Skeletal Development in the Pre-School Child. In *Pre-School Child Malnutrition: Primary Deterrent to Human Progress*, 43–62. International Conference on Prevention of Malnutrition in the Pre-School Child, 1964. Washington, D.C.: National Academy of Sciences.

———. 1970. *The Earlier Gain and the Later Loss of Cortical Bone*. Springfield, Ill.: Charles C Thomas.

———. 1972. The Course of Bone Gain and the Phases of Bone Loss. *Orthopedic Clinics of North America*, 3:503–520.

Garn, S.M., M.A. Guzman, and B. Wagner. 1969. Subperiosteal Gain and Endosteal Loss in Protein-Calorie Malnutrition. *American Journal of Physical Anthropology*, 30:153–156.

Garn, S.M., C.G. Rohmenn, M. Behar, F. Viteri, and M.A. Guzman. 1964. Compact Bone Deficiency in Protein-Calorie Malnutrition. *Science*, 145:1444–1445.

Holder, P. 1970. *The Hoe and the Horse on the Plains: A Study of Cultural Development among North American Indians*. Lincoln: University of Nebraska Press.

Hummert, J.R. 1983. Cortical Bone Growth and Dietary Stress among Subadults from Nubia's Batn El Hajar. *American Journal of Physical Anthropology*, 62:167–176.

Hummert, J.R., and D.P. Van Gerven. 1983. Skeletal Growth in a Medieval Population from Sudanese Nubia. *American Journal of Physical Anthropology*, 60:471–478.

Huss-Ashmore, R. 1978. Nutritional Determination in Skeletal Populations. *American Journal of Physical Anthropology*, 48:407.

Keith, M.S. 1981. Cortical Bone Loss in Juveniles of Dickinson Mounds. In D.L. Martin and M.P. Bumsted, eds., *Biocultural Adaptation*, 64–77. Amherst: Department of Anthropology, University of Massachusetts.

Lehmer, D.J. 1971. *Introduction to Middle Missouri Archeology*. Washington, D.C.: National Park Service.

Lehmer, D.J., and D. Jones. 1968. Arikara Archaeology: The Bad River Phase. *River Basin Surveys Publications in Salvage Archaeology*, 7. Washington, D.C.: Smithsonian Institution Press.

Orser, C.E., Jr. 1984. Trade Good Flow in Arikara Villages: Expanding Ray's Middleman Hypothesis. *Plains Anthropologist*, 29:1–12.

Owsley, D.W. 1985. Post Contact Period Nutritional Status and Cortical Bone Thickness of South Dakota Indians. In M. Thompson, M.T. Garcia, and F.J. Kense, eds., *Status, Structure and Stratification: Current Archaeological Reconstructions*, 199–207. Calgary, Canada: Archaeological Association of the University of Calgary.

Owsley, D.W., H.E. Berryman, and W.M. Bass. 1977. Demographic and Osteological Evidence for Warfare at the Larson Site, South Dakota. *Plains Anthropologist Memoir*, 13:119–131.

Ramenofsky, A.F. 1987. *Vectors of Death*. Albuquerque: University of New Mexico Press.

Ruff, C. 1987. Sexual Dimorphism in Human Lower Limb Structure: Relationship to Subsistence Strategy and Sexual Division of Labor. *Journal of Human Evolution*, 16:391–416.

SAS Institute Inc. 1985. *User's Guide: Statistics*. Cary, N.C.: SAS Institute.

Smith, G.H. 1980. *The Explorations of the La Verendryes in the Northern Plains, 1738–43*. Lincoln: University of Nebraska Press.

Trimble, M.K. 1979. *An Ethnohistorical Interpretation of the Spread of Smallpox in the Northern Plains Utilizing the Concepts of Disease Ecology*. Lincoln: National Park Service Midwest Archeology Center.

———. 1985. *Epidemiology on the Northern Plains: A Cultural Perspective*. Columbia: Department of Anthropology, University of Missouri.

Truteau, J.-B. 1913–1914. Journals of Jean-Baptiste Truteau. *The American Historical Review*, 19:299–333.

Van Gerven, D.P., G.J. Armelagos, and M.H. Bartley. 1969. Radiogenographic and Direct Measurement of Femoral Cortical Involution in a Prehistoric Mississippian Population. *American Journal of Physical Anthropology*, 31:23–38.

Wood, W.R. 1980. Plains Trade in Prehistoric and Protohistoric Intertribal Relations. In W.R. Wood and M. Liberty, eds., *Anthropology of the Great Plains*, 98–109. Lincoln: University of Nebraska Press.

SUMMARY OF AUDIENCE DISCUSSION: Porotic hyperostosis is not a useful indicator for stress in this population since the availability of bison prevented protein deficiency. Dental hypoplasia was absent and transverse (Harris') line frequency was not elevated. The pattern of native population decimation secondary to infectious disease shortly after contact with the early colonists is absent in the Plains population reported here. The native population was large, and initially the number of foreigners was low. In fact, the availability of horses enhanced the bison harvest and trade ameliorated the effects of drought. Not until after 1750 did the effects of the flow of eastern colonists moving to and through the Plains become apparent in the natives with a reduction in child growth and increased frequency of premature delivery and the development of an increase in both medullary diameter and the subperiosteal diameter. It is clear that Norden's Index is only of relative value.

Ethnohistorical accounts as a method of assessing health, disease, and population decline among Native Americans

Marc A. Kelley

Paleopathological inquiry, while typically relying on surviving hard and soft tissues, can also benefit from a variety of other sources. These include artistic representations (paintings, engravings, ceramics), coprolites, early medical texts (e.g., Greek, Roman, Chinese) and, as in this study, ethnohistoric accounts. These accounts, when used in conjunction with skeletal samples, not only provide meaningful insight into disease patterns present at time of contact between two cultures, but also record natives' accounts of afflictions and treatments existing prior to contact. I shall focus on the Contact and early Historic periods of New England to demonstrate the utility of this approach.

European contact with New England natives

As any American historian knows, the landing of the Plymouth Pilgrims in 1620 was preceded by more than 120 years of exploration, trade, Indian abduction into slavery, and foiled attempts at settlement on New England soil. Table 1 lists the official voyages from Europe, as well as some of the unofficial expeditions. The actual number of unofficial journeys will never be known, but no doubt well exceeds the documented trips.

From an epidemiological point of view, certain events during the exploratory period deserve closer attention. Hundreds of fishing vessels were visiting areas south of Newfoundland each year during the second half of the 16th century (Fite and Reese 1965; Brasser 1978). In 1602 Gosnold encountered natives wearing pieces of European clothing and understanding a fair number of European words (Purchas 1625). Five years later Popham and Gilbert attempted to establish a colony on the Maine coast (Winship 1968). This was abandoned a year later. In 1616 two different parties, headed by Vines and Hawkins, respectively, wintered at coastal locations in Maine (Gorges 1658; Howe 1942). One member of the Gorges party, Richard Vines, observed the natives to be suffering a plaguelike disease to which the English were seemingly immune (Gorges 1658). A few years later large tracts of New England were nearly void of inhabitants, thus paving the way for the Plymouth Pilgrims.

Chronology of epidemics

A close inspection of ethnohistorical documents suggests that the devastating plague arising in 1616 in New England was preceded by several pestilences in the second half of the 16th century. After an earthquake rocked New England in 1638 the founder of Rhode Island, Roger Williams, questioned the Narragansett elders regarding earlier earthquakes and found that they not only remembered previous earthquakes but associated each one with an epidemic:

> The younger natives are ignorant of the like; but the elders inform me that this is the fifth [earthquake] with these 4 score years in the land: the first about three score and ten years since: the second some 3 score and four years since, the third some 54 years since and the fourth some 46 since: and they always observe either Plague or Pox or some other epidemical disease followed; 3, 4 or 5 years after the earthquake. (LaFantasie 1988:159–160)

While the earthquake/epidemic dualism may be a metaphor of Algonquian speech, the existence of epidemics is not. According to the elders the date for these epidemics was 1572 ± 1 year, 1578 ± 1 year, and 1597 ± 1 year. However, if the Narragansetts had been affected by four epidemics their numbers were surprisingly robust by the early 17th century.

The so-called plague which began in 1616 continued at least until 1619 (Cook 1973b), perhaps until 1622 (Winslow 1841; Morton 1632), and swept away untold thousands of Indians. Graphic accounts have been passed down to us by several explorers and settlers. Thomas Morton (1632:18–19), for example, described scenes near Boston as follows:

> They died on heapes, as they lay in their houses and the living; that were able to shift for themselves would runne away, and let they dy, and let there carkases ly above the

TABLE 1. European voyages to New England prior to A.D. 1620

Date	*Voyage*
1497	Cabot
1500-01	Cortereal
1524	Gomez
1524	Verrazzano
1527	Rut Voyage (English, unofficial)
1530	Crignon
1536	Unofficial English voyage
1550	Approx. 60 ships/yr-Newfoundland
1555	Thevet (Maine, 5 days)
1568	Ingram
1578	Approx. 400 fishing ships/yr
1579	Fernadex
1580	Walker
1583	Gilbert
1600	200 English fishing ships/yr alone
1602	Gosnold
1603	Pring
1604	Champlain
1605	Champlain
1605	Waymouth
1606	Champlain
1606	Hanham and Pring
1607	George Popham and Gilbert
1609	Hudson
1610	Argall
1611	Harlow
1611	Biard (Jesuit)
1614	Block
1614	Smith
1614	Francis Popham
1614	Hunt
1614	Hobson
1615	Dermer
1616	Gorges expedition wintered in Maine
1616	Hawkins party wintered in Maine
1619	Dermer
1620	Plymouth Pilgrims land

SOURCES: Howe 1942, McManis 1972, Purchas 1625, Winship 1968.

> ground without buriall. For in a place where many inhabited, there hast been but one left a live, to tell what became of the rest, the livinge being (as it seemes) not able the bury the dead . . . and the bones and skulls upon the severall places of their habitations, made such a spectacle after my comming into those partes, that as I travailed in that forrest, nere the Massachussets, it seemed to mee a new found Golgatha.

In terms of geographic range, the coastal natives from Cape Cod to the Penobscot River (Maine) were the principal targets of this epidemic. That the Narragansetts were spared its effects is a point agreed on by all early authors (Winthrop 1908; Gookin 1792; Williams 1643). Possible explanations for the spared Narragansetts include the Narragansett Bay, which served as a natural barrier (Cook 1976), and weak social and trade relations between northern and southern New England tribes.

The exact agent or agents responsible for this pestilence remains obscure, but the following observations can be made: disease was not stayed by frost; English apparently were not susceptible; survivors exhibited sores upon their bodies; some Indians' bodies were exceedingly yellow; disease affected Indians only 20–30 miles inland; up to 90% mortality observed in some places; Indians well acquainted with smallpox claim this to be a different disease.

The extremely high mortality rate resulting from the 1616 pestilence is inconsistent with most epidemiological models, and I propose that this pestilence actually represented an unfortunate convergence of two or more diseases over a 3–6-year period. Probable pathogens include a form of the plague, yellow fever, and infectious hepatitis (see Spiess and Spiess 1987 for the argument supporting infectious hepatitis) exacerbated by inadequate health care delivery systems, social disruption, and famine. Indeed, evidence in support of a series of sweeping epidemics may be gleaned from John Smith's "Advertisements for the Unexperienced . . . " in which he states: "Three plagues in three years successively neere 200 miles along the Seacoast that in some places there scarce remained 5 of a hundred" (1631:9).

The next major epidemic in New England was smallpox from 1633 to 1634. Bradford's graphic account deserves some mention:

> They fall into a lamentable condition as they lie on their hard mats, the pox breaking and mattering and running one into another, their skin cleaving by reason thereof to the mats . . . they fell down so generally of this disease as they were in the end not able to help one another, no not to make a fire nor to fetch a little water to drink, nor any to bury the dead. (1970:270–271)

Some 950 Massachusett Indians (Bradford 1970:270–271) and 700 Narragansett Indians (Winthrop 1908:118) died as a result of this epidemic. Indian groups further inland were extensively affected as well.

TABLE 2. Epidemics in New England

Date	*Disease*	*Tribe/Region*
1568	Unknown	Narragansetts
1574	Unknown	Narragansetts
1584	Typhus?	Narragansetts
1592-93	Smallpox	Narragansetts
1616-19	"Plague"	New England, except Narragansetts
1633-34	Measles or smallpox	New England, incl. Narragansetts
1647	Influenza	New England Tribes
1649-50	Smallpox	Northeastern Tribes
1659	Diphtheria	New England and Canada
1664	Smallpox	Massachusett Indian
1669-70	Smallpox	French & British people in Northeast
1677-79	Smallpox	Northeastern Tribes
1713-15	Measles	New England Tribes
1729-33	Smallpox	New England Tribes
1735-36	Diphtheria	New England Tribes
1746	Smallpox	New York & New England Tribes
1755-60	Smallpox	From Canada to Northeast

SOURCES: Bradford 1970, Cushman 1622, DeForest 1852, Dermer 1619, Dobyns 1983, Hubbard 1815, James 1963, Jameson 1910, White 1630, Young 1846.

The 1633–34 epidemic signaled the last of the large-scale, acute disease episodes for New England Indians (see Table 2). While outbreaks of smallpox and other infections occurred intermittently in the years to follow, mortality levels were much lower by necessity of the fact that substantially fewer Indians remained. The question now arises, why did introduced diseases decimate 25%, 50%, or sometimes even 95% of the Indians? Can this be attributed solely to genetic susceptibility?

I should now like to examine more closely the health care systems of 17th century Native Americans and Europeans respectively.

New World and Old World technologies

Native New England pharmacopoeia was quite adequate for dealing with wounds, burns, snake bites, pulmonary ailments, toothaches and body aches (Josselyn 1674), but virtually no remedies existed for Old World pathogens such as plague, smallpox, measles, yellow fever, or influenza. Table 3 lists a small sampling of remedies for common ailments reportedly used in pre-Contact times. This is not to say that the colonists possessed a vast knowledge or set of remedies for such diseases. Even in the 20th century, we possess few drugs for treating viral infections. The 17th century colonial medical knowledge was so scant that John Winthrop was impelled to write to London for instruction on treating common ills. The brief eight-page reply, which served as the early colonists' principal source of medical knowledge, contained potions and elixirs certainly of no greater sophistication than practices by the Indians.

What the Europeans did possess was a familiarity with various Old World viral and bacterial maladies and the

TABLE 3. Examples of plant remedies used by early native Americans

Remedy	*Disease/Ailment*
Alder	Bruises, sprains, head and back aches
Balsam poplar	Burns, cuts, bruises
Bearberry	Scurvy
Blackberry	Dysentery
Burdock	Rheumatism
Dandelion	Rheumatism
Garlic	Arteriosclerosis, high blood pressure
Ginseng	Old age aches/pains
Goldenrod	Kidney disease
Hardhack	Dysentery
Irish moss	Bronchitis
Jack-in-the-pulpit	Sore throat, coughs, tuberculosis
Lobelia	Dysentery, epilepsy
Pipsissewa	Stomach disorders
Poke	Cancer
Sassafrass	Respiratory ailments
Sea tears	Scurvy
Snakeroot	Snakebites, pneumonia
Thistle	Tuberculosis
Tobacco	Earache
White Hellibore	Toothache
Wild carrot	Diabetes
Wild grape	Headache, fever
Wild plum	Asthma

SOURCES: Josselyn 1674, Krochmal and Krochmal 1973, Tantaquidgeon 1925–26, Thomson 1978, Vogel 1970.

efficacy of providing simple attendance to the ill—namely, insuring bedrest, warmth, plenty of fluids and emotional comfort. Edward Winslow's account (1841) of his treatment of the nearly dead Sachem Massasoit in 1623 is enlightening. The Sachem, in an advanced state of illness (suffering possibly from an intestinal virus) had lost his sight, his tongue had swollen, and he suffered from dehydration. Winslow administered some physick (which usually consisted of raisins, currants or other fruit), he then scraped the Sachem's tongue and was able to mix water with the physick, which Massasoit readily consumed. Within a half hour or so, Massasoit was improving, as was his vision.

So here we see that a man on the brink of death was aided by the simple administration of water and fruit, not by any sophisticated medical technology. As Massasoit continued to improve, Winslow graduated him to chicken broth, which made him stronger still. The colonists realized the value of fluids and the danger of fatty foods during recovery from such illnesses, but unfortunately the Indians did not. During his recovery, Massasoit nearly died a second time by gorging himself on fatty duck meat. As we know, Massasoit did survive and lived a long life, but thousands of other natives lost their lives abruptly because of unattended simple needs of the sick.

With this in mind, let us once again examine passages from Governor Bradford's journal concerning Indian suffering and lack of basic health care:

> And then being very sore, *what with cold and other distempers*, they die like rotten sheep. The condition of this people was so lamentable and they fell down so generally of this disease *as they were in the end not able to help one another*, no not to make a fire nor to fetch a little water to drink, nor any to bury the dead. But would strive as along as they could, and when they could procure no other means to make fire, they would burn the wooden trays and dishes they ate their meat in, and their very bows and arrows. And some would crawl out on all fours to get a little water, and *sometimes die by the way and not be able to get in again.* (1970:271, italics mine)

This passage indicates that (1) a synergism existed between smallpox and other distempers, which inevitably led to higher mortality rates than otherwise expected, (2) most members of a tribe were sick simultaneously, and (3) basic health needs went unattended. The English eventually took pity on the suffering Indians and tried to help them, but by then, one suspects, it was too late. One additional factor would have contributed to the natives' downfall: the psychological despair and apathy associated with epidemic sickness. For example, in the 20th century, outbreaks of viruses among remote South American tribes lead to a fatalistic outlook among not only those affected, but the unaffected as well. Had not the medical researchers intervened, mortality levels would certainly have been high.

The differences between European and Indian strategies for health care are thus obvious, but can we attribute such staggering mortality rates among the Indians simply to health care differences? For example, it has been argued that the Indians possessed greater genetic susceptibility to Old World pathogens. While this may in small part be true, I believe it has been greatly exaggerated by medical historians over the last several decades. It is important to remember that, though not so dramatically, smallpox, influenza, yellow fever, and tuberculosis claimed a steady toll of colonists each year. Such diseases were feared by both races. The possibility exists that Indians lacked certain *acquired* immunities to Old World pathogens. Viral infections such as measles and smallpox confer lifelong immunity if the victim survives. European immigrants were much more likely to have been exposed to such viruses in the high-density towns and cities of Europe and thus be immune to subsequent outbreaks occurring in the New World. I remain unconvinced that Europeans possessed an *inherent* genetic resistance to these viruses. Smallpox, for example, seems to have been imported from Asia into Europe only a few centuries prior to exploration of the New World. It would seem unlikely that any appreciable natural selection could have occurred among Europeans during that interval.

In effect, the bulk of evidence would suggest that while a certain amount of loss of life from imported disease was unavoidable, the devastating epidemics suffered by Indians were not *necessarily* inevitable.

Case study: Life for mid–17th century Narragansetts after the viral epidemics

The recent discovery and excavation of a Narragansett cemetery dating between 1650 and the 1670s (see Robinson et al. 1985 for additional background) provide us with an ideal opportunity to examine the biosocial context of the epidemiological transition to an endemic disease setting among these natives. This cemetery was located only three miles from where Richard Smith and Roger Williams had set up a trading post in 1637 or 1638. There is little doubt that these Indians experienced frequent and sustained interaction with the English settlers.

Aspects of Indian acculturation included employment by the colonists to build stone walls (Gookin 1792), the tending of livestock and use of English mills for maize from the 1640s onward (Cronan 1983; Lechford 1867; Williams 1874), and the widespread detrimental consumption of alcohol. This last factor was of sufficient magnitude to prompt the Rhode Island colonists to pass legislation prohibiting the sale of liquor to the natives at least five times during the 1650s (Bartlett 1856). The rich diversity of European goods buried among the 56 members coupled with skeletal evidence of certain chronic disease states provide further evidence of this coexistence.

The skeletal remains indicate an extraordinarily high frequency of skeletal tuberculosis with 30% of the cemetery exhibiting lesions of the spine, ribs and/or hip (Kelley and Robinson in prep.; Robinson et al. 1985; Kelley 1986). Since not all individuals with tuberculosis exhibit skeletal lesions, the number of individuals suffering from this infection must have been considerably higher.

The rise in tuberculosis rates among New England Indians was noticed by some colonists even during the 17th century. In the 1690s Daniel Gookin made the following, rather remarkable statement:

> Sundry of those Indian youths dies, that were bred up to school among the English. The truth is, this disease is frequent among the Indians; and sundry die of it, that live not with the English. A hectick fever, issuing in a consumption, is a common and mortal disease among them. *I know some . . . have attributed it unto the great change upon their bodies, in respect of their diet, lodging, apparel, studies; so much different from what they wer inured to among their own countrymen.* (1792:173, italics mine)

The association between altered lifestyle and elevated tuberculosis rates is widely acknowledged today. Figure 1 depicts the RI-1000 burial ground plan. An interesting pattern emerges when individuals with tuberculous lesions are colored in. Whether this represents disease spread within several family households, or a flare-up of tuberculosis in the community, or enhance chance distribution is uncertain. Williams's description of the Narragansett social practice of visiting the sick is perhaps insightful. He wrote:

> The visit of friends, and neighbours, a poore empty visit and presence, and yet indeed this is very solemne, unlesse it be in infectius diseases, and then all forsake them and flie, that I have often seene a poore house left alone in the wild woods, all being fled, the living not able to bury the dead: so terrible is the apprehensions of an infectious disease, that not only persons, but the houses and whole towne takes flight. (Williams 1643:210)

Such a practice may have been a key element in postepidemic Narragansett decline. Crowding around or simply cohabitating with a sick person who was perceived as not suffering an infectious disease (which would almost certainly include chronic tuberculosis) could result in the pattern observed in Figure 1.

In humans, tuberculosis occurs as an acute or chronic infection caused by either *Mycobacterium tuberculosis* (human form) or *Mycobacterium bovis* (bovine form). The human form is primarily transmitted from person to person by inhalation of the bacilli into the lung. This pulmonary infection, which is often contracted during infancy and childhood, may spread rapidly to other portions of the body or become encapsulated and remain dormant for years or decades. The extreme prevalence of tuberculosis was (and continues to be in certain areas of the world today) due to such factors as malnutrition, overcrowding, war, social upheaval, poverty, alcoholism, and smoking (Lester 1981:972; Burnet and White 1975:217). At least some of these conditions were present in mid–17th century southern New England Indian communities.

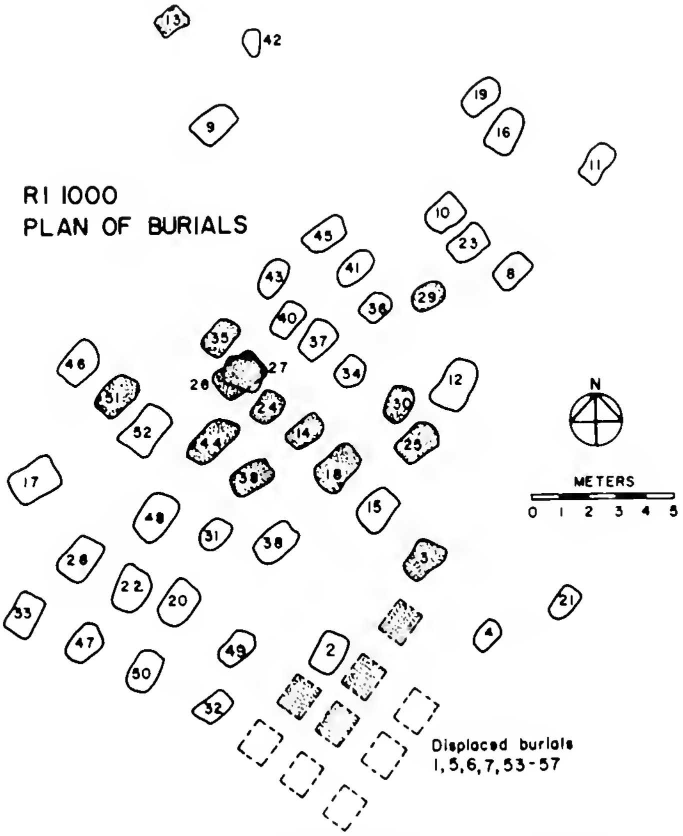


FIGURE 1. RI-1000 burial ground plan. Shaded areas indicate individuals displaying skeletal manifestation of tuberculosis.

Certainly other infectious diseases were taking a toll among the Narragansetts. Pneumonia and dysentery (gastroenteritis), while not leaving any telltale lesions on the bones, are noted in early accounts (Williams 1643) and indeed are still a serious problem in American Indians today. Evidence for a treponemal infection at RI-1000 was noted in one young adult female. The nasal cavity is extensively destroyed and the young woman's hands were placed immediately in front of her face—a pattern not seen in the other burials. Williams (1643) reported that the Narragansett "hothouse" was used in treating the French disease (i.e., syphilis). Finally, one all-pervasive, chronic disease present at RI-1000 was severe dental disease (see Kelley et al. 1987 for detailed discussion).

Each of these chronic conditions was capable of directly or indirectly claiming human life and no doubt contributed to the steady attrition of New England Indians in the mid-17th century. However, this attrition was not nearly so dramatic or devastating as were the earlier viral epidemics. Conceivably such native American groups eventually would have reached an equilibrium and the population would have begun to grow again. This scenario, however, apparently did not fit into the colonists' larger plan. Those who avoided death from microbes would next contend with guns and swords. It is widely acknowledged that warfare tactics differed for Europeans and Indians. The more ritualistic and symbolic style of warfare often practiced by Native Americans prompted Roger Williams to write:

> Their warres are farre lesse bloudy, and devouring then the cruell warres of Europe; and seldome twenty slaine in a pitcht field: partly because when they fight in a wood every tree is a bucklar. When they fight in a plaine, they fight with leaping and dancing, that seldome an arrow hits, and when a man is wounded, unless he that shot follows upon the wounded, they soone retire and save the wounded: and yet having no swords, nor guns, and all that are slaine are commonly slain with great valour and courage: for the conquerour ventures into the thickest, and brings away the head of his enemy. (1643:204)

The Narragansetts objected to the English warfare style, according to John Underhill (1638), because it "slays too many men." Furthermore, the natives had traditionally spared the lives of women and children, a practice not at all observed by the Dutch and English. While Indians slain during battle and massacres obviously contributed to population decline, the more profound blow occurred in the aftermath of hostilities—more specifically, the colonial practice of destroying corn fields and supplies. The result, of course, was famine and more disease—thus providing the rationale for including both disease and warfare in this report. An examination of Cook's tabulation of Indian losses during King Philip's War (1675–1676) illustrates the magnitude of secondary losses (Cook 1973a:21):

1,250	killed in battle
625	died of wounds
3,000	died of exposure and disease
1,000	sold as slaves
2,000	permanent refugees
7,875	total lost
3,875	remaining
11,600	Total

For the Narragansetts, who had been relatively fortunate with regard to introduced disease, King Philip's War effectively reduced them to a remnant population. Table 4 highlights the fate of 17th century Narragansetts by comparing them to McElroy and Townsend's (1985) medical model for 19th century Canadian Inuit natives. This table lists the stages of contact on one axis and the epidemiologic, demographic, nutritional, and health care subsystems on the other axis. Stage I is identical for each group. Stage II remains quite similar, but Stage III departs radically. Instead of population rebound, we see heavy warfare losses and subsequent famine, exposure, and disease. In addition, there was essentially no governmental assistance in the 17th century. In effect, 17th century natives had to face a deadly double-edged sword of epidemics and warfare.

To sum, the use of ethnohistorical records can prove enlightening in our effort to better understand the health patterns of early human populations. It is imperative to remember, however, that clinical descriptions can be vague (as well evidenced by the long-enduring controversy of syphilis origins based on written records). Nonetheless, many diseases are much less controversial (e.g., dysentery, measles, smallpox) and the ethnohistoric record can provide insight in such cases whereas the skeletal remains cannot. Where circumstances permit, this author recommends careful scrutiny and utilization of these alternate resources.

Literature cited

Bartlett, J.R. 1856. *Records of the Colony of Rhode Island and Providence Plantations in New England*, vol. 1 (1636–1663). Providence, R.I.: Greene and Brother.

Bradford, W. 1970. *Of Plymouth Plantation 1620–1647*. New York: Alfred A. Knopf.

Brasser, T.J. 1978. Early Indian-European Contacts. In B.G. Trigger, ed., *Handbook of North American Indians*, vol. 15, Northeast. Washington, D.C.: Smithsonian Institution Press.

Burnet, M., and D.O. White. 1975. *Natural History of Infectious Disease*. 4th edition. New York: Cambridge University Press.

Cook, S.F. 1973a. Interracial Warfare and Population Decline among the New England Indians. *Ethnohistory*, 20:1–24.

______. 1973b. The Significance of Disease in the Extinction of the New England Indians. *Human Biology*, 45:485–508.

______. 1976. The Indian Population of New England in the Seventeenth Century. *Publications in Anthropology*, vol. 12. San Francisco: University of California Press.

Cronan, W. 1983. *Changes in the Land: Indians, Colonists, and the Ecology of New England*. New York: Hill and Wang.

Cushman, R. 1622. Cushman's Discourse. In A. Young, ed., *Chronicles of the Pilgrim Fathers of the Colony of New Plymouth from 1602–1625*. Boston: C.C. Little and J. Brown.

DeForest, J.W. 1852. *History of the Indians of Connecticut from the Earliest Known Period to 1850*. Connecticut Historical Society. Hartford, Conn.: W.J. Hammersley.

Dermer, T. 1619. Letter to His Worshipfull Friend Samuel Purchas, Preacher of the Word, at the Hurcha a Little within Ludgate, London. In *Purchas*, vol. 19 (1905–1907). Glasgow: J. MacLehose and Sons.

Dobyns, H.F. 1983. *Their Number Became Thinned: Native American Population Dynamics in Eastern North America*. Knoxville: University of Tennessee Press.

Fite, E.D., and R. Reese. 1965. *An Economic History of the United States*. Boston: Houghton Mifflin.

Gookin, D. 1792 [1693]. Historical Collection of Indians of New England. *Massachusetts Historical Society Collections*, series 1, vol. 1.

TABLE 4. Comparative medical models

Medical model for 19th century native American Indian

	Stage I (pre-contact)	Stage II (contact)	Stage III (post-contact)
Epidemiology	Few pathogens in ecosystem; low immunities to infections	Epidemics of infectious diseases	Hyperendemic infectious and nutritional diseases
Demography	Births = deaths, population stable	Births < deaths, population decline	Births > deaths, population growth
Nutrition	High protein, low carbohydrate; fluctuating supply	Carbohydrate supplements; famine interacting with epidemics	High carbohydrate, low protein; food supply steady but nutritionally poor
Health care	Shamans and midwives fulfill limited medical and psychotherapeutic needs	Shamans discredited in epidemics; missions provide relief	Government and missions provide modern medical care

Modified medical model for 17th century Narragansetts

	Stage I	*Stage II*	*Stage III*
Epidemiology	Few pathogens in ecosystem; low immunities to infections	Epidemic of 1633 killed 700	Hyperendemic infectious and nutritional diseases
Demography	Births = deaths population stable	Birth < death, population decline occurs gradually	Steady attrition of population followed by rapid decline during King Philip's War, 1675-76
Nutrition	High protein, low carbohydrate; fluctuating supply	Carbohydrate consumption increases with introduction of sugar and flour	Widespread famine in aftermath of war
Health care	Tribal medicine men fulfill limited medical and psychotherapeutic needs	Relatively light loss of life compared to other New England tribes, medicine men not discredited	Little or no government assistance to surviving 17th century Narragansetts

NOTE: 19th century model adapted from McElroy and Townsend 1985.

Gorges, F. 1658. *A Briefe Narration of the Original Undertakings of the Advancement of Plantations in Parts of America*. London: E. Brudenell for N. Brook (Prince Soc. 1890).

Howe, H.F. 1942. Sources of New England Indian History prior to 1620. *Massachusetts Archeological Society Bulletin*, 3:19–24.

Hubbard, W. 1815. *A General History of New England from the Discovery to 1680*. Massachusetts Historical Society. Cambridge, Mass.: Hillard and Metcalf.

James, S.V., ed. 1963. *Three Visitors to Early Plymouth*. Letter from E. Altham to Sir Ed. Altham (Sept. 1623). Massachusetts: Plymouth Plantation.

Jameson, F., ed. 1910. *Johnson's Wonder Working Providence 1628–1651*. New York: Charles Scribner's Sons.

Josselyn, J. 1674. *An Account of Two Voyages to New England*. London: Printed for Giles Widdons.

Kelley, M.A. 1986. Disease, Warfare and Population Decline among Seventeenth Century New England Indians. Paper presented at Peoples in Contact: Indians and Europeans in the Seventeenth Century, Haffenraffer Museum of Anthropology, Bristol, R.I., Sept. 27.

Kelley, M.A., T.G. Barrett, and S. Saunders. 1987. Diet, Dental, Disease and Transition in Northeastern Native Americans. *Man in the Northeast*, 33:113–125.

Kelley, M.A., and P.A. Robinson. In prep. Coping with Change: The Effects of European Colonization on the Narragansett Indians in the Seventeenth Century.

Krochmal, A., and C. Krochmal. 1973. *A Guide to the Medicinal Plants of the United States*. New York: New York Times Book Co.

LaFantasic, G.W. *Correspondence of Roger Williams*, vol. 1. Hanover and London: University Press of New England.

Lechford, T. 1642. *Plain Dealing or News from New England*. J.H. Trumbull, ed. (1867). Boston: J.K. Wiggin and William Parsons Lunt.

Lester, W. 1981. Tuberculosis. In A.I. Braude, ed., *Medical Microbiology and Infectious Disease*, 971–980. Philadelphia: W.B. Saunders.

McElroy, A., and P. Townsend. 1985. *Medical Anthropology in Ecological Perspective*. Boulder, Colo.: Westview Press.

McManis, D.R. 1972. *European Impression of the New England Coast, 1497–1620*. Chicago: University of Chicago Press.

Morton, T. 1632. *The New English Canaan or New Canaan*. Amsterdam: J.F. Star.

Purchas, S. 1625. *Hakluytus Posthumus* or *Purchas His Pilgrimes: Contayning a History of the World in Sea Voyages and Lande Travells by Englishmen and Others*. 20 volumes (1905–1907). Glasgow: J. MacLehose and Sons.

Robinson, P.A., M.A. Kelley, and P.E. Ruberton. 1985. Preliminary Biocultural Interpretations from a Seventeenth-Century Narragansett Indian Cemetery in Rhode Island. In W.W. Fitzhugh, ed., *Cultures in Contact: The Impact of European Contacts on Native American Cultural Institutions A.D. 1000–1800*, 107–130. Washington, D.C.: Anthropological Society of Washington.

Smith, J. 1631. Advertisements for the Unexperienced or the Pathway to Erect a Plantation. *Massachusetts Historical Society Collection*, series 3, 3:1–53.

Spiess, A.E., and B.D. Spiess. 1987. New England Pandemic of 1616–1622: Cause and Archaeological Implication. *Man in the Northeast*, 34:71–83.

Tantaquidgeon, G. 1925–26. Mohegan Medicinal Practices. *43d Annual Report of the Bureau of American Ethnology*. Washington, D.C.

Thomson, W.A.R., ed. 1978. *Medicines from the Earth: A Guide to Healing Plants*. Maidenhead, U.K.: McGraw-Hill.

Underhill, J. 1638. *News from America or, a New and Experimental Discoverie of New England*. London: printed by J.O. for Peter Cole.

Vogel, V.J. 1970. *American Indian Medicine*. Norman: University of Oklahoma Press.

White, J. 1630. *The Planters Plea, or the Grounds of Plantations Examined, and the Usual Objections Answered*. London: W. Jones.

Williams, R. 1643. *A Key into the Language of America: Or An Help to the Language of the Natives in That Part of America, Called New England*. Publications of the Narragansett Club.

———. 1874. In J.R. Bartlett, ed., *Letters (1632–1682) of Roger Williams*. Publications of the Narragansett Club.

Winship, G. 1968. *Sailors Narratives of Voyages along the New England Coast 1524–1624*. New York: B. Franklin.

Winslow, E. 1841. Winslow's Relation "Good Newes from New England: . . . (1624). In A. Young, ed., *Chronicles of the Pilgrim Fathers of the Colony of Plymouth, from 1602 to 1625*, 269–376. Boston: C.C. Little and J. Brown.

Winthrop, J. 1908 [1630–1649]. *History of New England*, vol. 1. New York: Charles Scribner's Sons.

Young, W. 1846. *Chronicles of the First Planters of the Colony of Massachusetts Bay from 1623–1636*. Boston: C.C. Little and J. Brown.

SUMMARY OF AUDIENCE DISCUSSION: Tuberculous involvement in one-third of an archeological population is an unprecedented rate. The author of this study defended his diagnosis on the basis of including only lesions characteristic of tuberculosis seen in the spine, hip and ribs. Costal periostitis in an appropriate pattern reflects the presence of empyema of tuberculous origin since it could not be identified on x-ray in 400 modern patients with pyogenic pneumonia. Such a study, however, needs to be carried out on modern patients with pyogenic empyema. Recognition of characteristic, empyema-induced tuberculous costal periostitis in an archeological population will usually double the tuberculous frequency predicted by the more traditional measures. Known close interaction between these natives and the colonists makes higher-than-usual frequencies of tuberculosis plausible. Since historical records in earlier periods may be influenced by imprecision of diagnostic terminology and even by political influences (deriving from administrative, self-serving reports as may have occurred during the South African colonial era), such evidence should not be used in isolation but may, as in this case, be considered supportive.

Interpretation of infectious skeletal lesions from a historic Afro-American cemetery

Jerome C. Rose and Philip Hartnady

Afro-American history is a complex subject which has engendered considerable interest and numerous debates involving not only historians, but anthropologists and demographers as well. During Reconstruction (1865–1877) the lives of Afro-Americans went through numerous changes where the former plantation slaves undertook the transition from a life dictated by others to one of self responsibility. This transition was aided by temporary provision of housing, food, and medical care by the occupying Union military forces and other agencies both government and private (Stampp 1965). In contrast to the well-documented slavery era, health-related data for both the Reconstruction and post-Reconstruction (1878–1930) periods are scarce and, at times, of questionable quality. No geographically specific understanding of Afro-American demographic processes between 1860 and 1930 can ever be achieved because of the questionable quality of the 1870, 1890, and 1920 censuses (Farley 1970:3). This scarcity of both demographic and disease data can be attributed primarily to the lack of record keeping and inadequate census procedures resulting from the turmoil of "carpetbag rule" in the former Confederacy and a continuation of this situation for Afro-Americans with the establishment of legalized segregation during the post-Reconstruction period. Until recently, the skeletal remains of people from this time period have been largely unavailable. Yet, skeletal analysis can provide information critical to understanding the conditions of life and health during this historic period.

The analysis of skeletal remains collected during the relocation of Cedar Grove (3LA97), a rural Afro-American cemetery in southwest Arkansas, is ideally suited for addressing issues of postemancipation health. Demographic and paleopathological data are used to test the imperfect historic reconstructions of postemancipation life and provide a more detailed picture, at least for this sample, of diet, health, and the general quality of life. The analysis of the Cedar Grove skeletal sample also provides an opportunity to test the validity of paleodemographic and paleopathological interpretations by comparing them with those derived from census data and historic documents. It is not uncommon for the utility of paleodemography to be called into question. For example, Bocquet-Appel and Masset (1982) contend that paleodemography cannot provide a true or realistic reconstruction of a population using skeletal data. Although this criticism has been clearly answered by a number of authors (Buikstra and Konigsberg 1985; Van Gerven and Armelagos 1983), the Cedar Grove data are used to test the concordance of paleodemographic interpretations with those derived from census data. Similarly, doubts have often been raised concerning paleopathology, in particular the inaccuracies of lesion diagnosis (see Ubelaker 1982:344–345) and the utility of using skeletal lesions to reconstruct disease patterns (see Buikstra and Cook 1980:439–440). Again, concordance between the paleopathological interpretations and the historic literature is tested with the Cedar Grove material.

Materials and methods

During the construction of a revetment along the Red River, the U.S. Army Corps of Engineers encountered what was thought to be a small historic cemetery and a prehistoric American Indian farmstead. After determination of eligibility for nomination to the National Register of Historic Places, the marked historic graves were relocated and the prehistoric site excavated by the Arkansas Archeological Survey. During the excavation, an additional 104 unmarked grave outlines were located. Historic investigation established that this cemetery had been used by the Afro-American community associated with the Cedar Grove Baptist Church, which lost use of the cemetery when it was covered by almost two meters of silt during the 1927 flood. After extensive negotiations and legal determinations, those 79 graves scheduled for destruction by revetment construction were excavated, analyzed, and relocated to a new cemetery. The skeletal remains and all associated grave contents were excavated using standard archeological techniques and analyzed in a field laboratory prior to reburial in a new cemetery plot. All archeological and osteological analyses were conducted in the field within portable buildings brought to the excavation site and used laboratory and photographic equipment brought from the University of Arkansas campus. All skeletal materials were washed, inventoried, and photographed. Age for subadults was determined using dental development (Schour and Massler 1941) and epiphyseal union (Krogman and Iscan 1986:50–97). Age determination for adults used pelvic criteria. Each pubis was scored using the Todd system with the Brooks modification (as cited in Krogman and Iscan 1986:148–154), and the pubic cast system for males (McKern and Stewart 1957) and females (Gilbert and McKern 1973). The auricular surface age system employing both textual and photographic descriptions of each stage was also applied (Lovejoy et al. 1985). Macroscopically observed skeletal lesions were recorded using the system adapted by Powell (1985:433–434). This system records each pathological lesion by a numerical code, textual description, and color-coded drawings on a skeleton outline provided on the recording forms. The four-digit numerical code provides the following information: type of lesion (i.e., resorptive, proliferative, trauma, and neoplasm), location on bone, extent of lesion, and status of lesion (i.e., active or remodeled). The textual descriptions and drawings provided clarification of the code for each lesion and a photograph was taken of each. The numerical code was entered, along with age and sex, into a computerized data base for this analysis.

TABLE 1. Demography of the Cedar Grove historic cemetery

Age	*Unknown*	*Males*	*Females*	*Total*
Birth	16	–	–	16
0.3 – 0.9	6	–	–	6
1 – 1.9	11	–	–	11
2 – 4.9	1	–	–	1
5 – 9.9	5	–	–	5
10 – 14.9	5	–	–	5
15 – 19.9	–	1	–	1
20 – 29.9	–	2	4	6
30 – 39.9	–	3	11	14
40 – 49.9	–	7	3	10
50 +	–	2	3	5
TOTALS	44	15	21	80

TABLE 2. Percent active osteolytic/proliferative lesions by age in years

	Birth	*0.1–0.9*	*1–5*	*6–10*	*11–29*	*30–39*	*40–50*	*50+*
Basiocranium	38	50	17	0	0	0	0	0
Calvarium	44	17	17	0	0	0	0	0
Endocranium	38	17	33	0	0	0	0	0
Frontal	19	17	17	0	0	0	0	0
Orbit	19	33	8	0	0	0	0	0
Maxilla	6	0	0	0	0	0	0	0
Mandible	25	0	8	0	0	0	0	0
Cervical	6	0	0	0	0	0	0	0
Thoracic	6	0	8	0	0	7	0	0
Lumbar	6	0	8	0	0	0	0	0
Ribs	56	33	33	0	11	7	10	0
Clavicle	12	17	8	0	0	0	0	0
Scapula	69	17	8	0	11	7	0	0
Humerus	75	67	17	0	11	7	0	0
Radius	81	50	25	0	11	21	0	0
Ulna	75	33	25	13	11	7	0	0
Innominate	69	33	8	0	11	0	0	0
Femur	81	33	33	0	22	0	0	0
Tibia	81	33	50	13	11	0	0	20
Fibula	81	17	42	0	11	0	0	0
SAMPLE SIZE	16	6	12	8	9	14	10	5

Results

Both historical and archeological evidence established that all excavated individuals were interred between 1890 and 1927. The 79 excavated graves produced a total number of 80 individuals, as one grave contained two individuals (seven-month in utero twins). The age and sex data (see Table 1) were analyzed using an abridged life table following the procedures of Swedlund and Armelagos (1976:63–64) and comparisons are made to model life tables (Weiss 1973:115–186). The most diagnostic feature of the Cedar Grove demographic profile is the large proportion (55.0%) of individuals younger than 15 years, which is identical to that (55.3%) produced by model life table 15.0–45.0 (Weiss 1973:118). This model table is considered to be the best fit for the Cedar Grove demographic data. The computed life expectancy at birth for Cedar Grove is 14 years which is only slightly below the 15 years predicted by the 15.0–45.0 model life table. The only major difference between Cedar Grove and this model life table is the much higher proportion of individuals less than one year of age at Cedar Grove (27.5%) than predicted (5.4%) by the model table. However, when the large number of neonatal deaths (20.0%) are removed (i.e., skeletons aged at birth or younger than birth), the resulting figure (7.5%) is comparable. The Cedar Grove skeletal series is most remarkable for the extremely high rate of skeletal lesions; almost 90% of the entire sample exhibit at least one lesion, and the average is 12 per individual (total lesions = 959). Five individuals are aged to younger than birth and probably were premature stillborns. Each of these exhibit active systemic periostitis indicating uterine infections which may be implicated in the premature births. Of the 11 neonates, 9 (81.8%) exhibit systemic active periostitis with additional lesions as follows: 4 (36.4%) with active cribra orbitalia, 3 (27.3%) with active endocranial periostitis, and 5 (45.4%) with periostitis of the ribs. One neonate has no lesions, while a second shows healed endocranial periostitis. The 17 children between 3 and 20 months of age display a lesion pattern comprising 23.5% craniotabes, 58.5% active cribra orbitalia, 41.2% active systemic periostitis, and 35.3% active endocranial periostitis. Although the most extensive lesion complex is systemic bacterial infection, two of the four cases of rib periostitis are not associated with other bone lesions. The pattern of childhood deaths is also of interest. The childhood deaths begin at 3 months of age and slowly increase to a peak at 18 months where 41.2% of the subadult deaths occur. The adult skeletal lesion pattern is far more diverse and exhibits a number of different lesion complexes. The lesion frequencies are high with 100% for the 15 males and 86% for the 21 females. The frequency of healed cribra orbitalia (males 13.3%, females 0.0%) and remodeled porotic hyperostosis (males 33%, females 23.8%) indicates prior experience with anemia. The periostitis rates of the lower limbs are high for both males (60.0%) and females (52.4%). Several of the lesions exhibit the morphological feature of subperiosteal hemorrhage. The frequencies of male spinal osteophytosis (46.7%), Schmorl's nodes (33.3%), osteoarthritis of the major joints (33.3%), hands (13.3%), and feet (26.7%) are all relatively high. The females exhibit a similar pattern of spinal osteophytosis (33.3%), Schmorl's nodes (19.0%), and osteoarthritis of the major joints (28.6%), hands (19.0%) and feet (19.0%).

TABLE 3. Percent healed osteolytic/prolifarative lesions by age in years

	Birth	*0.1–0.9*	*1–5*	*6–10*	*11–29*	*30–39*	*40–50*	*50+*
Basiocranium	0	0	0	0	0	0	0	0
Calvarium	0	0	0	0	0	0	20	0
Endocranium	6	0	0	0	0	0	0	0
Frontal	6	0	0	0	0	0	10	0
Orbit	0	0	8	0	0	0	10	0
Maxilla	0	0	0	0	0	0	0	20
Mandible	0	0	0	0	11	14	0	0
Cervical	0	0	0	0	0	0	0	0
Thoracic	0	0	0	13	0	0	0	0
Lumbar	0	0	0	13	0	0	0	0
Ribs	0	0	0	0	0	7	0	0
Clavicle	0	0	0	0	11	0	0	0
Scapula	0	0	0	0	0	0	0	0
Humerus	0	0	0	0	0	0	0	0
Radius	6	0	8	0	11	7	0	0
Ulna	6	0	0	13	0	7	0	0
Innominate	6	0	0	0	0	0	0	0
Femur	6	0	0	0	11	7	40	20
Tibia	6	0	17	13	56	50	90	80
Fibula	6	0	8	0	33	50	80	100
SAMPLE SIZE	16	6	12	8	9	14	10	5

Trauma is frequent with the males exhibiting 20.0% healed cranial fractures, 13.3% healed rib fractures, and one fatal bullet wound. Characteristically, the incidence of these lesions is much lower for the females with only two (9.5%) cases of healed rib fractures and one fatal bullet wound.

Having described the overall lesion pattern for the Cedar Grove skeletal series, we presented in greater detail the proliferative lesions (i.e., periostitis, osteomyelitis, and osteitis) characteristic of bacterial infection. Table 2 provides the percentage of active proliferative lesions by age at death; Table 3 provides the percentages of healed or healing proliferative lesions. It should be noted that we combined age groups 11–19 and 20–29 years to facilitate comparison by producing a sample size (9) approximately equal to the other age groupings. Table 2 shows that both the prematures and neonates (grouped together as aged at birth) have a high frequency of systemic proliferative lesions. Virtually every component of the skeleton is impacted: cranium, ribs, bones of the arm, pelvis, and bones of the leg. Of particular interest is the high rate of endocranial involvement (38%). The fact that these lesions are so extensive, even on those individuals aged younger than birth, strongly suggests that the infectious agent was contracted in utero. The patterning of lesions (i.e., distribution among the bones of the skeleton) remains relatively stable between birth and 10 months, but then, with the exception of the cranium and clavicle, the frequency of infected bones declines. The fact that no healed cranial lesions are found on individuals of any age (Table 3) indicates that few or none of those neonates and children with cranial infections survived. For example, examination of infections of the calvarium (Figure 1) shows a decline with age and only two healed lesions among those aged between 40 and 49 years. This pattern is roughly similar for all bones of the skull.

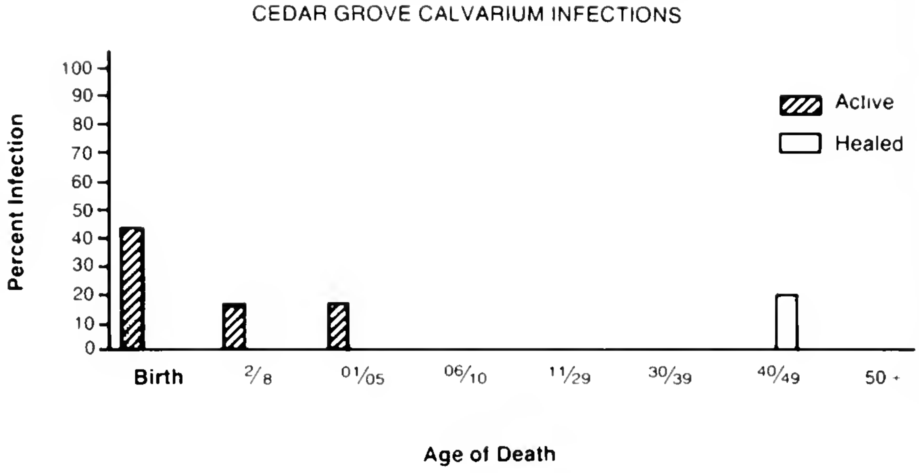


FIGURE 1.

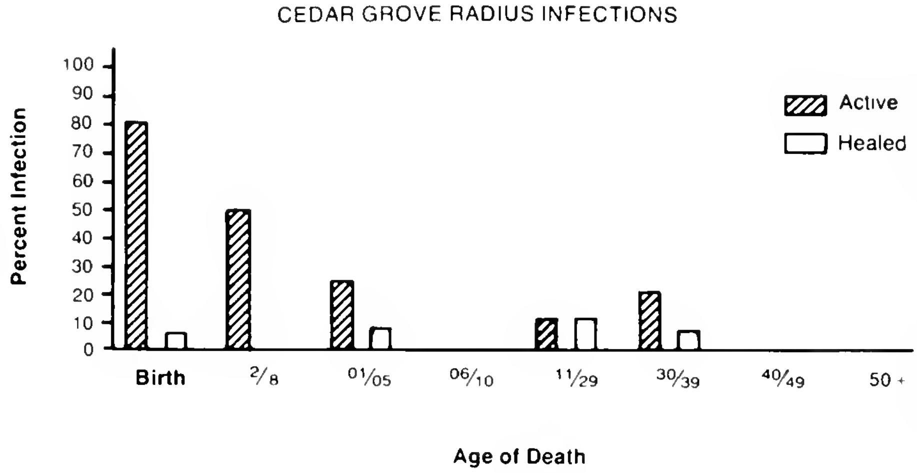


FIGURE 2.

The postcranial skeleton shows a slightly different pattern. Active lesions of the arm bones drop steadily from birth through 5 years and reoccur at sporadic low levels between 11 and 39 years. This pattern can best be illustrated by lesions of the radius (Figure 2). The few healed infections of the arm can best be attributed to late-occurring infections and not to survivorship of the infected infants. The arm lesions suggest a bimodal pattern of origin with the first peak of active infections occurring before and at birth and the second occurring between 11 and 39 years.

In contrast, the infectious lesions of the leg, especially the lower leg, show a trimodal distribution. Like the arm bones, the active lesions of the leg are highest at birth, drop significantly between birth and 10 months, exhibit a second peak between one and five years (actually a modal age of 18 months), followed by a third consisting of an age-cumulative increase of healed lesions. This pattern is best illustrated by the fibula (Figure 3).

The ribs have been singled out for special attention because in one respect they are similar to the arm bones in distribution, but different in that they are often not associated with lesions on other bones (Figure 4). Like the arm bones, active periostitis of the ribs drops between birth and five years and occurs at relatively low levels in adulthood, with only one healed lesion in the entire sample. The majority of the early lesions can be attributed to systemic infection, but two children (one at 13 months and the other at 20 months) display no other infectious lesion. The three adults (one male and two females) with active rib infections also show no other active lesion. This distribution suggests that a separate pathological process is responsible. The fact that there is only one healed rib lesion present in the sample suggests that rib lesions might be etiologically related to causes of death.

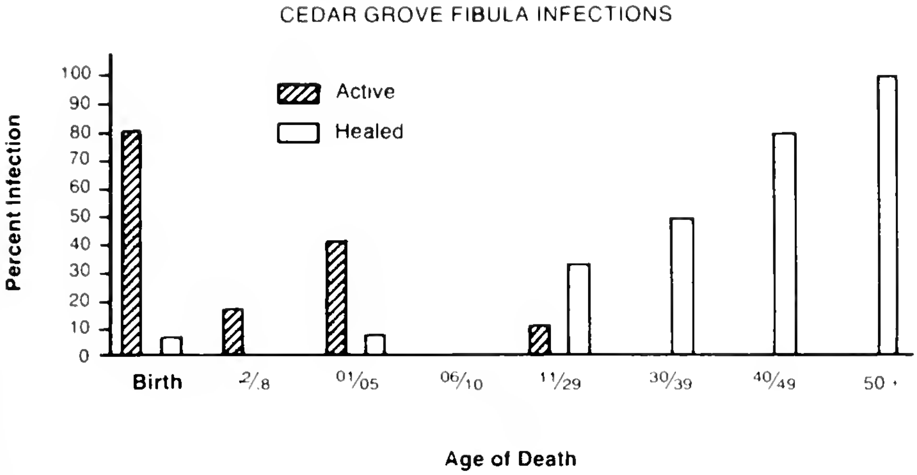


FIGURE 3.

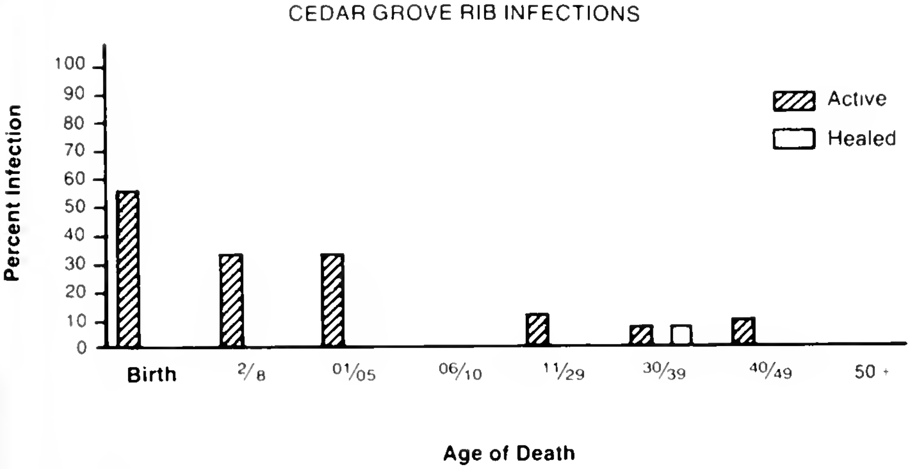


FIGURE 4.

Discussion

Two interrelated goals are pertinent to this analysis of the Cedar Grove demographic and paleopathological data. The first is establishing the contribution which these data can make to improving our understanding of Afro-American life after Reconstruction, at least for a part of southwest Arkansas. The second goal is demonstrating with a specific example the utility of paleodemographic and paleopathological analyses by comparing their results with those obtained from historical analysis of documentary data.

Since there is a dearth of specific data concerning postemancipation diet, dietary information from the better documented slavery period will first be summarized. Based on available documentary data the following dietary deficiencies have been postulated for southern slaves: the 70–77% lactose intolerance rates of Afro-Americans contributed to low milk consumption and calcium deficiency (Kiple and Kiple 1977:290; Kiple and King 1981:72); the low niacin content of the corn and pork diet resulted in pellagra (Gibbs et al. 1980:205; Kiple and King 1981:127); the low bioavailability of iron in a corn-based diet combined with frequently inherited Afro-American blood abnormalities (e.g., sickle cell) to produce frequent anemia (Kiple and Kiple 1977: 285; Kiple and King 1981:97); low milk consumption combined with dark skin produced vitamin D deficient rickets (Kiple and King 1981:195; Kiple and King 1981:93); and the low amino acid proportions of tryptophan, lysine, and methionine in both corn and pork proteins contributed to protein malnutrition (Kiple and Kiple 1977: 287). Other dietary inadequacies such as thiamin deficiency causing beriberi (Kiple and King 1981:119) and magnesium deficiency which lowers resistance to infection (Kiple and King 1981:98) have been mentioned.

For the postemancipation period there are only generalizations from various locations throughout the American South to work from. Donald (1952: 47) indicates that, on the whole, the diet of South Carolina Afro-Americans was coarse and consisted of hominy, cornbread, fat bacon, some pork, coffee, rice, molasses, and occasional vegetables. Similarly, Kiple and King (1981:189) state that the diet went from bad under slavery to worse under freedom. Within the Cedar Grove locale it is not certain exactly what dietary changes occurred after emancipation. The 1865 contract negotiated by the Freedmen's Bureau between the former slaves and Sentell family called for the provision of a wage, rations, housing, and one acre of land per household in exchange for labor on the plantation (Watkins 1985:12). At that point in time, it seems that the Afro-American diet was still being dictated by the landowner. For example, under the terms of the above-mentioned contract, no livestock except poultry could be owned (Watkins 1985:12). This surely would have limited the supply of meat in the diet and prevented the former slaves from using animal power to raise food on their one acre of allotted land.

This contract labor system soon came to an end and the large plantations were broken up into individual allotments, farmed by the Afro-Americans under a sharecropping arrangement (i.e., a portion of all crops was turned over to the landowner). The system of advancing loans for seed and supplies, which was commonly introduced throughout the South, usually served to keep the sharecroppers poor and in debt to the landowners and merchants (Christensen 1958). Conditions deteriorated further in 1888 when a rapid decline in cotton prices left all southern Arkansas farmers poor and in debt to the stores (Graves 1967:30). A further disaster occurred in 1905 when the boll weevil arrived in southeast Arkansas and virtually wiped out the cotton crops, the major source of cash income (Sylva 1981:52). These deteriorating agrarian conditions stimulated the Euro-American backlash which wiped out virtually all the social gains of the Reconstruction period. The process of segregation and political disenfranchisement began with the passage of new discriminatory voting laws and the first Arkansas segregation law, the separate coach act, in 1891 (Graves 1967:61–94). These political and social changes, in combination with the farm price crisis, should have seriously and adversely impacted Afro-American diet and health in the Cedar Grove area.

Sylva identifies a decline in the Afro-American population in southwest Arkansas during this period and attributes it to outmigration (1981:16). In contrast, Farley observes a national trend of significant slowing of the Afro-American population growth between 1880 and 1940 (1970:3). He notes that Afro-American women who began childbearing before 1850 and survived until menopause produced an average of seven children and less than 10% of these women produced no children. Women born between 1900 and 1920 not only had the lowest fertility before or since, but 30% never had a child and those that did had fewer (Farley 1970:3). These data suggest that a biological crisis occurred for the entire Afro-American population at the turn of the century. This trend was so noticeable that Holmes (1937) prepared a monograph predicting the disappearance of Afro-Americans.

The population decline is to be attributed not only to decreased fertility, but also to greatly increased mortality. Urban Afro-American life expectancy at birth in 1900 was 33 years for males and 35 years for females (Farley 1970:61). In 1900 the Afro-American mortality rate was 3.02%, nearly twice that of Euro-Americans with 1.73% (Holmes 1937:40; Kiple and King 1981:188). This high mortality rate is observed at all demographic levels. The non-Euro-American infant death rate was 275 per 1000 live births (Farley 1970:212). The non-Euro-American maternal mortality rate in 1920 was still 13 per 1000 live births, while the neonatal death ratio was 72 per 1000 live births (Farley 1970:209). Physicians commonly noted that the Afro-American stillbirth rate was two to three times higher than Euro-Americans (Kiple and King 1981:188). Farley, in an attempt to explain both the high stillbirth rate and decreased fertility, suggests venereal disease, citing as evidence a 20% infection rate among Afro-American females and a 1900 infant death rate of 2.7 per 1000 live births due to congenital syphilis (1970:12).

Donald's examination of the recorded disease patterns shows that Afro-Americans suffered a higher mortality rate than Euro-Americans from all diseases except cancer (1952:162). Using the 1900 census, Farley (1970:70) lists the most frequent causes of Afro-American deaths as tuberculosis, pneumonia, nervous disorders, diarrhea, typhoid fever, and malaria. Both tuberculosis and pneumonia were major killers of Afro-Americans (Kiple and King 1981: 188) with Afro-American tuberculosis rates being reported as three times higher than Euro-Americans (Holmes 1937:76).

The data and interpretations presented above are derived primarily from the national census and pertain to the areas of registration. They may or may not describe the situation in southwest Arkansas nor, in particular, the community of Cedar Grove. Keeping in mind this limitation of the data, they are compared and contrasted with those collected from the Cedar Grove skeletal sample.

Using the cross-sectional skeletal demographics as a true birth cohort allows the calculation of mortality rates. Using the ratio of skeletons aged to less than one year to total skeletons produces an infant mortality rate of 27.5%, which is identical to the national non-Euro-American infant mortality of 27.5% cited by Farley (1970:212) for this same time period. Using the ratio of skeletons aged younger than birth to total skeletons produces an estimated stillbirth rate of 6.2%, which is close to Farley's (1970:209) 1920 national non-Euro-American rate of 7.2%. Although life expectancy at birth obtained from the skeletal life table (14 years) is far below the 33 years for males and 35 years for females reported by Farley (1970:61), the average adult skeletal ages at death (males 41.2, females 37.7) are reasonably close to these national statistics. The entire Cedar Grove paleodemographic profile calculated from the skeletal data is in excellent concordance with the national statistics and the historic reconstructions. These results suggest not only that paleodemography can be used reliably, but also that the Cedar Grove community followed the national trends of increased mortality and decreased fertility. In other words, the Cedar Grove community was highly stressed during the post-Reconstruction period.

There is abundant evidence of dietary deficiencies in the Cedar Grove skeletal sample. The high rate of active cribra orbitalia among children (58%) and the rates of healed cribra orbitalia and porotic hyperostosis among adults (males 33%, females 24%) indicate extensive anemia. Most of the anemia can be attributed to iron deficiency resulting from a reliance upon corn and a lack of red meat in the diet, as suggested by the historical literature (Kiple and Kiple 1977:285). Some of these lesions can be attributed to sickle cell, but this genetic trait should account for only a small percentage of the observed cases (Ortner and Putschar 1981:254–258). The 24% craniotabes for children dying between 3 and 20 months can be attributed to vitamin D deficient rickets, associated with the historically postulated low milk consumption caused by a high rate of lactose intolerance common among Afro-Americans, and the documented scarcity of cattle among the sharecroppers (Kiple and Kiple 1977:290; Kiple and King 1981:72). At least some of the extensive childhood periosteal deposits and the ossified hematomas among the adults may be attributable to vitamin C deficiency (Ortner and Putschar 1981:271–272) caused by a lack of fruits and vegetables in the diet. These high rates of lesions specific for dietary deficiencies indicate a very inadequate diet in the Cedar Grove community between 1890 and 1927.

The high frequency of active systemic periostitis found among both prematures and neonates suggests the existence of at least one dominant disease. Farley's (1970:12) suggestion of congenital syphilis can be tested with the skeletal data. Steinbock (1976:98–99) describes early congenital syphilis as occurring between birth and three to four years, being associated with universal bone changes which include periostitis and diaphyseal osteomyelitis, and having a mortality of at least 50%. Ortner and Putschar (1981:198) state that congenital syphilis leads to early fetal death, delivery of a premature or mature diseased stillborn fetus, or delivery of a living infected newborn. They further state that syphilitic periostitis can have begun in utero and be present at birth (1981:198). Steinbock (1976:100–101) states that cranial osteitis can impact both cranial tables. The association of endocranial new bone formation and long bone periostitis found in a prehistoric Native American skeletal sample has been attributed to one of the treponematoses by Cook and Buikstra (1979:658).

All five Cedar Grove skeletons with dental ages younger than birth (seven to eight fetal months) exhibit systemic periostitis involving virtually every bone. All but 2 of the 11 individuals dying at birth exhibit systemic periostitis, while one of the exceptions has only endocranial periostitis. Of the 18 deaths between three months and 3.5 years, 48% exhibit systemic periostitis. Taken together, the high frequency of prematures with systemic periostitis, the high neonatal mortality associated with active systemic periostitis, and the absence of systemic periostitis after 3.5 years all suggest congenital syphilis as the dominant disease entity. This diagnosis is further strengthened by the presence of Hutchinson's maxillary incisors on one 10-year old. These four maxillary incisors match the classic description of Hutchinson's defect including notching, barrel shape, and convergent lateral margins (Steinbock 1976:107). The diagnosis as Hutchinson's incisors was also confirmed by histological examination (Marks 1984).

If the diagnosis of widespread congenital syphilis is correct, then the characteristic lesions of venereal syphilis should also be evident in the adult sample. The ranking by frequency of periostitis among the older individuals is tibia, fibula, radius, ulna, and femur. This ranking is fairly consistent with that reported for venereal syphilis (Steinbock 1976:112). Of particular importance is the high frequency of periostitis of the arm bones at Cedar Grove, which is a fairly uncommon location for other infectious diseases and consistent with the presence of venereal syphilis (Steinbock 1976:112). Absent from Cedar Grove are the characteristic cranial lesions of syphilis, the saber shins, and the extensive osteomyelitis of the tibiae (Ortner and Putschar 1981:201–218; Steinbock 1976:108–136). Despite the absence of undisputed acquired syphilis among the adults, the diagnosis of congenital syphilis as the major cause of stillbirths, high neonatal mortality, and widespread systemic infection appears very reasonable.

A second pathological phenomenon is indicated by the fact that, while the frequency of lesions in all other bones continues to decline, the tibia, fibula, and endocranium show a relatively large increase between one and five years (Tables 1 and 2). In fact, 58% of the deaths between one and five years occur at 18 months of age. The 18-month modal age at death, a common age of weaning, combined with an increase in infections of the tibia and fibula is highly suggestive of weanling diarrhea (Scrimshaw et al. 1968:216–260). This syndrome is characterized by low-protein weaning diets which contribute to lowered resistance to infection and initiate a cycle of diarrhea and infectious disease. The presence of protein malnutrition resulting from amino acid deficiencies associated with corn- and pork-dominated diets has been noted in the historic literature (Kiple and Kiple 1977:287).

The presence of rib periostitis that is not associated with systemic infection has been previously identified in two children (13 and 20 months), one adult male, and two adult females. The location of these lesions on the medial surface of the rib body and their gross appearance conform with the lesions identified by Kelley and Micozzi (1984) as being associated with pulmonary tuberculosis among cadaver specimens from the same time period as Cedar Grove. As tuberculosis is identified as the leading cause of death among Afro-Americans at the turn of the century (Farley 1970:70; Holmes 1937:76), assigning 6% of the Cedar Grove deaths to this disease is appropriate. This is not to say that the rib lesions associated with other infected bones are not also attributable to tuberculosis, but the 6% is a conservative estimate of the frequency of tuberculosis.

The final lesion complex to be discussed is the age-cumulative increase in healed infections of the lower leg. The location of the tibia just below the skin makes it vulnerable to frequent introductions of bacteria from relatively inconsequential accidental wounds (Ortner and Putschar 1981:132). Thus, the frequency of tibia infections should increase under conditions of reduced resistance to disease where the reduction of the body's defense mechanisms permits the infectious agents to become established. Periostitis of the tibia is frequently found in archeologically derived skeletal collections (Ortner and Putschar 1981:131; Steinbock 1976:82). In contrast, we have found periostitis of the fibula to be proportionally less frequent in prehistoric American skeletal samples (i.e., 50% or less than the tibia rate) and interpret the infections of the fibula as being associated with either major leg trauma or spread of infection from the tibia. Thus, increases in fibula infections can be used to indicate a reduction in disease resistance. The age-specific frequencies of adult healed tibia infections range between 56 and 90%, while the fibula rates range between 33 and 100% (Table 3). The virtual absence of active lower leg infections indicates that these lesions were not associated with the cause of death, but represent previous episodes from which the individuals recovered. Both the high frequency of tibia and fibula periostitis and the concordance of rates between the two bones suggest diminished disease resistance among the people of Cedar Grove. This interpretation is consistent with the historical literature which suggests poor diet and high stress for Afro-Americans in the post-Reconstruction South.

Conclusions

In this analysis of the Cedar Grove infection data we have attempted to achieve two goals: first, to improve our understanding of Afro-American disease patterns in the post-Reconstruction period of southwest Arkansas, and second, to demonstrate that paleodemographic and paleopathological analysis can provide interpretations compatible with historic interpretations. The Cedar Grove skeletal demography is in excellent concordance with the census data. There was high infant mortality (27.5%), high frequency of stillbirths (6.2%), and high adult mortality with average ages of death at 41 years for males and 38 years for females. Skeletal evidence for dietary deficiencies including iron, vitamins D and C, and protein are in agreement with the historical reconstructions.

The distribution of proliferative lesions by affected bones of the skeleton and age indicates the presence of four major disease clusters. The systemic infections among the neonates indicate the presence of widespread congenital syphilis. Although the classic stigmata of venereal syphilis are not found among the adults, the presence of lesions on the bones of the arm is suggestive. The peak mortality at 18 months and an associated increase in proliferative lesions indicate the presence of the weanling diarrhea syndrome and, by implication, protein deficiency. A dramatic age-cumulative increase in healed lesions of the tibia and fibula indicates continued adult malnutrition and high overall stress loads. The frequent rib infections are consistent with a high frequency of pulmonary tuberculosis. All of these conclusions are compatible with the interpretations produced by historical analysis of the documentary data.

Acknowledgments

Financial support for the Cedar Grove Cemetery excavation and analysis was provided by the U.S. Army Corps of Engineers, New Orleans District, to the Arkansas Archeological Survey and the senior author.

Literature cited

Bocquet-Appel, J-P., and C. Masset. 1982. Farewell to Paleodemography. *Journal of Human Evolution*, 11:321–333.

Buikstra, J.E., and D.C. Cook. 1980. Paleopathology: An American Account. *Annual Reviews of Anthropology*, 9:433–470.

Buikstra, J.E., and L.W. Konigsberg. 1985. Paleodemography: Critiques and Controversies. *American Anthropologist*, 87: 316–333.

Christenson, D.E. 1958. The Negro's Changing Place in Southern Agriculture. In *The Research Council of the Florida State University, The Negro in American Society*, 41–52. Tallahassee: Florida State University.

Cook, D.C., and J.E. Buikstra. 1979. Health and Differential Survival in Prehistoric Populations: Prenatal Dental Defects. *American Journal of Physical Anthropology*, 51:649–664.

Donald, H.H. 1952. *The Negro Freedman: Conditions of the American Negro in the Early Years after Emancipation.* New York: Henry Schuman.

Farley, R. 1970. *Growth of the Black Population: A Study of Demographic Trends.* Chicago: Markham Publishing.

Gibbs, T., K. Cargill, L.S. Lieberman, and E. Reitz. 1980. Nutrition in a Slave Population: An Anthropological Examination. *Medical Anthropology*, 4:173–262.

Gilbert, B.M., and T.W. McKern. 1973. A Method for Aging the Female Os Pubis. *American Journal of Physical Anthropology*, 38:31–38.

Graves, J.W. 1967. The Arkansas Negro and Segregation 1890–1903. M.A. thesis, University of Arkansas, Fayetteville.

Holmes, S.J. 1937. *The Negro's Struggle for Survival: A Study in Human Ecology.* Berkeley: University of California Press.

Kelley, M.A., and M.S. Micozzi. 1984. Rib Lesions in Chronic Pulmonary Tuberculosis. *American Journal of Physical Anthropology,* 65:381–386.

Kiple, K.F., and V.H. King. 1981. *Another Dimension to the Black Diaspora: Diet, Disease and Racism.* Cambridge, U.K.: Cambridge University Press.

Kiple, K.F., and V.H. Kiple. 1977. Slave Child Mortality: Some Nutritional Answers to a Perennial Puzzle. *Journal of Social History,* 10:284–309. Krogman, W.M., and M.Y. Iscan. 1986. *The Human Skeleton in Forensic Medicine.* Springfield, Ill.: Charles C Thomas.

Lovejoy, C.O., R.S. Meindl, T.R. Pryzbeck, and R.P. Mensforth. 1985. Chronological Metamorphosis of the Auricular Surface of the Ilium: A New Method for the Determination of Adult Skeletal Age at Death. *American Journal of Physical Anthropology,* 68:15–28.

Marks, M.K. 1984. Congenital Treponematosis in an Early Twentieth Century Rural Black American Cemetery. *American Journal of Physical Anthropology,* 63:190.

McKern, R.W., and T.D. Stewart. 1957. Skeletal Age Changes in Young American Males, Analyzed from the Standpoint of Identification. *Technical Report Ep–45.* Natick, Mass.: Headquarters, Quartermaster Research and Development Command.

Ortner, D.J., and W.G.J. Putschar 1981. Identification of Pathological Conditions in Human Skeletal Remains. *Smithsonian Contributions to Anthropology* 28. Washington, D.C.: Smithsonian Institution Press.

Powell, M.L. 1985. Health, Disease, and Social Organization in the Mississippian Community at Moundville. Ph.D. dissertation, Northwestern University, Evanston, Ill.

Schour, I., and M. Massler. 1941. The Development of the Human Dentition. *Journal of the American Dental Association,* 28:1153–1160.

Scrimshaw, N.S., C.E. Taylor, and J.E. Gordon. 1968. *Interactions of Nutritions and Infection.* Geneva: World Health Organization.

Stampp, K.M. 1965. *The Era of Reconstruction 1865–1877.* New York: Alfred A. Knopf.

Steinbock, R.T. 1976. *Paleopathological Diagnosis and Interpretation: Bone Diseases in Ancient Human Populations.* Springfield, Ill.: Charles C Thomas.

Swedlund, A.C., and G.J. Armelagos. 1976. *Demographic Anthropology.* Dubuque, Iowa: William C. Brown.

Sylva, M.F. 1981. Black Populations in Arkansas. M.A. thesis, University of Arkansas, Fayetteville.

Ubelaker, D.H. 1982. The Development of American Paleopathology. In F. Spencer, ed., *A History of American Physical Anthropology 1930–1980,* 337–356. New York: Academic Press.

Van Gerven, D.P., and G.J. Armelagos. 1983. Farewell to Paleodemography? Rumors of its Death Have Been Greatly Exaggerated. *Journal of Human Evolution,* 12:353–360.

Watkins, B. 1985. Historical Background. In J.C. Rose, ed., *Gone to a Better Land: A Biohistory of a Rural Black Cemetery in the Post-Reconstruction South,* 6–17. Fayetteville: Arkansas Archeological Survey.

Weiss, K.M. 1973. Demographic Models for Anthropology. *Society for American Archaeology Memoirs,* 27.

SUMMARY OF AUDIENCE DISCUSSION: The frequency of infections in this reported group is four to five times higher than that found in the preceding slave period. Historical literature from the 1890–1930 period indicates biologists found Afro-Americans in such a severe state of demographic disequilibrium that they predicted their extinction in America by 1950. It may have been their participation in the public assistance programs of the 1930s that reversed the situation.

The endocranial periostitic lesions are intriguing since, if they were infectious, it is not easy to postulate how fatal meningitis was avoided for a period long enough to permit the observed osseous response. One possibility may be that the lesion was a reflection not of acute, pyogenic disease but rather of intrauterine treponematosis. This consideration is based on the slender evidence of observing that all cases were in neonates or feti and that Hutchinson's teeth evolve in a milieu of spirochete-infected tissue.

While it might be intriguing to view this as a population healthy enough to resist major infections, their calculated infant mortality and stillbirth rates are identical with the disastrous contemporary demographic statistics, indicating that this population was not healthier than others, but instead was in a devastatingly unhealthy state.

Paleopathological study on infectious diseases in Japan

Takao Suzuki

Today's clinical features of infection including its incidence, symptoms, course, and pathological morphology in living tissues have been dramatically changed by the widespread use of antibiotics after World War II. Inflammatory changes commonly found in archeological skeletal remains provide good evidence of the vivid reaction of extrinsic insults to the bone. However, it is not necessarily easy to diagnose the precise, causative disease producing such inflammatory changes based on the morphological features. On this point, Putschar (1966:59–60) stated as follows:

> Inflammatory bone lesions pose much more serious diagnostic problems and therefore deserve more detailed discussion. Periosteal deposition of bonewith or without thickening and architectural change of the underlying cortex of the diaphysis of long bones is probably the most common inflammatory lesion found in prehistoric material. The lesion often appears to be in a chronic or healed stage, and it is frequently impossible to ascertain whether the changes are traumatic or infectious in origin.

In the present study, inflammatory lesions appearing in archeological skeletal materials have been examined macroscopically and categorized in order to use them as an indicator of the health status in earlier populations. The procedure of this study deals with the classification of the inflammatory lesion in terms of gross morphology, and its limitations and availability in reconstructing a pattern of health. Even the equivocal lesions existing between nonspecific and specific disease, which may be an unavoidable problem in paleopathology, have been considered as a part of the available information for epidemiological analysis.

In this context secular changes regarding the frequency of infectious diseases in the skeletal populations in Japan have been examined and interpreted with respect to biocultural background. Among specific diseases, tuberculosis and syphilis have been extensively studied. These two diseases are viewed from the standpoints of origin, dissemination and prevalence in the Japanese archipelago.

Geographic, historic, and archeological background

The Japanese archipelago is made up of four main islands (Honshu, Shikoku, Kyushu, and Hokkaido) which together with more than 4000 smaller islands lie off the east coast of Asia showing a crescent shape. The climate of these islands, ranging from subfrigid to subtropic, is mostly a temperate and oceanic type with four distinct seasons. More than 60% of the land is mountainous, and most of this is covered with forests. All of these environmental factors may have a connection with the diseases of the inhabitants as well as the ecological, biological, and sociological factors.

The origin of the Japanese people is not altogether clear with respect to location and time. However, paleolithic stone implements suggest that their ancestors inhabited these islands for more than ten thousand years. The beginning of the Japanese neolithic period, Jomon, is still controversial and unproven to many anthropologists. However, this prehistoric period lasted at least several thousand years, and ended at about the third century B.C. The main subsistence strategies during the Jomon period were hunting, fishing and gathering. The Jomon people made a lot of clay utensils including pottery characterized by cord-marking (Jomon). The Jomon people were buried under shell mounds which have protected their skeletal remains from volcanic and acid soil.

During the Eneolithic period, Yayoi (third century B.C. to about third century A.D.), which followed the Jomon period, a large number of immigrants migrated to the western part of Japan via the Korean Peninsula. Immigrants mixed with the Jomon people who were aboriginals in this area. It was during this period that the Japanese mastered the art of rice cultivation, began to use metal implements, and set the fundamental pattern of Japanese tradition and life. From the paleopathological point of view, some new infectious diseases seem to have been transmitted from the Asian continent to Japan with such a huge number of immigrants at that time. According to the traditional history of Japan, the migration from the Korean Peninsula continued until around the eighth century A.D. Japanese living in western areas were likely to be affected by such migrants physically, although the Ainu in Hokkaido seemed to remain unmixed (Hanihara 1985). Therefore, the influence of the admixture which took place after the Yayoi period is still evident in western Japan. However, the eastern Japanese maintain some characteristics which are similar to the Ainu to a greater or lesser extent (ibid.).

After consolidation of Japan into a single nation in the fourth century, successive emperors strengthened the foundation of the country by introducing various aspects of continental learning and culture. These included the Chinese writing system, ideology (Confucianism) and religion (Buddhism). In particular, Buddhism recommended cremation, and after the protohistoric period people did not make shell mounds as a burial site, so that complete skeletal remains belonging to the protohistoric period are difficult to find except for some special burial cases.

Materials

The skeletal materials used in this study were from collections of various periods and sites housed in several universities. Only adult skeletons were examined for their pathological changes because of the abundance in quantity and quality. Four major skeletal series can be considered as the core of this study:

1. The Jomon skeletal series consisted of 272 individual skeletons from eight sites (seven from shell mounds and one from a cave site). Among them, six skeletal series (Hobi, Ko, Nakazawahama, Ubayama, Kasori, Yosekura) are housed in the Department of Physical Anthropology of the University Museum, The University of Tokyo, and they are registered and catalogued by Endo and Endo (1979). Two other Jomon skeletal series from Tsukumo and Yoshigo are housed in the Department of Physical Anthropology, Kyoto University.

2. The Edo skeletal series consisted of 308 femora and 253 tibiae housed in the Department of Physical Anthropology of the University Museum, The University of Tokyo. Whole, individual skeletons of the Edo period (latest medieval, earliest modern) could scarcely be excavated because most burial sites of this period, particularly in Edo (old Tokyo) city, were secondary and reburied sites. Therefore, the skeletal materials which were from five Edo sites (Unko-in, Joshin-ji, Fudo-ji, Hoden-ji and Edogawa-bashi) could not be identified as to age, sex, or individual. Besides the long bone material of femora and tibiae, 923 skulls of the Edo period which have already been studied by the author with respect to cranial syphilis (Suzuki 1984a) were used for this study.

3. The Ainu skeletal series consisted of 178 individual skeletons, and were from two northern islands: Hokkaido and Sakhalin. These skeletal materials, believed to be from the latest medieval and earliest modern Ainu, correspond to the Edo period and were collected by Koganei in Hokkaido (Koganei 1894) and by Kiyono in Sakhalin (Kiyono 1943,1949). The materials from the Hokkaido Ainu, called "Koganei collection," are housed in the Department of Anatomy of the University Museum, The University of Tokyo. The materials of Sakhalin Ainu, a part of the "Kiyono collection," are housed in the Department of Physical Anthropology, Kyoto University. The detailed paleopathological studies on these major Ainu skeletal series have already been conducted and reported by the author (Suzuki and Ikeda 1981; Suzuki 1984b,1985b).

4. The Meiji Japanese (early modern) skeletal series consisted of 113 whole, individual skeletal materials from the Kanto area (central part of Honshu). These materials are now housed in the Department of Anatomy, Sapporo Medical College. Their demography (sex, age and birthplace) is well recorded. These individuals ranged from 20 to 80 years old. They died between 1927 and 1944. Some osteological and physical anthropological studies on this series have been carried out by several authors (Mitsuhashi 1958; Wada 1975; Hashizaki and Kaneko 1979; Higuchi 1983).

The locations of the major sites of these four skeletal series are indicated in Figure 1 and detailed contents of individuals and skeletal parts of these series are listed in Table 1.

Furthermore, in addition to these four major skeletal series a few cases exhibiting interesting pathological conditions were used in the study. These special cases will be described as to the sources and data in each case.

Methods

GROSS OBSERVATION

Pathological changes appearing on the skeleton from Jomon to modern Japanese skeletal series were examined by gross observation and, in most cases, by x-ray film. Neither a histological nor a microscopic study has been carried out.

The examination was carried out only on adult skeletal remains, because the subadult skeletons were few and most of them were so badly damaged that the identification and pathology were difficult to ascertain. The skeletal parts observed in this study included skull, sternum, and vertebral column, and also limb girdle bones such as the scapula, clavicle, and pelvis, as well as long bones of the extremities including humerus, radius, ulna, femur, tibia, and fibula. Small bones of the hand and feet were excluded as well as fragmented ribs. From the Edo period only skull, femur, and tibia were examined because of the commingled state caused by reburials.

Lesions appearing in both the maxilla and mandible caused by periodontal diseases were excluded in this study. Those inflammatory changes, though frequently appearing in archeological specimens with abscess formation, should be classified into another category, such as "lesions of jaws and teeth" (Ortner and Putschar 1981:436–456).

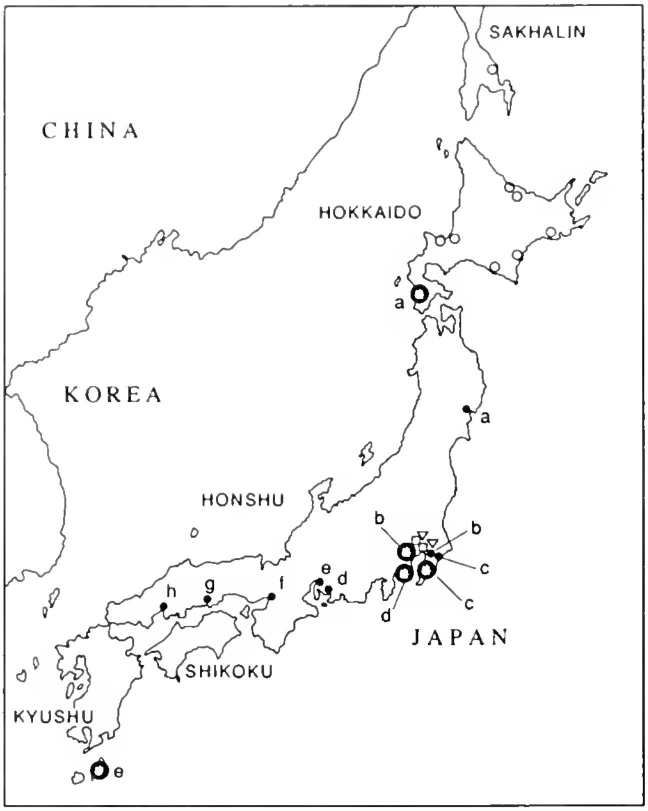


FIGURE 1. Major archeological sites from which skeletal remains were used in this study. Jomon sites *(small black circles)*: *a*, Nakazawahama; *b*, Ubayama; *c*, Kasori; *d*, Hobi; *e*, Yoshigo; *f*, Ko; *g*, Tsugumo; *h*, Yosekura. Yayio, Kofun, and Medieval sites *(large black circles)*: *a*, Katsuyama-Tate; *b*, Unoki-Kofun; *c*, Shiroyama-Kofun; *d*, Zaimokuza; *e*, Ichino-Torii. Edo (old Tokyo) sites: *open squares*. Early modern Japanese sites: *open triangles*. Ainu sites: *open circles*.

TAXONOMY OF INFLAMMATORY CHANGE IN THE BONE

The periosteum, cortex, and medulla of bone have such a close relationship that infectious change occurring in one part of the bone cannot help but influence another part. In dry-bone specimens, almost all infections usually can be classified as either osteomyelitis or periostitis. Periostitis is defined as periosteal reactive bone changes with irregular, fine-porous and spongy deposition located only exterior to the cortex with no involvement of the underlying cortex. The most common feature of such periosteal reaction tends to be "plaquelike" periostitis (Figure 2) which may show various degrees of severity or stages, as stated by Stothers and Metress (1975). Osteomyelitis is defined as inflammatory changes spread through the medullary cavity and, in many cases, the cortical bone. The most common feature of suppurative osteomyelitis is characterized by sequestrum, involucrum, and cloacal formation, particularly in the long bones (Figure 3). As is well known, there is an uncommon form of chronic osteomyelitis characterized by remarkable sclerosing of the lesion without any cloacal openings, which can usually be seen in the shafts of the lower extremities of adults (Figure 4), the so called "sclerosing osteomyelitis of Garré."

From the viewpoint of modern clinicopathology, inflammatory changes in living tissue are usually classified into two categories: nonspecific inflammation and specific inflammation. This classification is also available in the paleopathological field and actually has been used by some authors (e.g., Putschar 1966:60; Stothers and Metress 1975; Steinbock 1976:60,86; Ortner and Putschar 1981:104,129–138; Goodman et al. 1984). In this study, therefore, inflammatory bone changes are also categorized into two groups, nonspecific and specific bone inflammation. In the dry-bone specimen, nonspecific inflammation can be defined as an

TABLE 1. Skeletal material studied

Period	*Total no.*	*Sex*		*Skull*	*Vertebra*	*Femur*		*Tibia*	
						R	*L*	*R*	*L*
Joson	272	M	163	246	97	165	167	158	166
(prehistoric)		F	109						
Edo Japanese	923	M	636	923	-	172	136	139	114
(17th–19th C. A.D.)		F	287						
Meiji Japanese	113	M	86	106	113	113	113	113	113
(19th C. A.D.)		F	27						
Ainu	178	M	102	173	67	99	101	94	100
(early modern)		F	76						

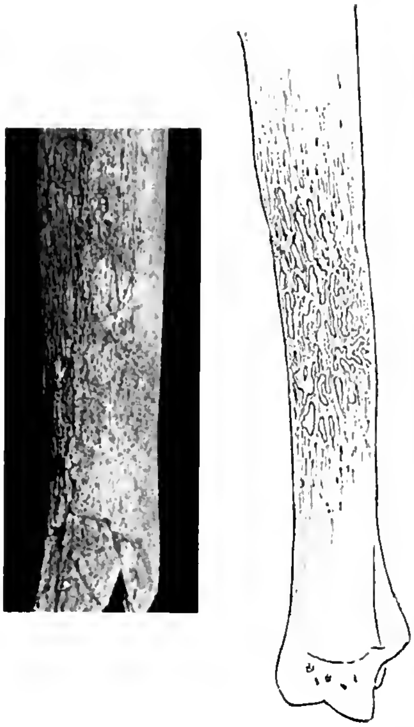


FIGURE 2. Schematic representation of moderate "plaquelike" periostitis and a case from Jomon site (Tsukumo shellmound).

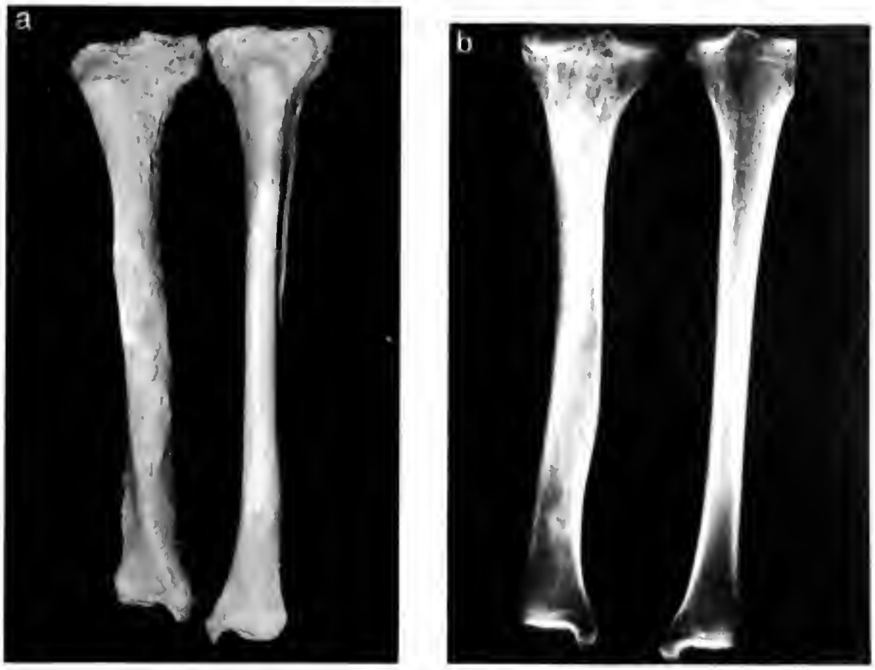


FIGURE 4. Sclerosing osteomyelitis of Garré from modern Japanese skeletal material: *a*, pathological *(right)* and normal *(left)* bone specimen; *b*, x-ray film.

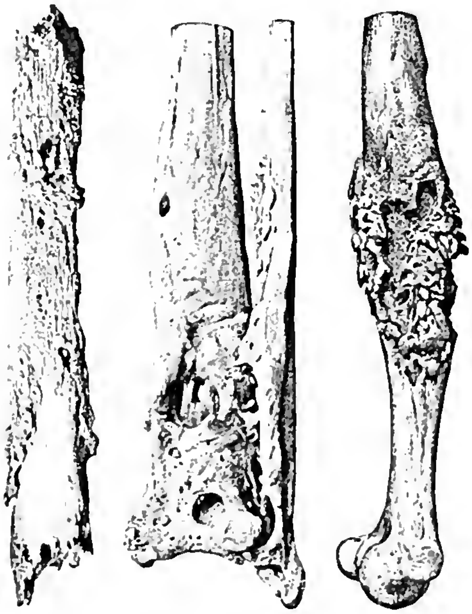


FIGURE 3. Schematic representation of advanced chronic osteomyelitis with sequestrum, involucrum, and cloaca formation (MacCallum 1920).

ordinary inflammatory reaction represented mainly by periostitis with plaquelike bone deposition and, in the advanced case, by osteomyelitis. On the other hand, specific inflammation can be characterized by a peculiar granulomatous lesion even in the dry bones which may be detectable in gross examination. Actually, in the typical/advanced cases of tuberculosis and syphilis, their inflammatory changes can be diagnosed by characteristic morphology and peculiar distribution of the lesion in the skeleton. In some cases, of course, geographical and epidemiological information should be considered in order to evaluate the bone lesion and differentiate between possible diseases which affect the skeleton in a similar fashion (Buikstra 1976).

However, in the case showing slight inflammatory bone change or even in the advanced changes with ordinal periosteal reaction, it is not always possible to differentiate these two categories with certainty. Only typical and demonstrable cases showing peculiar morphology caused by specific infection can be diagnosed in the dry-bone specimens. In other words, it is quite natural that overlapping between nonspecific and specific inflammatory change, particularly in the early stage of bone infection, would occur in archeological specimens. Therefore, the etiology of periostitis, which is very common in archeological specimens, cannot always be identified. This is one of the reasons why periostitis has often been treated as an independent entity in paleopathology. On this problem, Ortner and Putschar (1981:131) stated the following:

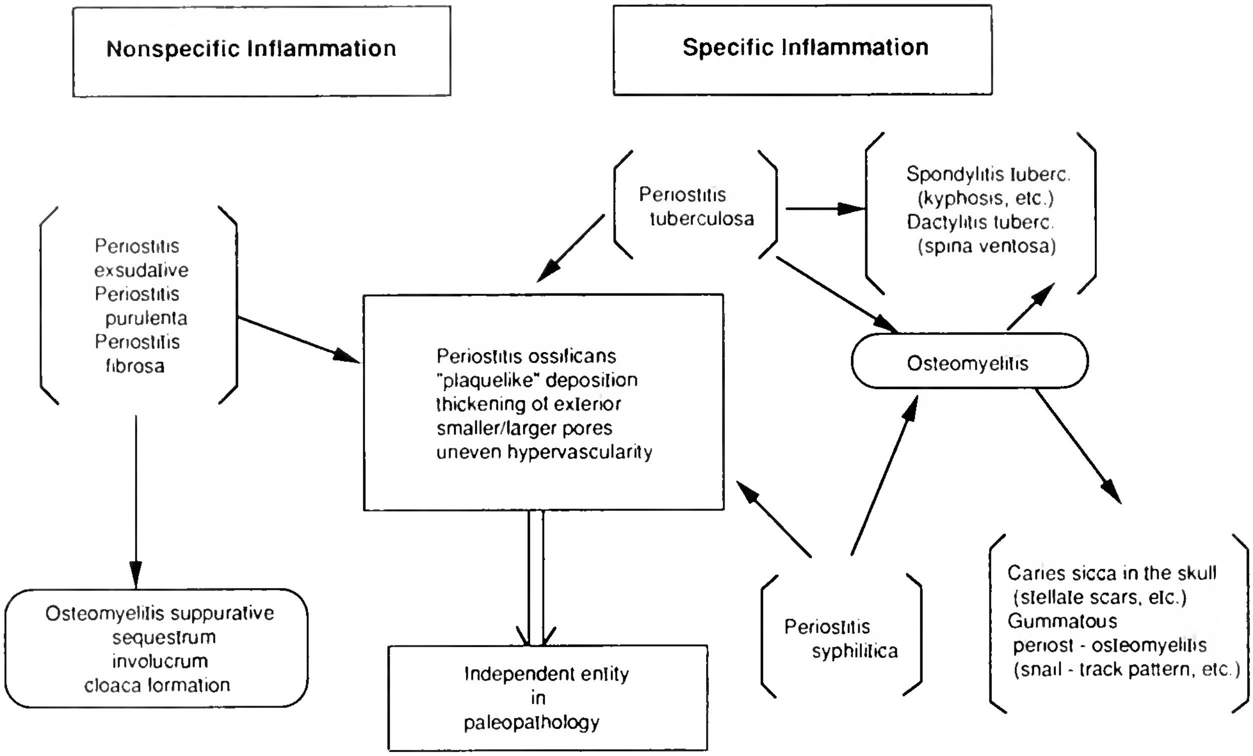

Nonspecific Inflammation
Specific Inflammation
Periostitis tuberculosa
Spondylitis tuberc. (kyphosis, etc.) Dactylitis tuberc. (spina ventosa)
Periostitis exsudative Periostitis purulenta Periostitis fibrosa
Periostitis ossificans "plaquelike" deposition thickening of exterior smaller/larger pores uneven hypervascularity
Osteomyelitis
Osteomyelitis suppurative sequestrum involucrum cloaca formation
Independent entity in paleopathology
Periostitis syphilitica
Caries sicca in the skull (stellate scars, etc.) Gummatous periost - osteomyelitis (snail - track pattern, etc.)

FIGURE 5. Lesions in specific and nonspecific inflammatory conditions of bone.

> The main reason for this difference between clinical and paleopathology is that many of the periosteal reactions may be part of the expression of a specific disease process, which can be identified in a living patient, whereas in archeological specimens the pathological characteristics necessary to make a specific diagnosis are not available. This would have the effect of increasing the frequency of nonspecific periostitis in archeological skeletal series.

Such a special situation of periostitis in paleopathology is shown in Figure 5. Thus in the present study, due to the possible admixture of these two kinds of infections among the archeological specimens, the author cannot help but deal with all inflammatory changes in the bone as nonspecific inflammation, excluding only the typical/demonstrable case showing specific changes of bone tuberculosis and syphilis.

Results

Gross examination of the infectious changes was conducted on four skeletal series: the Jomon (prehistoric) series including 272 individuals, the Ainu series of 178 individuals, the Meiji (early modern; antibiotic era) Japanese series of 113 individuals, and the Edo (latest medieval/earliest modern) Japanese series consisting of 923 skulls, 308 femora, and 253 tibiae. The data on cranial syphilis of the Edo series was quoted from the previous study carried out by the author (Suzuki 1984a).

FREQUENCY OF THE INFECTIOUS LESION. The results are shown in Table 2. The highest frequency can be seen in the Meiji Japanese series and the lowest was in the Jomon series.

SEX DIFFERENCES OF THE FREQUENCY. Except for the Edo Japanese series, the number and total frequency of infectious lesions in both sexes are shown in Table 3. There is no statistical significance between the male and female frequencies [$\chi^2 = 0.267 < p\,(0.01)$]. The frequency of cranial syphilis in the Edo series also fails to reveal statistical significance between both sexes [$\chi^2 = 2.04 < p\,(0.01)$].

SEVERITY OF INFECTIOUS CHANGE. Most infectious lesions of bone can be generally classified as either periostitis or osteomyelitis. As is well known, periostitis shows various degrees of involvement, from slight and weak change to severe and drastic (Stothers and Metress 1975; Lallo et al. 1978). In this study the severity of such infectious change was basically classified into periostitis and osteomyelitis, and periostitis was subdivided into two categories, slight and severe. Slight periostitis was defined as the extent of involvement of the periosteal surface (Lallo et al. 1978) corresponding to the stage I–II proposed by Stothers and Metress (1975). Severe periostitis was defined by the nature of tissue destruction—whether pitted, ridged, scarred, or showing sinus tracts (Lallo et al. 1978). The result is shown in Table 4. It should be noted that there is no clear evidence of osteomyelitis in the Jomon series.

TABLE 2. Frequency of infectious lesion types

Period (no.)	*Category of infection*	*No. (%) individuals*	*Total/no. (%)*
Jomon (272)	Nonspecific	26 (9.6%)	
	Specific tbc.	0	
	" syph.	0	
			26/272 (9.6%)
Ainu (178)	Nonspecific	14 (7.9%)	
	Specific tbc.	2 (1.1%)	
	" syph.	2 (1.1%)	
			18/178 (10.1%)
Meiji (113)	Nonspecific	9 (8.0%)	
	Specific tbc.	2 (1.8%)	
	" syph.	2 (1.8%)	
			13/113 (11.6%)
		subtotal	57/563 (10.1%)
Edo	Infectious lesion		
	Skull	89	89/923 (9.6%)
	Femur	21	21/308 (6.8%)
	Tibia	31	31/253 (12.3%)
	Syphilis in skull	50	50/923 (5.4%)

TABLE 4. Severity of the infectious lesion

Period	*Periostitis*		*Osteomyelitis*	*Total*
	slight	*severe*	*no. (%)*	
Jomon	21	5	0	26
Ainu	8	2	4 (28.6%)	14
Meiji	5	2	2 (22.2%)	9

TABLE 3. Sex differences of lesions' frequencies

Period	*Category of lesion*	*Male*	*Female*	*Total*
Jomon	Nonspecific	15	11	26
	Specific tbc.	-	-	-
	" syph.	-	-	-
Ainu	Nonspecific	6	8	14
	Specific tbc.	1	1	2
	" syph.	1	1	2
Meiji	Nonspecific	6	3	9
	Specific tbc.	2	-	2
	" syph.	2	-	2
	TOTAL infections	33/351	24/212	57/563
	TOTAL (%)	(9.4%)	(11.3%)	$X^2=0.267$
				$p<.01$
Edo	Syphilitic lesion	39/636	11/287	50/923
	in skull (%)	(6.1%)	(3.8%)	$X^2=2.04$
				$p<.01$

TABLE 5. Secular changes of the frequency of lesions in the femur and tibia

Period	*No./femur (%)*	*No./tibia (%)*
Jomon	17/332 (5.12%)	41/324 (12.65%)
Edo	21/308 (6.81%)	31/253 (12.25%)
Ainu	6/200 (3.00%)	3/194 (1.55%)
Meiji	6/226 (2.65%)	12/226 (5.31%)
Total	50/1066 (4.69%)	87/997 (8.73%)

SECULAR CHANGES OF THE FREQUENCY IN THE FEMUR AND TIBIA. The frequencies of infectious changes appearing in the lower extremities were compared. The reasons why the femur and tibia were selected in this examination are the following: (a) these bones, especially the tibia, are by far the most frequently affected by such infectious change (Goldstein 1957; Brothwell 1961), (b) their broad periosteal surface enables us examine them by gross observation with ease, and (c) a great number of isolated and unidentified femurs, tibias, and skulls in the Edo series at least can be used in the comparison of frequencies. The result is shown in Table 5. The frequencies which represent the infected bones, including both nonspecific and specific changes, are calculated from the total number of femurs and tibias from both sides.

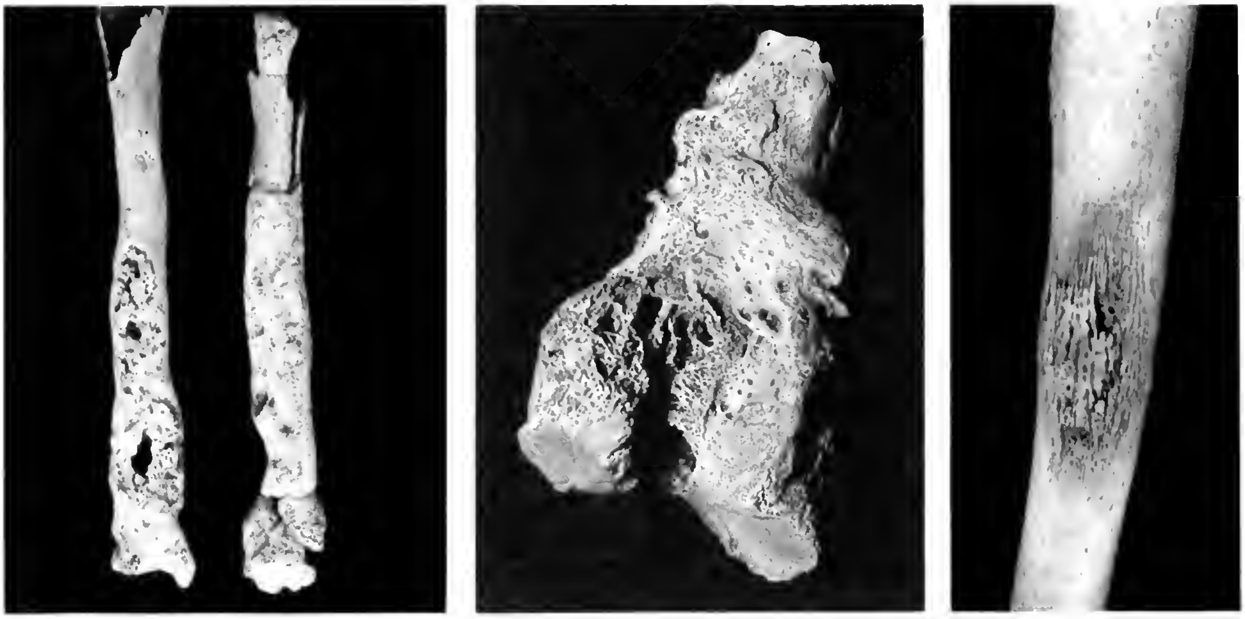


FIGURE 6. Three cases of advanced chronic osteomyelitis from Medieval period: *left*, right tibia of mature female from Ichino-Torii site (courtesy of Dr. A. Naito); *center*, distal end of right femur of adult from Zaimokuza site; *right*, left tibia of young individual from Katsuyama-Tate site.

EVIDENCE OF SPECIFIC INFLAMMATION OF BONE. There was no possible case of tuberculosis or syphilis in the Jomon skeletal series. Except for the Jomon series, typical cases of both tuberculosis and syphilis could be detected in the Edo, Ainu, and Meiji skeletal materials, as shown in Table 2. The frequency of these specific inflammatory diseases varies from 1.1 to 5.4% among the later three skeletal series.

Discussion

Based on a paleopathological study of the inflammatory lesions in the skeletal materials of the Japanese from the Jomon (prehistoric) to Meiji (early modern) periods and the Ainu population, the qualitative transition of the infectious disease will be discussed here from the standpoint of their bioecological background.

Taxonomically two major categories can be recognized: nonspecific inflammatory change including ordinary periostitis and osteomyelitis, and specific inflammatory change caused by tuberculosis and syphilis, which can be defined by the typical/demonstrative morphology and distribution of the lesion. In a field of paleopathology dealing with dry-bone specimens, it is sometimes inevitable to encounter the borderline case showing equivocal inflammatory lesion between nonspecific and specific. This means that periostitis appearing in the dry bones may be treated as an independent entity. In this study, cases showing such equivocal periosteal reaction ("periostitis ossificans") were included as nonspecific inflammation. In this sense, specific bone diseases diagnosed as tuberculosis or syphilis were detected with certainty only by the characteristic changes and distribution of bone lesions.

QUALITATIVE TRANSITION OF THE INFECTIOUS DISEASES

It should be emphasized that a relatively higher frequency of nonspecific inflammatory lesions represented by plaquelike periostitis appeared in the Jomon skeletal series. Furthermore, almost all of them showed slight/moderate periostitis limited to the exterior surface of the bone, and there was no case showing severe/advanced chronic osteomyelitis with such characteristic features as involucrum, sequestrum, and cloaca formation. It should be noted that no such advanced case of osteomyelitis has been found to date anywhere among the Jomon skeletal remains, whose number is the largest in this country. One of the oldest cases of severe advanced nonspecific osteomyelitis is from the medieval period (Figure 6). It seems that such advanced infectious cases may be found more frequently among the population after the Jomon period provided that the number of skeletal remains from Yayoi and Kofun period are complete and sufficient to examine paleopathologically.

Why is the frequency of advanced infectious cases considerably lower in the Jomon series? The Jomon society depended on hunting, gathering, and fishing activities. The Jomon population density was estimated to be quite low.

TABLE 6. Population number from earliest Jomon (prehistoric) to Kofun (protohistoric) period (Koyama 1984)

	Jomon					*Yayoi*	*Kofun*
Period	*earliest*	*early*	*middle*	*late*	*latest*		
Population number	20100	105500	261300	160300	75800	594900	5399800
Density/km²	0.07	0.36	0.89	0.55	0.26	2.02	18.37

TABLE 7. Reported palaeopathological cases of bone tuberculosis

Designation	*Period*	*Sex*	*Age*	*Location*	*Reporter*
Shiroyama – No. 3	Kofun	M	Adult	Spine (lumbar-sacrum)	Ogata 1972
Unoki – No. 3	Kofun	F	Mature – senile	Spine (thoracic-lumbar)	Suzuki 1978
Asahidai – No. 9	Kofun	M	Mature – senile	Spine (thoracic-lumbar) Ribs	Tashiro 1982
Ainu – A – 1336	Ainu	F	Adult	Sacrum	Suzuki 1985a

Koyama's (1984:10–39) estimation of the population from the earliest Jomon to the protohistoric period is shown in Table 6. The average population density calculated in the Jomon period (0.43/km²) is about one-fifth of the Yayoi period (2.02) and one-fortieth of the Kofun period (18.37). Furthermore, Kobayashi (1967) stated that the average age at the time of death for the individual over 15 years of age was 31.1 for males and 31.3 for females. It is quite probable that the Jomon people had rather short lives, probably caused by some environmental factors such as unstable food supply, hard labor in hunting-gathering-fishing activities, and unsanitary living conditions. These environmental factors also may have influenced the inflammatory process in the bone. The following tendencies can be suspected among the Jomon people: infection may extend rather easily to the bone, and individuals involved in such an infectious process tend to die before periostitis becomes chronic and develops into more advanced osteomyelitis.

On the other hand, during the historic periods, probably due to the development of agriculture and gradual improvement of hygienic conditions, individual resistance against infectious diseases may change to produce more severely infected conditions of the bone as well as lengthen the average span of life at the social level. However, at the same time it should be noted that other new epidemic diseases had been introduced and prevailed widely in this country at that time, accompanied by an increase in population density and domiciliation. These epidemic diseases consisted of the two specific infections, tuberculosis and syphilis.

ORIGIN AND PREVALENCE OF SPECIFIC DISEASES IN JAPAN

It is remarkable that there have been neither typical nor suspected cases showing specific inflammatory bone changes among a huge number of Jomon skeletal remains excavated from various archeological sites in the Japanese archipelago. In the present study also, except for the Jomon skeletal series, typical/demonstrable cases of tuberculosis and syphilis were limited to the Edo and Meiji Japanese skeletons as well as the Ainu skeletal series.

Four typical cases of spinal tuberculosis have been reported to date in Japan (Ogata 1972; Suzuki 1978; Tashiro 1982; Suzuki 1985a) as shown in Table 7. None of them belonged to the prehistoric (Jomon) population from which a great deal of well-preserved skeletons have been studied. The three oldest cases (Figure 7) belong to the protohistoric (Kofun) population in which there are considerably fewer skeletons than in the Jomon population. There seem to be two alternative possibilities why no case of bone tuberculosis has been found among so many Jomon skeletal remains. The first hypothesis attributes it to the lower population density of the Jomon period. Tuberculosis is a density-dependent disease and may have become established in human populations as the result of the appearance of such population aggregates (Cook 1984). In this sense tuberculosis might have existed in a very limited area as a local endemic form. Tuberculosis reached epidemic proportions after the Yayoi or Kofun period

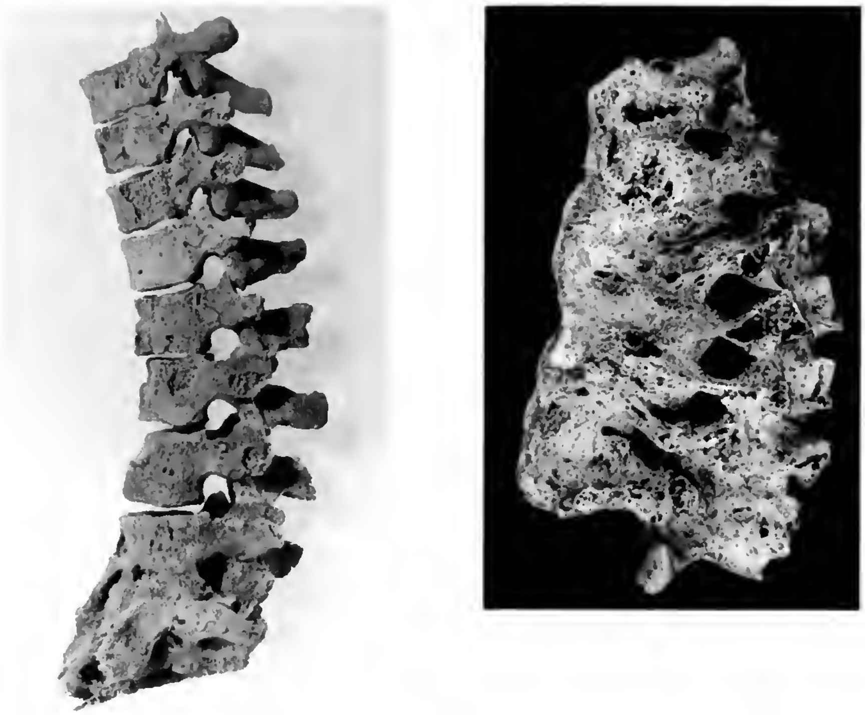


FIGURE 7. Two cases of tuberculosis in the Kofun period: *left*, spine of adult male from Shiroyama-Kofun site (courtesy of Dr. T. Ogata); *right*, spine of mature female from Unoki-Kofun site.

following a rapid increase of population density. Nevertheless, it is still strange that not a single case of bone tuberculosis has been found from the Jomon skeletal remains. As Morse (1961) pointed out, if prehistoric tuberculosis did exist there should be many cases of typical spinal tuberculosis found among the large amount of excavated skeletal material. The second hypothesis is more probable, dealing with the migration of the causative organism *Mycobacterium tuberculosis*. A great number of immigrants from the Asian continent migrated to this island country via the Korean Peninsula during the Kofun period (protohistoric, ca. third to seventh century A.D.). Not only did they introduce various cultural characteristics including the Chinese writing system, agricultural methods, and crafts, but they brought some new infectious diseases, probably including smallpox and tuberculosis, which disseminated throughout the country accompanying a rapid increase of population. The oldest written record of respiratory tuberculosis appeared in a Buddhist medical book published in the 12th century in Japan. Many medical documents, particularly in the Edo period, described a high prevalence of lethal, respiratory tuberculosis among the people.

Unlike tuberculosis, the origin, dissemination and prevalence of venereal syphilis have already been elucidated by the old medical documents. According to medical historians (e.g., Fujikawa 1904:172–177,1912:42; Dohi 1921:70–75), the first outbreak of a virulent venereal infection appeared in this country in the decade following 1510, the late Muromachi period, and suddenly prevailed in epidemic proportions. The oldest documents describing the appearance of syphilis in Japan are two documents written in A.D. 1512 and 1513. They showed that the first large outbreak of syphilis was in the western part of Japan and then spread to the northeastern part within a year or so. The people at that time called this curious epidemic disease *Tau-mo* (Chinese eruption) or *Ryukyu-mo* (Ryukan eruption), which represent the original epidemic area before they were involved. This virulent epidemic of syphilis struck the unsuspecting Japanese without distinction as to age and sex. During the Edo period, the people were very familiar with syphilis and called it by various names. Many documents deal with the clinical manifestations and therapies for syphilis during the mid and latter Edo period.

Koganei (1894) described a case of an Ainu archeological skeleton showing typical changes of osseous cranial syphilis (Figure 8), and Suzuki (1963:13–15) reported the oldest case of osseous cranial syphilis from the Muromachi period (Figure 9), stating that 3 out of 23 skulls (13.0%) had the typical features of osseous syphilis. Another old case showing syphilitic change in the skeleton from a ruin of a medieval castle in

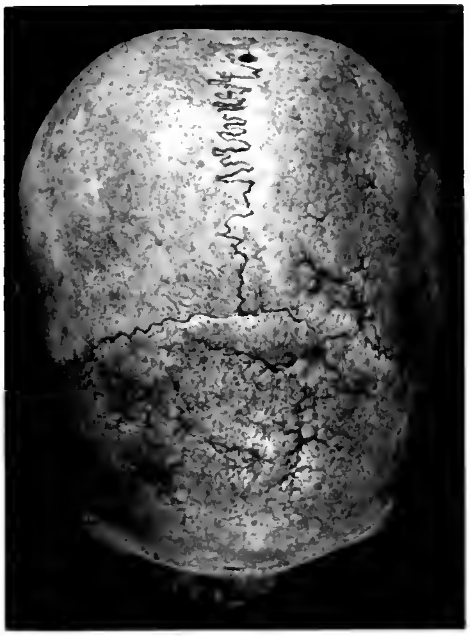


FIGURE 8. Typical demonstrable case of cranial syphilis from an Ainu skeletal remains (originally reported by Y. Koganei in 1894).

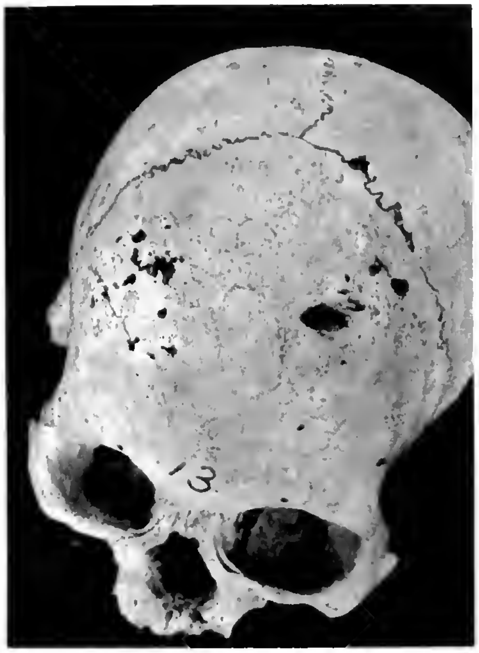


FIGURE 9. Typical demonstrable case of cranial syphilis from the Muromachi (medieval) skeletal remains (originally reported by H. Suzuki in 1963).

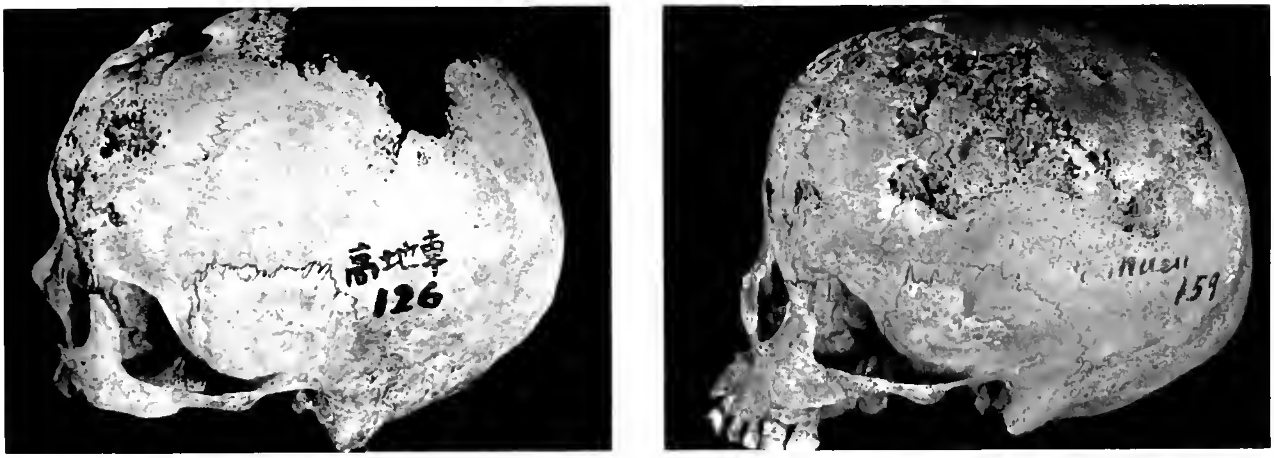


FIGURE 10. Two typical demonstrable cases of cranial syphilis from the Edo (latest medieval/earliest modern) skeletal remains (originally reported by the author in 1984.)

Hokkaido has been reported by the author (Suzuki 1984c). Furthermore, on the subject of the epidemic of syphilis during the Edo period, 50 of 923 (5.4%) Edo skulls could be diagnosed as demonstrable cases of osseous syphilis (Figure 10) and in the biocultural background of the Edo people and its society these were interpreted as proving a high prevalence of syphilis (Suzuki 1984a).

Another major specific disease that affects the skeletal system, particularly the facial and foot bones (Møller-Christensen 1964), is leprosy. Recently some archeological cases from the medieval and early modern periods have been thought to be possible cases of leprosy in this country, though to date they have not been published. There are also no cases of leprosy found in the Jomon skeletal series.

Nonvenereal forms of treponematosis such as yaws (frambesia) and pinta must be differentiated from venereal osseous syphilis if these two diseases coexist in the same area. In Japan, however, there has been no evidence of the existence of such tropical, endemic, nonvenereal infections either in the past or at the present time.

Conclusions

A paleopathological study emphasizing infectious bone diseases was conducted on the skeletal materials from Jomon (prehistoric) to Meiji (early modern) Japanese and the Ainu people. The frequencies, sex differences, and severity of the inflammatory lesions in the bones are presented as well as some interesting cases from various archeological sites. A taxonomical problem in the description of inflammatory changes is also discussed.

Among a large number of the Jomon skeletal remains there was no evidence of specific infectious diseases. The inflammatory changes represented by slight/moderate plaquelike periostitis in the Jomon series seemed to be caused only by nonspecific infections. On the other hand, the other skeletal series included not only typical/demonstrable bone tuberculosis and syphilis but also chronic/advanced osteomyelitis besides ordinary plaquelike periostitis, which document the coexistence of both nonspecific and specific infections in the Edo, Meiji, and Ainu populations. This difference found between the Jomon and the other series was consistent with biocultural differences such as the introduction of agriculture, settlement, increase of population density and urbanization in this country.

The qualitative changes of bone infection by the two specific infectious diseases of tuberculosis and syphilis were also considered from the standpoint of their origin (transition), dissemination and prevalence in this country.

Acknowledgments

The author is deeply indebted to Professor Akio Yamauchi of the Department of Anatomy (III), Faculty of Medicine, The University of Tokyo, Professor Kazuro Hanihara and Associate Professor Banri Endo of the Department of Anthropology, Faculty of Science, The University of Tokyo, and Professor Jiro Ikeda of the Department of Anthropology, Kyoto University, for their kind permission to examine the valuable materials in their universities.

The author is also very grateful to Professor Yoshiatsu Naito of the Department of Anatomy (II), Faculty of Medicine, Nagasaki University, and Professor Takahiko Ogata of the Department of Oral Anatomy (II), Kagoshima University Dental School, for allowing him to use their valuable illustrations of infectious diseases in the bones.

The author also wishes to express his gratitude to Donald J. Ortner, Curator of the Department of Anthropology, Smithsonian Institution, for his kindness in reading the manuscript and offering many helpful suggestions.

Literature cited

Brothwell, D. 1961. The Paleopathology of Early British Man: An Essay on the Problems of Diagnosis and Analysis. *Journal of the Royal Anthropological Institute*, 91:318–344.

Buikstra, J.E. 1976. Differential Diagnosis: An Epidemiological Model. *Yearbook of Physical Anthropology*, 20:316–328.

Cook, D.C. 1984. Subsistence and Health in the Lower Illinois Valley: Osteological Evidence. In M.N. Cohen and G.J. Armelagos, eds., *Paleopathology at the Origin of Agriculture*, 235–270. New York: Academic Press.

Dohi, K. 1921. *The World History of Syphilis*. Tokyo: Keiseisha.

Endo, Y., and B. Endo. 1979. *Catalogue of Skeletal Remains from Neolithic Jomon Period in Japan Preserved in the University of Tokyo*. Material Report 3. Tokyo: University Museum, University of Tokyo.

Fujikawa, Y. 1904. *The History of Medicine in Japan*. Tokyo: Keiseisha.

———. 1912. *The History of Diseases in Japan*. Tokyo: Nipponsha.

Goldstein, M.S. 1957. Skeletal Pathology of Early Indians in Texas. *American Journal of Physical Anthropology*, 15:299–307.

Goodman, A.H., J. Lallo, G.J. Armelagos, and J.C. Rose. 1984. Health Changes at Dickson Mounds, Illinois (A.D. 950–1300). In M.N. Cohen and G.J. Armelagos, eds., *Paleopathology at the Origins of Agriculture*, 271–305. New York: Academic Press.

Hanihara, K. 1985. Origins and Affinities of Japanese as Viewed from Cranial Measurements. In R. Kirk and E. Szathmary, eds., *Out of Asia*, 105–112. Canberra: Journal of Pacific History.

Hashizaki, T., and M. Kaneko. 1979. Study of the Spinal Canal Stenosis with Special Reference to Its Bone Factors. *Sapporo Medical Journal*, 48:143–156.

Higuchi, Y. 1983. A Study on the Morphological Changes in the Human Cervical Apophysial Joints with Increasing Age on Macerated Skeletons. *Sapporo Medical Journal*, 52:181–204.

Kiyono, K. 1943. *A Study on the First Man in Japan*. Tokyo: Hagiwara-Seibunkan.

———. 1949. *The Racial Problems Based on the Osteological Studies of the Human Skeletal Remains in Japan*. Tokyo: Iwanami-Shoten.

Kobayashi, K. 1967. Trend in the Length of Life Based on Human Skeletons from Prehistoric to Modern Times in Japan. *Journal of the Faculty of Science*, 3:109–160. University of Tokyo, Section 5, Anthropology.

Koganei, Y. 1894. Beitrage zur Physischen Anthropologie der Ainu. 1. Untersuchungen am Skelete. *Mitteilungen Medicinischen Fakultat Kaiserlich*, 2:1–250. Japan Universitat.

Koyama, S. 1984. Jomon Period. *Chuko-Shinsho*, 733. Tokyo: Chuko-Koronsha.

Lallo, J., G.J. Armelagos, and J.C. Rose. 1978. Paleoepidemiology of Infectious Disease in the Dickson Mounds Population. *Medical College of Virginia Quarterly*, 14:17–23.

MacCallum, W.G. 1920. *A Text-book of Pathology*. 2d edition. Philadelphia: W.B. Saunders.

Mitsuhashi, K. 1958. Anthropologic Studies of the Skulls of the Inhabitants of Kanto District in Japan. *Journal of the Chiba Medical Society*, 34:248–283.

Møller-Christensen, V. 1964. Evidence of Leprosy in Earlier Peoples. In D. Brothwell and A.T. Sandison, eds., *Diseases in Antiquity*, 295–306. Springfield, Ill.: Charles C Thomas.

Morse, D. 1961. Prehistoric Tuberculosis in America. *American Review of Respiratory Disease*, 83:489–503.

Ogata, T. 1972. Paleopathological Studies on the Ancient Japanese Skeletons. *Niigata Medical Journal*, 86:466–477.

Ortner, D.J., and W.G.J. Putschar. 1981. Identification of Pathological Condition in Human Skeletal Remains. *Smithsonian Contribution to Anthropology*, 28. Washington, D.C.: Smithsonian Institution Press.

Putschar, W.G.J. 1966. Problems in the Pathology and Paleopathology of Bone. In S. Jarcho, ed., *Human Paleopathology*. 57–65. New Haven, Conn.: Yale University Press.

Steinbock, R.T. 1976. *Paleopathological Diagnosis and Interpretation—Bone Diseases in Ancient Human Population*. Springfield, Ill.: Charles C Thomas.

Stothers, D.M., and J.F. Metress. 1975. A System for the Description and Analysis of Pathological Changes in Prehistoric Skeletons. *Ossa*, 2:3–9.

Suzuki, H. 1963. *Human Skeletal Remains of the Ancient Japanese Population*. Tokyo: Iwanami-Shoten.

Suzuki, T. 1978. A Paleopathological Study of the Vertebral Columns of the Japanese from Jomon to Edo Period. *Journal of the Anthropological Society of Nippon*, 86:321–336.—-. 1984a. Paleopathological and Paleoepidemiological Study of Osseous Syphilis in Skulls of the Edo Period. *University Museum, University of Tokyo Bulletin*, 23:1–48.

———. 1984b. Paleopathological Study on Osseous Syphilis in Skulls of the Ainu Skeletal Remains. *Ossa*, 9–11:153–168.

———. 1984c. Typical Osseous Syphilis in Medieval Skeletal Remains from Hokkaido. *Journal of the Anthropological Society of Nippon*, 92:23–32.

———. 1985a. Paleopathological Diagnosis of Bone Tuberculosis in the Lumbosacral Region. *Journal of the Anthropological Society of Nippon*, 93:381–390.

———. 1985b. Paleopathological and Paleoepidemiological Studies on the Human Skeletal Remains from Hokkaido and Sakhalin Islands. *Journal of the Anthropological Society of Nippon*, 93:216.

Suzuki, T., and J. Ikeda. 1981. A Paleopathological Study of Craniosynostosis–A Case of Plagiocephaly. *Journal of the Anthropological Society of Nippon*, 89:479–492.

Tashiro, K. 1982. Paleopathological Study of Human Bones Excavated in Kyushu, Japan. *Nagasaki Medical Journal*, 57:77–102.

Wada, J. 1975. Study of Changes in the Vertebral Bodies of Japanese with the Advance of Age. *Sapporo Medical Journal*, 44:139–152.

SUMMARY OF AUDIENCE DISCUSSION: Historical records suggest venereal syphilis was introduced into Japan from China in 1512, a record consistent with the absence of treponemal lesions in the series reported here from Japan's Jomon period. Tuberculosis is not identifiable in these series until after the Jomon period either. The Jomon people had no or few domestic animals.

Epidemiological aspects of paleopathology in Denmark: Past, present, and future studies

Pia Bennike

Paleopathological studies have been carried out in Denmark throughout the last hundred years, and with a few exceptions, the developmental stages do not seem to differ much from the general history of paleopathology known in other countries. The three stages are: the descriptive stage, the epidemiological stage, and the analytical stage. The number and subject of the studies within each stage may vary considerably, dependent on the time and the traditions of the geographical area.

Some of the early Danish studies of epidemiological character have obtained international interest. This is due to several factors, one of them being the long archeological tradition in Denmark resulting in a well-documented skeletal collection with reliable datings. In large parts of Denmark the soil contents provide extremely good conditions for the preservation of bones, and many Danish skeletons and bog bodies have been known to a wide audience, owing to the many details which could be studied. The size and the relative homogeneity of the Danish population is also very suitable for studies of epidemiological aspects. Finally, an important factor is the role of the investigator, which must not be forgotten.

In the following pages, we give a short presentation of the more important Danish studies dealing with paleopathology from past and present. Together they show the development, but also the restrictions, of this topic in Denmark over the years. We then discuss the results of the different studies in relation to the planning of future projects.

Past

As paleopathological studies in Denmark are mainly dependent on remains of human bones, the history can naturally not be traced farther back than to the establishment of the skeletal collection.

In an 1837 publication the Danish scientist Eschricht requested that more skeletal material be preserved during excavations. At that time the number of prehistoric skulls could be counted on the fingers of one hand. Eschricht's interest in acquiring more skeletal material was based on his wish to test certain theories concerning the Danish prehistoric population. Several scientists had proposed that the ancestors of the Scandinavian people may have been either Eskimos or perhaps Lapps, and this hypothesis could only be tested by studying a larger number of skulls (Nilsson 1838; Eschricht 1841). The theories were based on a study of very few skulls with some eskimoid traits, but later studies showed that those traits were not common in most skulls and may be considered as an isolated variation. It also demonstrates that the smaller the volume of material studied, the easier it is to draw conclusions. This is the case in studies of both physical anthropology and paleopathology. Whether the subsequent increase in the number of recovered prehistoric skulls was due to Eschricht's request or was a natural result of the developing scientific interest in anthropology at the time is not known.

By the time the German professor Virchow in 1870 visited Copenhagen to study the Danish Neolithic population, more than 50 skulls were available (Virchow 1870). As most scientists of that time, Virchow focused on questions of normal variation and racial types and not on pathological conditions, but from that time on skeletal material was available for paleopathological studies. The studies of the period on paleopathological aspects were, however, few and the topics were mostly restricted to mere descriptions, often of lesions, infections or trepanations on single finds (Engelhardt 1877; Hansen 1889,1913; Kjær 1912; Nielsen 1911).

At an international conference in France in 1867 Broca demonstrated that traces on a Peruvian skull, previously interpreted as a lesion caused by a wound, were probably due to treatment, the so-called trepanation (Broca 1867). It is not difficult to imagine how exciting it must have been to the participants of that conference to look for similar cases in their "own" collections after their return. Several studies on trepanation were published in the years shortly after Broca's presentation, showing that at least some of his colleagues had success in "discovering" skulls with evidence of trepanation (Hansen 1889,1913; Nielsen 1911).

Toward the end of the last century, a tradition of sending skeletal material from excavations to the Institute of Anatomy in Copenhagen was established. Usually the old bones were not paid much attention there, but professor of anatomy Dr. Fr. C.C. Hansen was one of the few who became interested in physical anthropology. He was a coauthor of *Crania Groenlandica* (Furst and Hansen 1915) but is probably best known in the paleopathological world for his work on the remains of 25 skeletons of Norsemen excavated at Herjolfnes in Greenland (Hansen 1924).

From written sources we know that the Norsemen settled in Greenland in the middle of the 10th century, but we also know that they perished for some reason, probably during the 15th century. Theories on their disappearance have been proposed, such as: the Norsemen were killed by the Eskimos, the Norsemen may have become assimilated with the Eskimos, the Norsemen may have sailed away, or the Norsemen died from starvation. In spite of several studies, it is interesting that nobody has ever been able to confirm any of the theories and no satisfactory explanation has ever been advanced of how the medieval Norse settlers in Greenland perished. On the contrary, several findings from studies of the Norsemen's environment have shown that none of the theories above can stand alone.

After studying the skeletons from a churchyard at Herjolfnes in the older East settlement of Greenland, Hansen, however, drew the provoking conclusion that the Norsemen became extinct owing to inbreeding. His conclusion was based on the appearance of minor skeletal changes such as osteophytes, caries, paradontosis, and similar changes "commonly" seen in paleopathological studies. A pelvic bone according to Hansen was marked with rachitic changes but Fischer-Møller (1942) later demonstrated that it was so badly damaged and deformed by postmortem changes in the grave that no clear conclusions could be drawn on the basis of this material. From an anthropometric point of view, Hansen furthermore based his conclusion on the small stature and also the relatively small volume of the skulls.

All the single cases on which Hansen based his theory were later reexamined by Fischer-Møller, who also studied newly excavated skeletons of Norsemen from Greenland. Fischer-Møller was able to reject almost all the cases. Contrary to Hansen he did not find any evidence of abnormal degeneration as a result of inbreeding. It would be too extensive to go into the details of Fischer-Møller's rejection of the single cases here. A repetition of Hansen's concluding remarks would probably be useful in showing today's paleopathologists how dangerous it can be to draw conclusions from paleopathological studies without having long experience in studying skeletal changes and without having any comparative studies to lean on, in order to learn which changes are common and which are not. Hansen wrote:

> The vigorous northern race that originally colonized Greenland degenerated in the course of the centuries under the influence of the hard and at last constantly deteriorating life conditions and other unfavorable conditions, especially isolation both intellectually, materially and as regards race hygiene. It became a race of small people, frail, physically weakened, with many defects and pathologic conditions.
>
> That a race so small in number, so weakened both by internal and external unfavorable life conditions has nevertheless been able to stay so gallantly at its post so long, much longer than was before conceived possible, speaks highly of the original quality of the race.
>
> In the fight the Norse must no doubt have been superior to the Eskimos, but the descendants of the people who sailed "westward on the sea" in small open boats and settled on the inclement shores of Greenland, defying nature through centuries, often at war with one another, did not succumb in struggle with men alone. Against constantly more severe physical conditions, against cold and a slow periodical starvation and in greater and greater isolation the northern race could not at last defend itself. Influences of nature which slowly and insidiously through the short lifetime of several generations undermined the vitality of the race itself could in the long run only be conquered by help from the main country and by an inflow of fresh blood to give vitality to the enfeebled race. . . . This did not happen. . . . Its doom was sealed by the ice of Greenland. (Hansen 1924:520–521)

The basis of this conclusion may be due to the investigator's poor knowledge of what is normally seen of so-called pathological changes in skeletal studies. If Hansen was right in his interpretation based on less severe degenerative traits such as osteoarthritis, the whole human race should have become extinct thousands or maybe millions of years ago. Almost all adult skeletons show evidence of osteoarthritic changes.

When most paleopathologists first started the study of human skeletons, they probably remember how often they became excited by bones with pathological changes, which later turned out to be quite common and almost within the frame of normality. It is probably evident to all that the study of paleopathology demands both long experience and an extremely wide knowledge of the nonpathological variations. Most colleagues of anthropology are specially trained in this field and are familiar with the literature available. If we want to exclude mistaken conclusions such as those Hansen drew, it is necessary to develop a strong and wide collaboration between the anthropologists who have long experience in skeletal studies and the specialists of the different medical topics.

It must be admitted that most anthropologists working in the field of paleopathology are clearly somewhat restricted in their studies because of their relatively small experience in modern anatomy and pathology. A collaboration with experts in some medical fields is therefore highly recommended. It is, however, not always a pathologist who is needed. Sometimes a specialist in orthopedic surgery, a clinical radiologist, or a dentist may be the best person to involve in the different cases.

If we look at the epidemiological aspect of the past, studies of this kind already began in Denmark with the previously mentioned work on Norsemen by Hansen (1924), but it was soon followed in 1936 by a study of 400 medieval skeletons excavated at Øm monastery (Isager 1936). Isager, who examined the bones, had a medical background as a general practitioner and was naturally most interested in describing the unusual pathological finds. Without having contemporary material for comparison his study was mainly a description of the pathological appearance of single bones. He also counted the number of bones with different kinds of defects, for example bones with evidence of healed fracture. However he did not count the number of normal bones, so no frequencies of the pathological changes in this material are known. Epidemiologically the study is therefore of limited value, but contrary to Hansen, Isager was aware of not going to extremes in his interpretation of the results before he had comparative material.

In the following decades the works of Møller-Christensen totally dominated the field of paleopathology in Denmark (1953, 1958, 1961, 1963, 1967, 1978). Møller-Christensen started his activity in 1935 by excavating more than 700 medieval skeletons at Æbelholt monastery on Zealand (Møller-Christensen 1958). He mainly studied the many pathological changes, which resulted in an excellent and useful publication, which unfortunately to date has been published only in Danish. The book contains tables with frequencies of almost all kinds of pathological changes seen in the medieval skeletons, and the results can therefore be compared to all later results of paleopathological studies. This makes the book on the skeletons from Æbelholt monastery much more useful than the one based on the skeletons from Øm monastery. It is also interesting to see the difference in the number of diagnoses mentioned in the two studies by Isager and Møller-Christensen. The number of different diagnoses is quite small in the book by Isager, while Møller-Christensen mentions more than three times as many. This may primarily be owing to the different years in which the studies were carried out and to new methods developed during the second half of this century, which allowed more precise diagnoses to be made and described.

After Møller-Christensen finished his study on diseases in the Middle Ages based on the many skeletons from Æbelholt monastery, he carefully planned his next study. One of the Æbelholt skeletons had shown some changes that might have been caused by leprosy. During his search for comparable bones affected with leprosy he realized that very little was known of skeletal changes due to this disease. He therefore decided to elucidate this topic. From historical records it was known that the so-called St. George houses were scattered throughout Denmark during the Middle Ages. People affected with leprosy were forced to live there for the rest of their lives, separated from their friends and families. Møller-Christensen succeeded in locating the place where one of these houses and the surrounding graves were situated.

After acquiring permission to start excavating the graves in 1941, more than 200 skeletons were recovered. This material made it possible for the first time to study changes in bone due to leprosy on the basis of several hundred skeletons affected with the disease. This was indeed a collection of high value for paleopathological studies (Møller-Christensen 1961,1967,1978). Møller-Christensen's exceptional study made it possible to add new points to the clinical diagnosis of leprosy. The typical changes of the maxilla and of the nasal aperture with the loss of the nasal spine had not been described in skeletons before. His studies may also in an international sense be labeled as unique. Often our studies in paleopathology are restricted by a small sample size or material somehow selected to produce underrepresentation of certain age or sex groups. In such cases its epidemiological value may be questioned. Sample size was not the problem in Møller-Christensen's material from the leprosarium. The skeletons excavated at Næstved are naturally not representative for the average medieval population, but together they form an interesting group consisting of those who were expelled from the normal community because of a specific disease. The study of this group was the aim of Møller-Christensen's work.

In recent years a new group of skeletons of several hundred individuals from almost the same area and period has been excavated. Future detailed studies will show whether the medieval people from Næstved really managed to diagnose and expel all those affected with leprosy. Møller-Christensen found that about 80% of the skeletons from the leprosarium showed some of the characteristic changes in bone due to leprosy, whereas this author, in a preliminary study of the skeletons from a common cemetery within the town, did not find any changes due to leprosy in the skeletons.

Today, when Acquired Immunodeficiency Syndrome (AIDS) seems to threaten the modern world's population and the discussions of how to prevent and restrict contamination are varied and often heated, it is interesting to go back 600–700 years to see what happened in a somewhat similar situation. At that time people who were suspected of having leprosy were doomed to spend the rest of their lives isolated in the St. George houses, and it is the remains of those people we are able to study today. Today leprosy is almost extinct in the West, but is still known in many developing countries.

From modern studies we now know that leprosy is not highly infectious, and it is not necessary to force the affected people to live their whole lives isolated and expelled from the community; they can lead a normal life (Korn 1982). The best way to prevent the disease is to provide better living conditions. In a very poor area in Norway the disease disappeared only in the beginning of this century. The medieval people at the leprosarium seem to have suffered not only from the disease but also from condemnation by healthy people who wanted to protect themselves. Let us not repeat this mistake by isolating those affected with AIDS. In this case the bones of the dead may teach the living what should NOT be done.

Present

Within the last decade several studies on different paleopathological aspects have been carried out, and several reports on dentition (Lunt 1978) and cervical changes of the back in relation to dental abscesses (Ingelmark 1956) have been based on the medieval skeletons from Æbelholt monastery.

A very interesting study was also carried out by Andersen (1969) who compared the skeletal changes of the medieval skeletons from Næstved with people affected with leprosy in living populations from India.

Smaller studies of pathological changes found on mainly newly excavated skeletons have continuously been published.

A recent study by the author was primarily based on Danish prehistoric skeletons and seems to constitute an additional step in the development of pathological studies on epidemiological aspects (Bennike 1985). As an important point, the study provided us a catalogue of information on the dating of all Danish prehistoric skeletons available for future studies of paleopathological aspects. This introductory part of the study was based on an archeological/anthropological collaboration which later resulted in several joint publications of some of the special finds (Bennike and Ebbesen 1986; Bennike et al. 1986). As a part of the work on the paleopathology of prehistoric skeletons all bones were registered in a computer system containing changes of pathological, traumatic, or degenerative character of the joints, the long bones, the irregular bones, and the skull. The many results have provided us with incidences of a number of pathological changes, comparative analysis of the results between different groups (age and sex) and between the prehistoric periods. This may be very useful in the planning of future studies in paleopathology of epidemiological and analytic character.

Future

Shortly, we will begin a study of epidemiological aspects which may be considered a development and continuation of the previous paleopathology study of Danish skeletons mentioned above. The title of the new study is: " A comparative analysis of methods to study nutritional and/or age dependent changes of the skeleton." The applied methods will include measurements of bone mineral content (BMC), osteon-analyses (individual age determination), radiography (proximal femur spongiosa, Harris' lines), and finally the measurement and weight of a removed bone core (osteoporosis). With regard to the dentition, the presence of enamel hypoplasia and paradontosis will be registered. The study may be divided into three steps: a test of the applied methods by the dissection of material of known age and sex, an analysis of possible correlation between the results of the different methods applied, and finally a discussion of the state of nutrition and the onset of aging in prehistoric man, based on the results of the study.

With regard to the chemical analyses, especially the BMC, a control study of possible correlation between the content of calcium in the surrounding soil and in the bones will be performed. The skeletal material forming the basis of this analysis has been carefully selected. Most of the well-preserved skeletons were recently recovered in graves from the Viking period, and most skeletons were studied in situ by the author.

Conclusions

The Danish paleopathological studies of the past, present and future together form a developmental pattern of three stages: the descriptive, the epidemiological, and the analytical. In the years of the establishment of a Danish skeletal collection the pathological studies could naturally only be of a purely descriptive character. At that time nothing more could actually be done owing to the small number of bones available, often from different locations. The lack of comparative studies has also been a very important factor. In addition, most scholars had only a restricted experience in studying human bones and especially bones several thousand years old.

Skeletal samples of a considerable size from the same excavation and site were later recovered allowing new kinds of paleopathological studies emphasizing epidemiological aspects. They were, however, still restricted to a presentation of tables of incidences of pathological skeletal and dental changes. The danger of making analytical studies and interpreting the results at that time, without having comparative studies available, is clearly seen in the conclusion stated by Hansen as a result of his study of the Norse skeletons from Greenland.

The almost contemporary work of Isager on medieval skeletons from Øm monastery has not been of much value to later studies either, but he did not draw any false conclusions.

A new type of investigation started with the epoch of the many studies by Møller-Christensen. His registration forms clearly show a new kind of development of the paleopathological aspects. Systematically and in detail he registered both the normal and the pathological parts of the bones. There are several reasons why his work on the skeletons of medieval people affected with leprosy has been so highly acknowledged on the international stage: his systematic work, his comparison with studies of modern populations, and finally his well-planned projects. As one of very few scholars of paleopathology he also succeeded in transferring his results to clinical medicine. The facial changes of the skeletons affected with leprosy turned out to be useful in clinical diagnosis of the disease.

Today almost 90% of all excavations take place without extensive planning because of the construction of new roads or buildings. The skeletal material is recovered by chance, and it is almost impossible to determine which skeletal samples should be studied next. In the days of Møller-Christensen he was able to plan his future studies even before the skeletons had been located. Real epidemiological studies allowing comparative analyses of results between different groups and periods first appeared in Denmark with some of the works of Møller-Christensen on medieval skeletal material. Recently the paleopathological study of prehistoric skeletons by the author has followed this tradition.

The development of paleopathological studies ought not, however, to stop here, but the current database should rather be considered a necessary platform for new investigations using modern methods developed for paleopathology.

A future study of prehistoric Danish skeletons has been planned mainly by the author, but many scientific specialties are included, and will result in an extensive collaboration with other scholars in this interdisciplinary project. Scholars from many different areas will be involved, mainly within the medical faculty. Other future works will probably also be marked by much more developed teamwork between scholars of different specialties, allowing more detailed methods to be presented and evaluated critically.

Studies based on interdisciplinary cooperation have already been common in archeology for several years. An increasing number of scholars, mainly of natural sciences, are involved in solving the many problems of prehistoric man's culture. In teamwork of this kind archeologists are naturally placed in the center, being those who gather the threads and synthesize the whole.

Similarly anthropologists usually have an education extending over a rather broad spectrum which may place them naturally in the center of specialists from different areas of medical, dental, paleontological, and chemical disciplines in studies dealing with problems of prehistoric man's biology. Thus, the future of paleopathology may prove to be very promising.

Literature cited

Andersen, J.G. 1969. *Studies in the Medieval Diagnosis of Leprosy in Denmark.* Copenhagen: Costers Bogtrykkeri.

Bennike, P. 1985. *Palaeopathology of Danish Skeletons. A Comparative Study of Demography, Disease and Injury.* Copenhagen: Akademisk Forlag.

Bennike, P., and K. Ebbesen. 1986. The Bog Find from Sigersdal. *Journal of Danish Archaeology,* 5:85–115.

Bennike, P., K. Ebbesen, and L. Bender Jørgensen. 1986. Two Early Neolithic Bog Skeletons from Bolkilde. *Antiquity,* 60:199–209.

Broca, P. 1867. Cas Singulier de Trépanation Chez les Incas. *Bulletins et Mémoires de la Société d'Anthropologie,* 2:403–408.

Engelhardt, C. 1877. Skeltgrave paa Sjælland og det Østlige Danmark. *Aarbøger for Nordisk Oldkyndighed og Historie,* 347–402.

Eschricht, D.F. 1837. Om Hovedskallerne og Benradene i vore gamle Gravhøje. *Dansk Folkeblad,* 3:109–116.

———. 1841. *Det kongelige Videnskabernes Selskabs Afhandlinger,* 8, del 55.

Fischer-Møller, K. 1942. The Mediaeval Norse Settlements in Greenland. Anthropological Investigations. *Meddelelser om Grønland,* 89(2). Copenhagen: Reitzels Forlag.

Fürst, C.M., and Fr. C.C. Hansen. 1915. *Crania Groenlandica. A Description of Greenland Eskimo Crania.* Copenhagen: A.F. Höst and Søn.

Hansen, Fr. C.C. 1924. Anthropologia Medico-Historica Groenlandiæ Antiquae I. Herjolfnes. *Meddelelser om Grønland,* 67:293–547.

Hansen, S. 1889. Om forhistorisk Trepanation. *Aarbøger for Nordisk Oldkyndighed og Historie,* 170–185.

———. 1913. Primitiv Trepanation. *Medicinsk Historiske Smaaskrifter,* 5. Copenhagen: Wilhelm Trydes Forlag.

Ingelmark, B.E. 1956. *De Funktionellt Anatomiska Förhållandena i Ryggraden med Särskild Hänsyn til dess Småleder.* Gothenburg, Sweden: Göteborg Universitets Årsskrift.

Isager, K. 1936. *Skeletfundene ved Øm Kloster.* Copenhagen: Levin og Munksgaard.

Kjær, H. 1912. Et mærkeligt arkæologisk-antropologisk fund fra Stenalderen. *Aarbog for Nordisk Oldkondskabs Historie,* 58–72.

Korn, J. 1982. Rehabilitering af Leprapatienter. *Ugeskrift for Læger,* 501.

Lunt, D.A. 1978. Molar Attrition in Medieval Danes. In P.M. Butler and K.A. Jowsey, eds., *Development, Function and Evolution of Teeth.* London: Academic Press.

Møller-Christensen, V. 1953. *Ten Lepers from Næstved in Denmark. A Study of Skeletons from a Medieval Danish Leper Hospital.* Copenhagen: Danish Science Press.

———. 1958. *Bogen om Æbelholt Kloster.* Copenhagen: Dansk Videnskabs Selskab.

———. 1961. *Bone Changes in Leprosy.* Copenhagen: Munksgaard.

———. 1963. Skeltfundene fra St. Jørgens Kirke i Svendborg. *Fynske Minder,* 35–69.

———. 1967. Evidence of Leprosy in Earlier Peoples. In D.R. Brothwell and A.T. Sandison, eds., *Diseases in Antiquity,* 295–307. Springfield, Ill.: Charles C Thomas.

———. 1978. *Leprosy Changes in the Skull.* Odense, Sweden: University Press.

Nielsen, H.A. 1911. Yderligere Bidrag til Danmarks Stenalderfolks Anthropologi. *Aarbøger for Nordisk Oldkyndighed og Historie,* 81–205.

Nilsson, S. 1838. *Skandinaviska Nordens Ur-Invånare.* 1st edition, 2–16.

Virchow, R. 1870. Die altnordischen Schädel zu Kopenhagen. *Archiv für Anthropologie,* 4:55–91.

SUMMARY OF AUDIENCE DISCUSSION: A Yugoslav (Serbian) physician was actually the first to describe the destructive nasal lesions of leprosy, reported in 1904 at a Berlin symposium and published in an obscure circular from that meeting. The theory attributing the fate of the southern Greenland colony of Norsemen to assimilation with Eskimos is not supported by physical evidence of dental traits or mandibular torus frequencies, but it could be evaluated further by testing the blood group pattern in the skeletal remains and comparing them with appropriate English, Danish, Norwegian, and Eskimo population samples.

Human skeletal pathology in pre-Columbian populations of northern Chile

Juan R. Munizaga

Pre-Columbian populations on the arid coast of northern Chile succeeded in surviving during thousands of years in a very inhospitable environment and under strong selection pressures. Since diseases are one of the forces through which such pressures act, we may be able to detect them by the analysis of injurious effects left in the human skeletal remains.

However, two great difficulties arise when studying the bone pathology of former populations of the Chilean arid coast. On the one hand, we know that those populations were never numerous and that most of their cemeteries have been disturbed. For these reasons it may be difficult to obtain an adequate sample. On the other hand, the bone reaction to disease is limited in variability and the types of injurious effects we found may not be identifiable with respect to etiology. Therefore, the knowledge we have of diseases in such populations is likely to remain incomplete for some time.

One way to overcome these difficulties consists in analyzing the human skeletal remains available using an ecological approach, in which we try to establish the reactive patterns of bone caused directly by environmental stimuli. For the population under study, the health level will be primarily defined on the basis of the potential bone deterioration caused by those kinds of stimuli and the age attained by the individuals surviving in that environment. From this point of view, we tried to assess the health level of populations that lived on the arid coast of Chile and from which we have only a small number of skeletal remains.

The sample and its ecological background

The specimens used in this study constitute a sample of human skeletal remains from populations which lived in a fragile, ecological balance on the arid coast of northern Chile. They were collected by L. Nuñez (1971) during his excavations at the mouth of the Loa River and neighboring coves. The sample is made up of at least 50 individuals represented by skulls and a lesser number of bones from the remaining part of the skeleton, all of which are in different degrees of conservation. Most of them belong to immediately pre-Spanish periods. A very small number come from periods of about 2000 years ago.

From a morphological point of view, the population corresponds to a Mongoloid population living on such a coast during several thousands of years, which occasionally received small migrations or visits from individuals coming from inland regions from both the Andean range of mountains and the Amazonian jungle.

Two components of the physical environment must be distinguished. On the one hand, a narrow, coastal band, bounded inland by a chain of steep but low hills and the vast Atacama Desert that isolated it, is characterized by its extreme aridity (less than 5 mm of rainfall per year) and lack of vegetation. Its climate is humid due to "evaporation from the sea blown inland by the prevailing coast winds which is turned into mist by condensation when passing over the cold Humboldt current. In winter, this mist is left on the coastline hills, thus keeping a high degree of humidity in the atmosphere" (Weiss 1951:151). Water for drinking comes from highly brackish springs or "aguadas," whose solid concentration ranges between 8.3 and 16.0 g/l versus drinking water where the maximum is 0.5 g/l (Lagos 1980:40). On the other hand, an offshore sea current is full of fish, shellfish, and mammals which, considered as food resources, are enough to support a great number of individuals.

During several thousands of years, these populations kept on improving their cultural adaptation in such a way that in the late period, from which come most of the human skeletal remains here analyzed, they had attained an efficient navigational expertise. Moreover, they mastered swimming and diving. This, added to their fishing or hunting food collection and preservation techniques, enhanced the groups' survival.

In spite of that, evidence in the archeological, ethnographic, and historical data as well as chroniclers' and travelers' statements suggest that the ecological balance was unfavorable for the pre-Columbian people who lived in small numbers in that coastal area. During the time of the Spanish contact, various epidemic diseases affected their populations, which in time disappeared.

Methods

Our purpose was to detect the greatest number of injurious effects allowed by the examination techniques applied. We oriented our examination along two main lines: (1) pathologic conditions described by other authors, considered as common in the pre-Columbian populations in America (Hrdlicka 1914; Stewart 1979:271; Weiss 1958); (2) possible effects that can be caused by pressures on a specific ecosystem. In our case, we think that the most affected apparati and systems may be the following:

Cardiorespiratory system. Pressures upon this system are exerted through the humid environment produced by the coastal morning mists and the dust raised by the wind, which affect the respiratory tract of children and adults (X. Vivanco, pers. comm.). Apnea and fast decompression during diving may cause serious health problems among fishermen of the coastal area up to the present (Bittmann 1986:54).

Locomotory apparatus. The physical effort made in swimming, rowing, and diving, as well as in fishing, hunting, and food collection and transport, placed the locomotory apparatus under almost perpetual stress. We will use the degenerative changes that may have occurred in the joint surfaces involved in these actions as indicators of its effect.

Digestive system and water and salt balance. In this case, pressures were made by the consumption of food with abrasive (sand) and toxic elements, such as mammal liver and fish rich in vitamin A (Barnicot and Datta 1956:525), in addition to the ingestion of brackish waters. The indicators of their action will be wear and the loss of teeth in addition to infectious disease and metabolic bone alterations.

The presence of disease was determined through inspection, palpation and x-rays (Harris' lines).

The degree of degeneration of joint surfaces was diagnosed by the presence of lipping, erosion, and eburnation. The first was scored according to the degree of development of osteophytes, and the last two, on the basis of the extent of the destruction produced in articular surfaces (Stewart 1969; Ortner 1968; Yamaguchi 1984).

Age determination was based on fusion of epiphysial and cranial vault sutures. For this purpose skulls were classified in the following categories: Adult 0, vault sutures open, basioccipital synchondrosis closed; Adult 1, outer suture open, inner suture in the process of closing; Adult 2, outer and inner sutures closing; Adult 3, outer and inner sutures in advanced stage of closure.

Results

I classify three groups of stress according to their frequency and to the knowledge we have of their causes: Group I, a set of signs corresponding to reaction patterns deriving from well known environmental stimuli common to the whole population; Group II, signs whose causes are known but their frequency is low; Group III, signs whose causes are unknown but that appear somewhat frequently in this population.

GROUP I

LOCOMOTOR APPARATUS AND PHYSICAL EFFORT. The effort made in swimming, rowing, diving and getting food in this environment forced the individuals of this population to stress their joints, which led first to a plastic response, which enabled them to increase their motion capacity through the creation of new joint facets or the extension of the normal ones. However, on occasion the stress was too strong, causing the progressive destruction of joint surfaces. It should be noted that the highest degree of destruction is found in the newly formed joint facets (excepting the front tibial facet) and then the facets of the elbow and knee joints. This finding is in agreement with that of other authors such as Stewart (1969), Jurmain (1977,1980), and Yamaguchi (1984). My findings are shown in Table 1.

MASTICATION APPARATUS AND FOOD. As a result of chewing hard food containing abrasive elements, a clear, reactive pattern is observed which, through wear, caries and fractures, leads to a completely edentulous status. In most cases, compensating phenomena, such as secondary dentine formation, do not compensate for the wear. Compensating mechanisms are also hampered by infection which, through the roots, causes periapical problems leading to the destruction of alveolar bone. In addition, owing to the loss of the back teeth, the front teeth are subject to greater use, so that the temporomandibular joint receives abnormal stress, sometimes giving rise to the degeneration of the joint surface. My findings are shown in Table 1.

GROUP II

Owing to the variety of features found in this group and to their low frequency, we give some details of each case. Frequencies are shown in Table 2.

Harris' lines. The average number of growth arrest lines observed per tibia is 1.1 and the percentage of affected tibiae in the population is 40.0. These values are similar to those found for pre-Columbian populations in the northernmost Chilean coast (1.5 and 35.6% respectively), but they are different from those occurring in the inland farming populations, which show a great frequency of these lines both at an individual and at a population level: 6.2 and 67.8% respectively (Allison et al. 1981:270).

Hypoplastic lines of enamel. Though we established their presence in several individuals, we could not determine their frequency due to the high rate of postmortem loss of teeth found in this population sample.

TABLE 1. Injurious effects and reactive patterns of mastication and locomotor apparatus

Group I

MASTICATION APPARATUS	Nt (%)
Dental caries	? (-)
Dental fractures	? (+)
Antemortem dental absence	35 (54.2)
Periapical abscess	35 (40.0)
Palatal abscess	35 (11.4)
Antral fistula	35 (5.7)
Tumors or cysts	35 (2.8)

LOCOMOTOR APPARATUS	Nt (%)
Third condyle	25 (44.0)
Radioulnar joint (extension)	17 (88.2)
Poirier's facet	20 (65.0)
Tibial facet	17 (100.0)

		Nt	(1)	(2)
Humerus	P	14	0.0	0.0
	D	17	23.5	52.9
Ulnae	P	18	5.5	11.1
	D	18	0.0	0.0
Radius	P	20	0.0	0.0
	D	17	0.0	0.0
Femur	P	20	0.0	0.0
	D	16	0.0	25.0
Tibia	P	17	5.8	11.7
	D	17	0.0	0.0
Fibula	P	15	6.6	6.6
	D	18	0.0	0.0

NOTE: Nt, total number cases; (%), frequency; (−), absence; (+), presence; (1), lipping (medium degree or greater); (2), incidence of erosion; P, proximal articular surfaces; D, distal articular surfaces.

TABLE 2. Injuries and skeletal lesions

Group II	*Nt (%)*
Dental hypoplastic	? (+)
Harris' lines in tibiae (av. 1.1)	15 (40.0)
Fracture	
Single	? (+)
Multiple	? (+)
Disc hernia (')	? (+)
Paraplegia (')	20 (5.0)
Alteration of lumbar column	? (+)
Periostitis	16 (31.2)
Infection	20 (5.0)
Cholesteatoma	40 (2.5)

Group III	*Nt (%)*
Alteration longitudinal superior sinus	40 (100.0)
Cribra Cranii	
Orbitalia	45 (6.6)
Parietalia (active stage)	45 (2.2)
Thickness of bones over top of cranial vaults	
8 mm and up	26 (57.6)
9 mm and up	26 (46.1)
Obelionic osteoma	40 (13.3)
Auditory osteoma	40 (12.5)

NOTE: Nt, total number of cases; (%), frequency; (+), presence; ('), belong to same skeleton.

Single fracture. Consolidated fracture of the zygomatic arch of a male adult individual. The skull shows an intentional deformation of a tabular type, indicating that it comes from an inland population.

Multiple fractures and deformations. These were seen in a male adult showing consolidated fractures in the second and third ribs; deformation and diminution in size in the clavicle of the same side; radius alteration (fracture?) with signs of inflammation; alterations in the areas of the serratus magnus and latissimus dorsi muscle insertion in the anterior face of the lower angle of both scapulae. Cause: possible fall on his shoulders during heavy load transport.

Disc hernia? An oval defect 10 mm wide and 5 mm deep was found in the lower face of the 11th dorsal vertebra body and which communicated with the vertebral canal. A similar but smaller injury was found in the upper face of the 12th dorsal vertebra body. No reactive bone formation is observed.

Paraplegia. A symmetrical alteration in the shape of femoral diaphysis was present in a male adult in the form of a slight decrease of its diameter and several symmetrical depressions of oval shape and longitudinal direction. It might correspond to a bone involution produced by a paralysis of the vastus medialis muscle, since in this individual the defect occurred between the 11th and 12th dorsal vertebrae described above and, owing to its opening to the spinal canal, it might have involved the lumbar plexus, whose upper fibers innervate that muscle.

Alterations of the lumbar column. A case of incomplete sacralization of the fifth lumbar vertebra and one of transverse apophysis separated from the third? lumbar were found. This diagnosis is based on the presence of slight joint facets. Both trauma and hereditary defect are diagnostic possibilities (Stewart 1969:448).

Periostitis. Signs such as longitudinal striations were observed in long bones and, in some cases, deposits of thin bone sheets on the outer table.

Generalized infection. This was found in a female adult whose lower limb bones appear thickened with an irregular surface.

Cholesteatoma. A globular widening of the auditory canal with a thinning of the tympanic plate (Stewart 1979:268).

GROUP III

Pathologic signs of this group are shown in Table 2.

Injuries

GROUP I

The bone reactive patterns we have described are well known and their relationship to the environment we have described are apparent. When interpreting their elements, confusion may arise concerning the boundaries existing between the normal, plastic response and the pathological response.

GROUP II

Injuries described for this group may be analyzed under three separate headings according to their most general causes.

GROWTH ALTERATIONS. Evidence arising from Harris' lines and hypoplastic lines in the enamel of this population is rather contradictory and, based on such evidence, it would be impossible to provide a clear diagnosis of poor health conditions during childhood.

ACCIDENTS. The presence of traumatic accidents caused by aggression are minimal in this population, since the only available trace of intentional blows appears in an individual whose origin must be sought in inland populations where the signs of violence appearing in the skulls range between 4 and 18% (Munizaga 1974:38). The remaining injuries falling under this heading seem to correspond to accidents during work activities. Thus, the rib fractures, disc herniation and associated paraplegia, and even the lumbar column malformations we have described may have these causes, as well as infections whose origin seems to be located in the legs. The latter can be understood if we remember that the highest risk was run by individuals of this group while sailing in rafts made of inflated hides, their legs being the most exposed parts of their bodies. An author who observed these rafts in 1780 tells us that "sometimes it happens that dolphins, sharks or other large fish puncture them and fishermen are left in a dangerous situation" (Bittmann et al. 1980:70).

ENVIRONMENTAL ACTION. We would have to analyze cholesteatoma under a separate heading. Stewart (1979:268), who is perhaps the only one who has recorded it, describes a pattern of differential distribution for Eskimos and Aleuts on the one hand, among which he has recognized 15 cases, and the more southern natives on the other hand, where he only mentions one case coming from Peru. Based on the medical evidence available for diseases of the ear in the present populations of Alaska, he poses the possibility that this pathologic condition might correspond to a cholesteatoma and that it may be related to the cold weather of the Arctic region.

The finding of a case in tropical latitudes is, therefore, hard to explain. Nevertheless, we have found two other cases showing this pathological condition in pre-Columbian individuals of coastal populations in the vicinity of the one we have described (Pisagua and Cobija). Then, considering the number of cases examined for this area, their frequency in Alaska and the arid coast of Chile is likely to be similar. In addition, if we remember that the fishermen we have described dive in the waters of the Humboldt current with its characteristic low temperatures, the causal agent may also be the same.

GROUP III

I am not certain about the causes of the injurious effects we have described in this group, but various hypotheses may be posed about their origin. I think that they may be explained by three patterns of bone reaction.

ALTERATION OF THE ENDOCRANIAL RETURN CIRCULATION. The swimming stroke used in the coastal region consists of maintaining only the head outside the water. In contrast, hunting and food collecting on land tends to direct the eyes and head downward. These head positions induce a hyperextension and hyperflexion of the occipitoatloidal joint as shown by the frequency of the third condyle and the degenerative changes in the joint surfaces of all of them. I think that these sustained and frequent movements must have hindered in a mechanical way the intracranial return circulation at the level of vessels and plexus surrounding the foramen magnum. It is also possible that, during the first years of life, an internal cause has been added: brain edema. This condition might be caused by the increase of intracranial pressure due to fluid retention produced by the ingestion of brackish water. This would alter the return circulation, as shown by the alteration of the course of the superior longitudinal sinus (formation of an endobregmatic lagoon or cistern), which would be a more apparent sign (see Table 2).

ALTERATION OF THE BLOOD CALCIUM LEVEL. Sudden decompression during diving and perhaps the ingestion of food rich in calcium may have raised the level of this element in the blood. For mechanical reasons, calcium should tend to concentrate in the blood stasis zones of the brain and produce extravasation of the emissary vessels in the passing zones, specifically in the ectocranial surface surrounding them on their way out. This might explain the bone neoformation described for the obeliac region and even the osteoma of the auditory meatus.

THICKNESS INCREASE OF CRANIAL BONES. A bone reaction pattern which led to a thickness increase in the bones was posed a long time ago for pre-Columbian populations in the arid coast of northern Chile, without an explanation for its causes (Vergara-Flores 1894, 1905; Fonck 1906). Findings in this population confirm its presence, but its frequency and intensity are lower than those established for a great number of populations in America (Munizaga 1984:40).

Various causes have been given to explain this bone reaction pattern: diet, such as vitamin C (Bourne 1956) and D deficit, hypervitaminosis A (Fonck 1906); anemia (Angel 1967; Munizaga 1965); and inheritance (Weidenreich 1943). In this case, while an exaggerated thickness in reinforcement zones is likely to be explained from a genetic point of view, the increased thickness of the vault bones may also be due to the first two already mentioned causes. It must be pointed out that we have not found the orbital and parietal sieve plates characterizing porotic hyperostosis. It might be that its appearance was modified by the alterations of the endocranial return circulation appearing in this population in infancy. The sole case we have found of sieve plates in their active phase is similar to the one described by Ortner and Putschar (1981:275, Figure 417) as an example of rickets. In this case, it would be difficult to accept the presence of such a pathologic condition.

TABLE 3. Sex and age distribution on basis of skulls

		Group I		
Ages	*Infants*	*Males*	*Females*	*Total*
A0		1	15	16
A1		5	10	15
A2		10	1	11
A3		2	0	2
A?		0	1	1
TOTAL	4	18	27	49

NOTE: A0, vault sutures open, basilar closed; A1, outer open, inner in closing process; A2, outer and inner in closing process; A3, outer and inner in advanced closing process.

Health relations to ecological balance

The results of the examination of bones belonging to these populations are surprising for two reasons: (1) low frequency of cases of massive infection, of disabled individuals or ones with advanced joint deterioration, as these frequently occur in pre-Columbian to agricultural populations; and (2) absence of old individuals and high mortality of women in early adulthood (see Table 3). Perhaps these human skeletal remains were collected in a selective manner, as has been shown to occur in other collections (Stewart 1969:444), or selective loss of skeletal remains of one age group occurred, such as when these are left on the surface (as we confirmed in a neighboring field, where the loss of children's bones was four times higher than that of adults' bones; Munizaga 1980:206). Llagostera (pers. comm.) has also observed important variations in age classes in pre-Columbian cemeteries of this zone.

In spite of the possibilities analyzed, which might explain this demographic distribution, I am inclined to think that these populations are in balance and any normal or abnormal alteration hindering the integration of an individual may be the cause of his removal from the system. This would also occur with pregnancy and might explain the mortality of young women observed. In other words, human skeletal remains of these populations in fragile balances are likely to appear rather free of pathology, since any disease may cause the death of individuals long before such disease has time enough to attack the bone system.

Conclusions

On the basis of the bones examined, this population shows a high health level, characterized by a low prevalence of chronic infections, absence of signs of violence, and a low rate of accidents. However, the following reactive patterns of bone may be distinguished: reinforcement and deterioration of joint surfaces, loss of teeth and periapical processes, alterations of the endocranial return circulation, osteomas of the ear and the obeliac region, and increased thickness in bones. There is no doubt about the validity of the first two. The others are presented as hypotheses.

However, the apparent high level of health observed in this population seems to be the result of the elimination of people who had difficulties in adjusting to the environment. This could happen even to people affected by minor and transitory disability, such as pregnancy. Perhaps those factors may explain the limited size of these populations observed in the arid northern coast of Chile by travelers visiting them in the past.

Literature cited

Allison, M., G. Focassi, and E. Gerszten. 1981. Estudio Radiográfico y Demográfico de Morbilidad y Mortalidad de Pueblos Precolombinos de Perú y Chile. *Chungara*, 8:256–274. Arica, Chile: Departamento de Antropologia, Universidad del Norte.

Angel, J.L. 1967. Porotic Hyperostosis or Osteoporosis Symmetrica. In D. Brothwell and A.T. Sandison, eds., *Diseases in Antiquity*, 378–389. Springfield, Ill.: Charles C Thomas.

Barnicot, N.A., and S.P. Datta. 1956. Vitamin A and Bone. In G.H. Bourne, ed., *The Biochemistry and Physiology of Bone*, 507–538. New York: Academic Press.

Bittmann, B. 1986. *Recursos Naturales Renovables de la Costa del Norte de Chile*, 1–66. Tokyo.

Bittmann, B., M.T. Ahumada, and N. Montenegro. 1980. El Surgimiento, Desarrollo, Decadencia y Abandono de Cobija-Lamar: Notas Históricas. In B. Bittmann, et al., *Cobija, Proyecto de Investigaciones Interdisciplinarias en la Costa Centro Sur Andina (Chile)*, 63–120. Antofagasta, Chile: Universidad del Norte.

Bourne, G.H. 1956. Vitamin C and Bone. In G.H. Bourne, ed., *The Biochemistry and Physiology of Bone*, 251–286. New York: Academic Press.

Fonck, F. 1906. Los Cráneos de Paredes Gruesas según Vergara Flores. Un Dato Más. *Revista Chilena de Historia Natural*, 10.

Hrdlicka, A. 1914. Anthropological Field Work in Peru in 1913, with Notes on the Pathology of the Ancient Peruvians. *Smithsonian Institution Miscellaneous Collections*, 61:54–61.

Jurmain, R.D. 1977. Stress and the Etiology of Osteoarthritis. *American Journal of Physical Anthropology*, 46:353–365.

———. 1980. The Pattern of the Involvement of Appendicular Degenerative Joint Disease. *American Journal of Physical Anthropology*, 53:143–150.

Lagos, R. 1980. Un Estudio Geoecológico de la Costa de Cobija: El Factor Geomorfológico y la Existencia de Agua. In B. Bittmann, et al., *Cobija, Proyecto de Investigaciones Interdisciplinarias en la Costa Centro Sur Andina (Chile)*, 31–47. Antofagasta, Chile: Univeridad del Norte.

Munizaga, J.R. 1965. Espongio Hiperostosis (Humperl y Weiss) u Osteoporosis Simétrica (Hrdlicka). Diagnóstico, Epidemiologia Antigüedad. *Revista de Antropologia*, 3(1):391–394. Universidad de Chile.

———. 1974. Paleoatologia Chilena. *Antropologia*, segunda epoca, 1:35–39. Santiago, Chile: Departamento de Ciencias Antropológicas y Arqueológicas, Universidad de Chile.

———. 1980. Restos Oseos Humanos de la Costa Norte de Chile. Análisis de Cementerios Disturbados de Cobija, II Región, Chile. In B. Bittmann, et al., *Cobija: Proyecto de Investigaciones Interdisciplinarias en la Costa Centro Sur Andina (Chile)*, vol. 1, 199–216. Antofagasta, Chile: Universidad del Norte.

———. 1984. Cráneos de Paredes Gruesas. *Revista Chilena de Antropologia*, 4:19–61. Santiago, Chile: Universidad de Chile.

Nuñez, L. 1971. Secuencia y Cambio en los Asentamientos Humanos de la Desembocadura del Rio Loa, en el Norte de Chile. *Boletin de la Universidad de Chile*, 3–25. Santiago, Chile.

Ortner, D.J. 1968. Description and Classification of Degenerative Bone Changes in the Disal Joint Surfaces of the Humerus. *American Journal of Physical Anthropology*, 28:139–156.

Ortner, D.J., and W.G.J. Putschar. 1981. Identification of Pathological Conditions in Human Skeletal Remains. *Smithsonian Contributions to Anthropology*, 28. Washington, D.C.: Smithsonian Institution Press.

Stewart, T.D. 1969. The Effects of Pathology on Skeletal Populations. *American Journal of Physical Anthropology*, 30:443–450.

———. 1979. Patterning of Skeletal Pathologies and Epidemiology. In W.S. Laughlin, ed., *First American: Origins, Affinities and Adaptations*, 257–274. New York: Wenner-Gren Foundation for Anthropological Research.

Vergara Flores, L. 1894. Cráneos de Indios Bolivianos. *Actes Societé Scientifique du Chili*, 4:231–250.

———. 1905. Cráneos de Paredes Gruesas. (Nuevas Investigaciones de 63 Cráneos de Quillagua y Algunos de la Costa). *Revista Chilena de Historia Natural*, 9:172–190.

Weidenreich, F. 1943. The Skull of Sinanthropus Pekinensis—A Comparative Study on a Primitive Hominid Skull. *Paleontologia Sinica*, new series D, 10. Pehpei, Chungking.

Weiss, P. 1951. Geografia de las Enfermedades en el Perú en Relación con las Zonas Climáticas. *Conferencias de Ciencias Antropológicas*, 140–167. Homenaje al IV Centenario de la Fundación de la Universidad Mayor de San Marcos de Lima.

———. 1958. *Osteologia Cultural*. Lima, Peru.

Yamaguchi, B. 1984. Degenerative Changes in the Distal Humerus and Femur. *Bulletin of the National Science Museum*, 10:9–17. Tokyo.

Observations on health, genetics, and culture from analysis of skeletal remains from Roonka, South Australia

Miroslav Prokopec and Graeme L. Pretty

Site and environment

The skeletal material reported here comes from the archeological site Roonka Flat on the right bank of the Murray River, 8 km north of Blanchetown in South Australia. The flat has a semicircular shape, is open to the west and bordered by lagoons on the northern and eastern sides. Steep cliffs of Tertiary deposits face the flat on the opposite side of the river. The Aboriginal tribe which occupied the area at the time of its colonization by Europeans in 1844 was known by the name Ngaiawang. Freshwater mussels, fish, marsupial game and rodents and various plant foods are assumed to have formed a large proportion of the diet of the original inhabitants. A sand dune located on an elevated place on the Roonka Flat was frequently used as a burial site. It is likely that it was used also as a camp site because of its pleasant position. Heaps of mussel shells were found at several places.

Human bones began to emerge from the soil in the early 1960s following floods and strong winds. Systematic excavations were commenced by one of us (G.L.P.) in 1968 with the support of volunteer labor and continued until 1976. Most of the excavated graves (n = 111) come from trench A, which was laid on the top of the dune, others from trenches 0A, 1A, 1B, B and from the bulks between the trenches. Several skeletons were brought to the museum prior to the beginning of systematic research.

Materials and methods

CHRONOLOGY OF INHUMATIONS

Carbon from the basal zone of trench A was dated 18,000 years B.P. (Phase Roonka I) and carbon associated with one of the older groups of skeletons was dated about 7000 years B.P. (Phase II). Intermediate group IIIa is dated approximately 4000 years B.P. and the youngest group IIIb is estimated to have terminated at circa A.D. 1850 (Pretty 1977). The site was thus used from time indefinite to the period of European contact. Questions posed to physical anthropologists were: who were the people; what did they look like; which diseases affected them and which were the most probable causes of death?

LABORATORY

The bones available for assessment in the laboratory were in a poor state. Most of the skulls had to be reconstructed and sealed together from many fragments. Most of the skeletons were incomplete.

Analysis of human osteological remains from Roonka showed that the same type of people occupied the flat for at least 7000 years and made a positive contribution to archeology by throwing light upon demography, health status, culture, way of life, and genetics of the past population. Attempts were also made to evoke the appearance of some individuals by drawing or modeling the missing soft parts on the skull.

DEMOGRAPHY

Current methods described (Martin and Saller 1957; Larnach and Freedman 1964; Hrdlicka 1947; Stewart 1952; Krogman 1962; Brothwell 1963; Trotter and Gleser 1952) were used to determine sex, age and stature from the bones. Pathological changes in the bones were reviewed with C.L. Manock and D. Pounder (pathologists), and D. Simpson, a neurosurgeon, reviewed a special case of a complete craniosynostosis in a child. Demographic aspects were studied according to Acsádi and Nemeskéri (1970) and Stloukal and Hanáková (1971).

Results

There were 165 individuals identified in Roonka, including 60 (36%) children and subadults, 58 males (35%) and 47 females (29%). The mean age of all individuals buried at

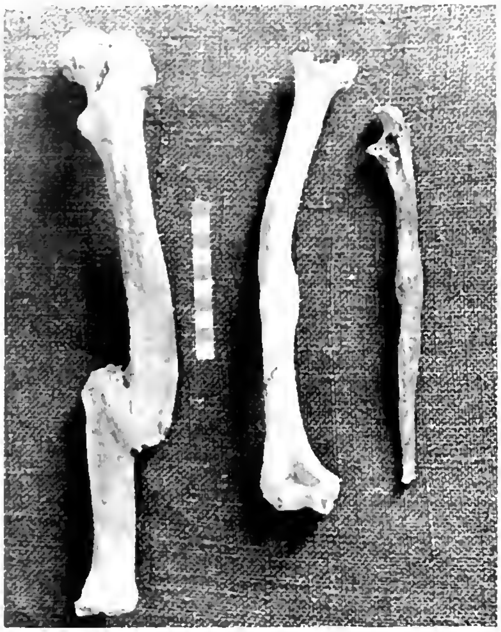


FIGURE 1. Healed fracture of femur with override and slight angulation of humerus and ulna. All photographs, with the exception of Figure 2, by M. Prokopec.

Roonka was about 24 years, of males over 20 years of age 36 years, and of females 32 years. The mean stature of males was 167.4 cm (SD 6.1 cm), and of females was 156.5 cm (SD 5.4 cm). The difference in the mean height between both sexes amounted to 10.9 cm. The greatest mortality was in early childhood and in the adult group between 20 and 30 years of age. The least represented age groups in the sample were children of approximately 10 years and those of the senile group over 60 years of age.

HEALTH STATUS

Conclusions on general health of people once living at Roonka may be drawn from the age at which people died, from stature, from size and shape of bones, and from traces of disease on teeth, jaws and bones (Smith et al. 1988). The presence of some diseases in the past population can be only assumed using a present day population, living in a similar environment, as a model.

Fractured bones and skull vaults and depressions in the skull vaults are evidence of trauma (Figures 1,2), though habits such as preventing the dead man or woman from returning to life by throwing stones into the grave may explain some traumatic changes which might have been inflicted after death.

A woman was buried at the moment of childbirth. A depression across her forehead was considered a fatal blow—the most probable reason being euthanasia (Pounder et al. 1983). Various pathological changes were found, such as an osteoma in the mandible in an old person, premature closure of all skull sutures (Simpson et al. 1983–1984; Prokopec et al. 1985) (Figure 3), arthritic changes in joints, signs of inflammatory processes on bones, an open sacral canal, and yaws or other trepanarid infections (Figures 2–7). It may be

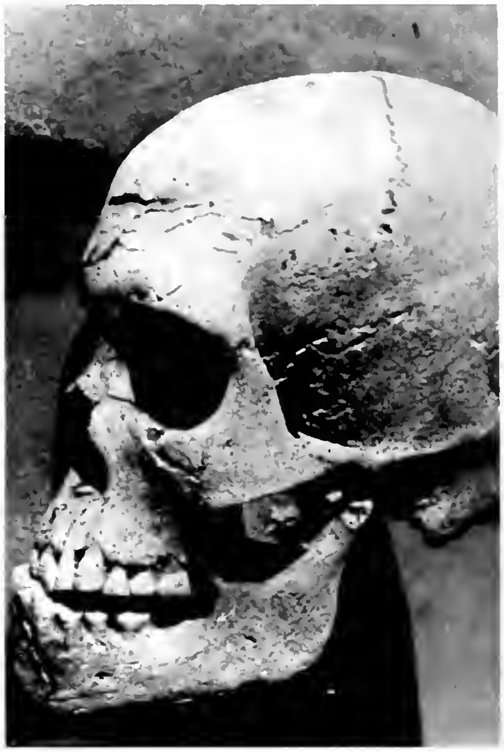


FIGURE 2. Skull of adult female, No. 110, in situ in grave from trench A, Roonka, showing depression over frontal bone following a blow by a narrow object causing fracture. Photograph by Lloyd Chilman.

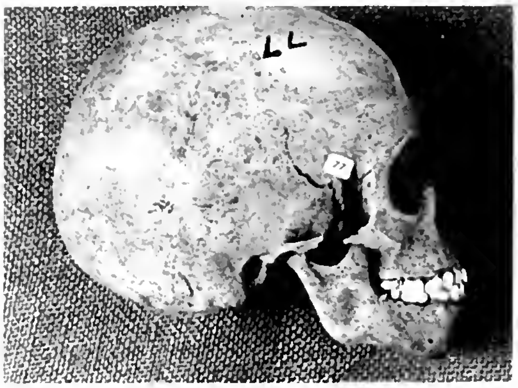


FIGURE 3. Skull of 5-year-old child with craniosynostosis and fracture of right temporal bone. Fine pitting on parietal and occipital bones. Grave No. 77, Roonka.

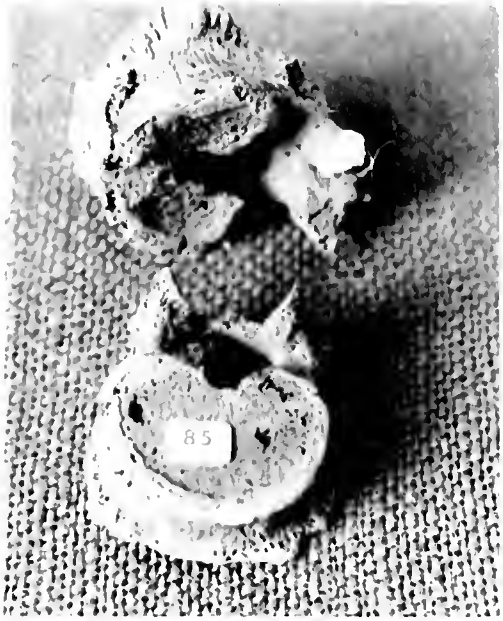


FIGURE 4. Osteophytosis especially of upper margins of upper lumbar vertebrae. Grave No. 85, Roonka.

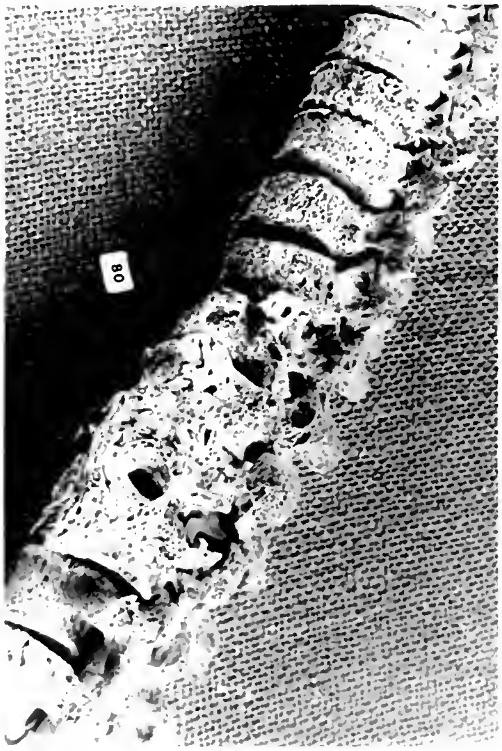


FIGURE 5. Compression fracture of body of T9 and accentuated thorax curvature. Fusion of all units through coalescence of osteophytes and apophyseal facets: T9/10, T10/11, T11/12 and T12/L1. Ankylosing spondylitis? Tomb No. 80, Roonka. Male, mature.

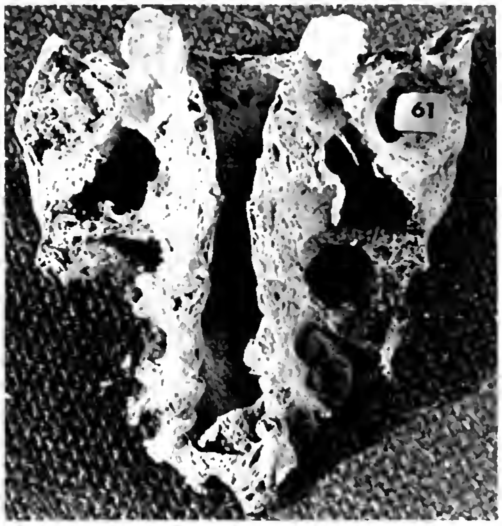


FIGURE 6. Open neural channel in sacrum. Tomb No. 61, Roonka. Male, mature.

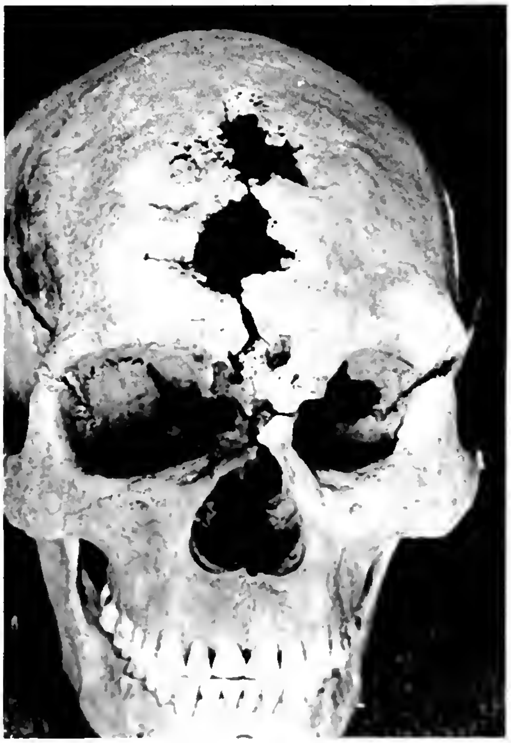


FIGURE 7. Lytic lesion with rugged edges and stellate scars on frontal bone due to treponarid infection yaws? Tomb No. 18, Roonka. Male, adult.

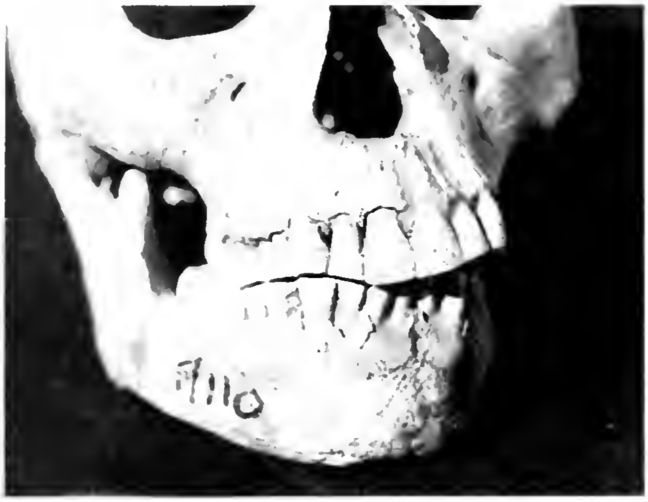


FIGURE 8. Enamel hypoplasia in upper incisors and canines and overbite in adult female. Tomb No. 110, Roonka.

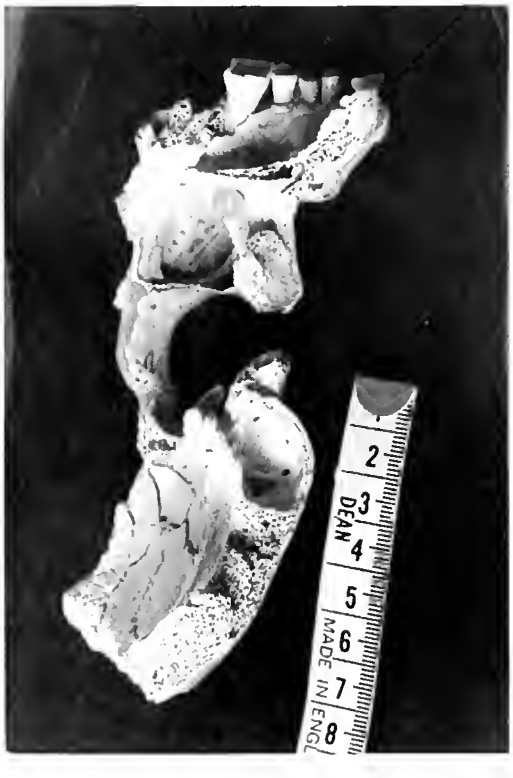


FIGURE 9. Thickening of diploe of frontal bone 14 mm thick. Tomb No. 7, Roonka. Female, adult.

said that these features, present in populations all over the world, connect the Australian Aboriginals with other populations (Rochlin 1965; Sandison 1973). Evidence of nutritional and metabolic disorders were found in some bones and teeth, such as fine pitting on the bone surface (Figure 2), enamel hypoplasia in front teeth (Figure 8), and thickening of the diploe of the frontal bone (Figure 9).

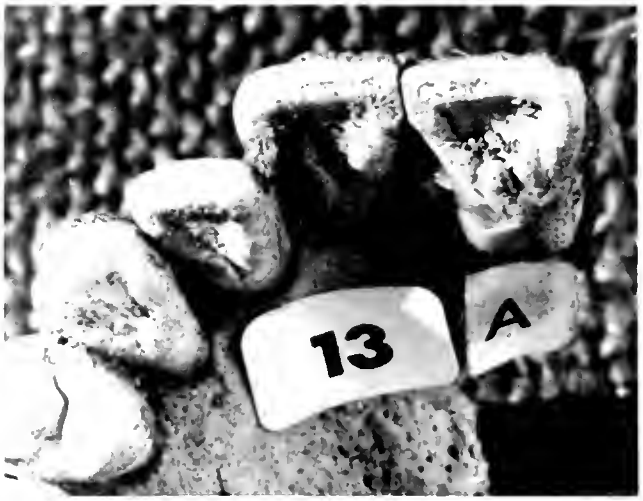


FIGURE 10. Shovel-shaped incisors and tuberculum dentale on left central upper incisor in juvenile individual male? Tomb No. 13A, Roonka.

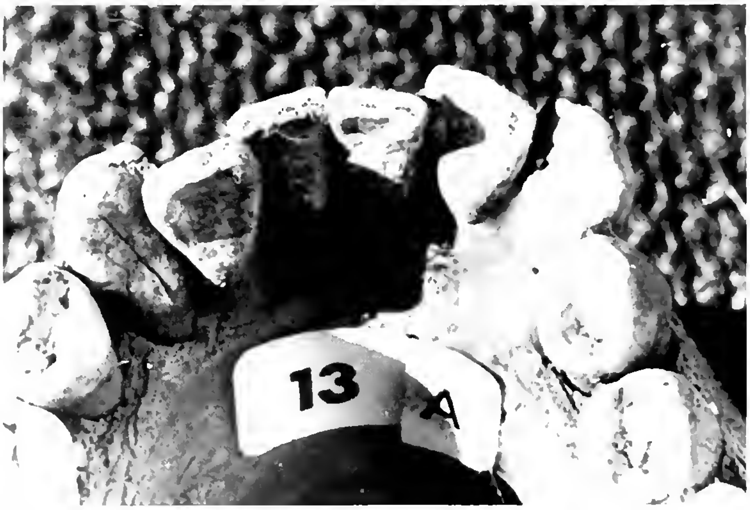


FIGURE 11. Compressed front teeth crowding in juvenile individual male? Tomb No. 13A, Roonka.

GENETICS

Inborn anomalies found in Roonka include supernumerary cusps on molars, shovel-shaped incisors, tuberculum dentale (Figure 10), protruding lower jaw (underbite), aplasia of lateral incisors, and other features. These are also found in every population in the world, sometimes perhaps with different frequencies.

Crowded teeth in lower jaws were quite frequent (28%) in the Roonka adult population (Figure 11). Heavy stress on the teeth since childhood may be blamed for this, since pressure acts in the direction from the molars toward the front teeth.

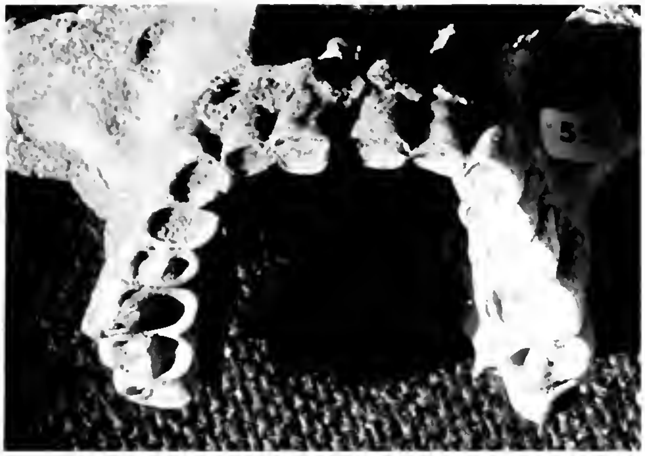


FIGURE 12. Horizontal abrasion of crowns of all teeth in maxilla with secondary dentine. Note concavities of surface of tooth crowns. Tomb No. 50, Roonka. Male, mature-senile.

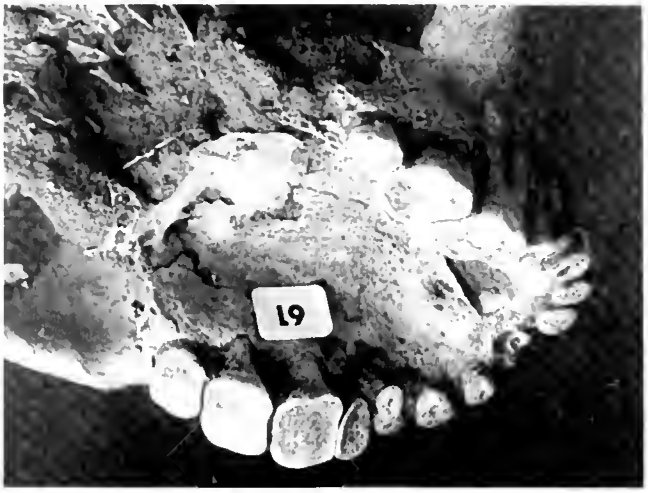


FIGURE 13. High-grade attrition of crowns of all teeth in maxilla with open root cavities of some teeth. Helicoidal form of attrition. Tomb No. 61, Roonka. Male, senile.

Genetically minded dental specialists explain it by independently inherited tooth size (broad teeth) and independently inherited mandible size (narrow mandibles). This phenomenon discovered in the Roonka population may contribute to discussions on the yet unknown origin of Aboriginals in Australia. It would mean that more than one strain of people were present in their ancestry—apparently a gracile and a robust type.

CULTURE

Culture has many facets. It is responsible for the behavior of the people, and to a certain extent it influences also their bodies. In the Roonka population the following features may be viewed as a sequence of cultural influences: deep abrasion of the crowns of the teeth (horizontal, helicoidal, interproximal) (Figures 12–15), ritual evulsion of one or two upper central incisors (Figure 16), and ear exostosis (thickening of the external auditory canal bone), explained by otolaryngologists as a reaction of the bone (periosteum) to cold temperature and frequently found in swimmers and divers (Hrdlicka 1935). It has been found in male skulls only (Figure 17).

According to Campbell (1925), the habit of ritual tooth evulsion existed only in North and Central Australia (Figure 16). Roonka revealed that this ritual was practiced also in the south of the continent for thousands of years. Skulls with evulsed central incisors were found even in the oldest graves (Campbell and Prokopec 1984).

Roonka showed that before Europeans came, the people were probably completely free from caries. This does not mean that they did not suffer from other gingival and dental diseases and discomforts. The principal problems were due to extensive abrasion of crowns. Despite the formation of secondary dentine which developed in the abraded parts of the crowns (Figure 18), deep abrasion led in some instances to penetration into the tooth cavity. As a rule bacterial infection followed and led to periapical inflammations and abscesses (Figure 19).

Food preparation over an open fire (during which ashes and sand inevitably got into the food) is probably the main reason for tooth abrasion. The habit of chewing herbs and leaves of alkaloid-containing plants mixed with ashes as well as the presumed habit of rubbing teeth of the lower jaw against those of the upper jaw in the absence of food could also hasten the process of tooth abrasion.

Another important agent which leads to dental deterioration is using teeth as tools in manufacturing weapons and other objects of daily use (Figure 15). Even stone implements were sometimes retouched by teeth according to Barrett (pers. comm.) (Prokopec 1979).

RECONSTRUCTION OF FACE FROM SKULL

A drawing or photograph of a skull may serve as a basis for two-dimensional face reconstruction using the method of Galina Lebedinskaya. In order to reconstruct the face properly, the skull should be intact and complete, in particular with respect to the preservation of the nasal bones and the anterior nasal spine (Sjøvold 1981). Any method of facial reconstruction is always associated with some inaccuracy. It is important to make allowances and adjustments for age of the person in question. A method of superprojection of a photograph of a man or woman belonging to the same population over a skull may show the probable appearance of the dead person. This is only the case when the skull and photograph match satisfactorily in a series of well-identified anthropometrical points on the skull and face and in a series of facial features and contours (Prokopec 1987). Photographs of

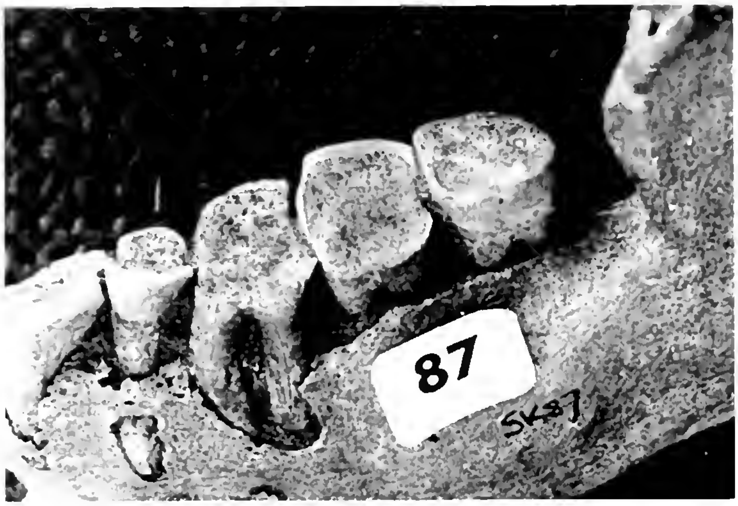


FIGURE 14. Horizontal and interproximal attrition of right M 1 and M 2 in mandible, Tomb No. 87, Roonka. Female, mature.

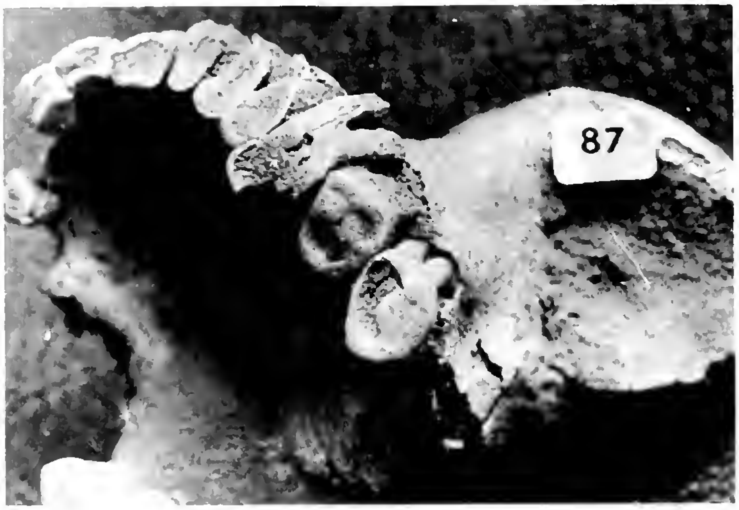


FIGURE 15. High-grade attrition with tilted left upper M 1. Buccal margin of teeth is more affected by attrition. Tomb No. 87, Roonka. Female, mature.

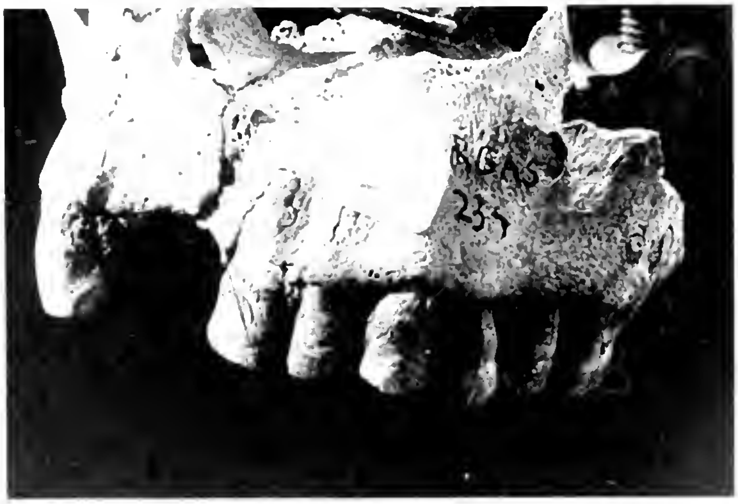


FIGURE 16. Ritual evulsion of right upper central and lateral incisors. Healed tooth sockets. High attrition in remaining teeth. Enamel hypoplasia. Alveolar bone resorption. Tomb No. 23, Roonka. Male, mature.

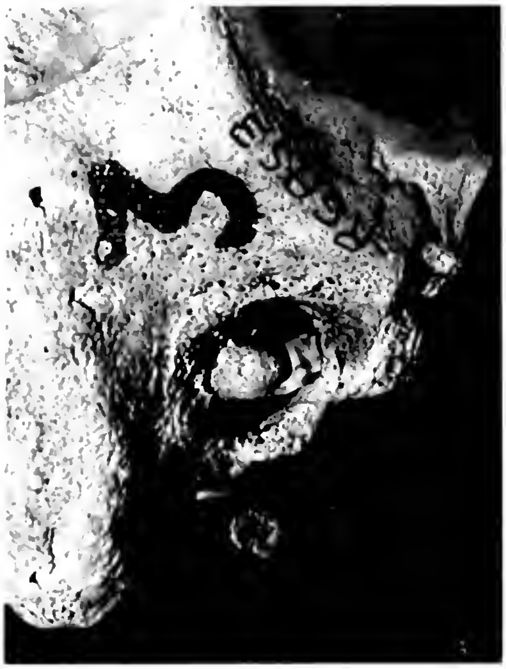


FIGURE 17. Auditory exostosis posterior. Both sides of skull affected. Tomb No. 3, Roonka. Male, senile.

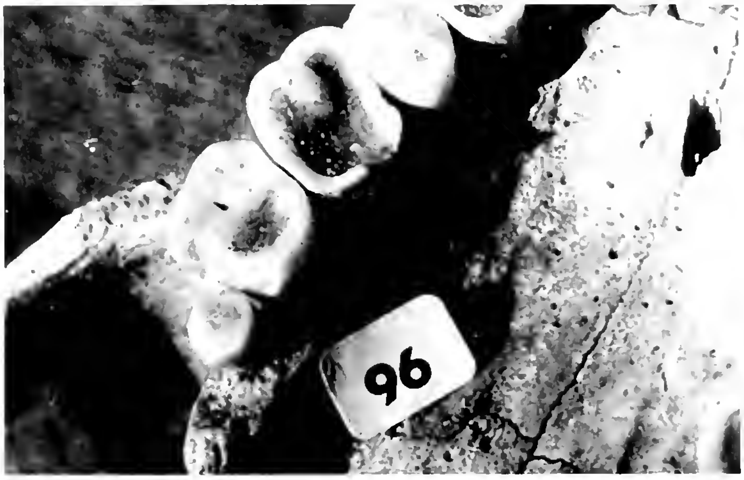


FIGURE 18. Peglike right upper third molar, secondary dentin on the crowns of M 1 and M 2. Tomb No. 96, Roonka. Female, mature.

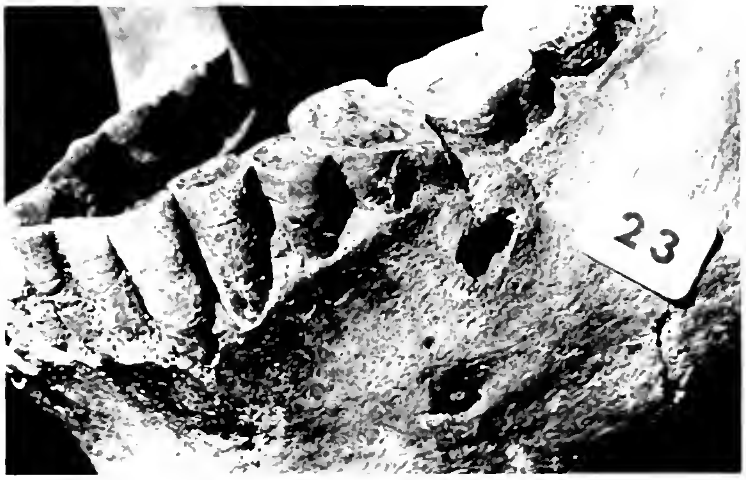


FIGURE 19. Considerable wear of tooth crowns with an open pulp cavity in first left lower molar, leading to an abscess. Tomb No. 23, Roonka. Male, mature.

Aboriginals from the Port Lincoln Aboriginal Reserve in South Australia taken in the last century were used for superprojection over some of the Roonka skulls. In one instance an oil painting of an Aboriginal chief was matched with a skull from one of the status graves from trench A from the Roonka Flat.

Conclusions

Anthropological analyses on a sample of human skeletal material from an archeological site (Roonka) in South Australia shed light on demographical structure, health status, genetics, culture, and probable way of life of the people who inhabited the site for at least 7000 years before the arrival of the first European settlers. The buried people belonged to a basically similar physical type throughout the time period.

Three different methods of face reconstruction from skulls were used in an attempt to restore the probable appearance of some of the buried persons.

Their mean stature and mean age at death are much lower in comparison to present-day standards. On the other hand, child mortality was much higher but consistent with the society of hunters and gatherers and their harsh way of life. Women and babies were always at risk at childbirth while males died frequently in fights. Diseases which leave marks on bones were present as in other societies, not only in nomads. Fine pitting in a skull of a child indicates the presence of anemia, and several instances of enamel hypoplasia bear evidence of nutritional disorders in early life. Caries was rare or absent, though deep abrasion of teeth and lack of oral hygiene caused trouble and discomfort. Contrary to literary sources the Roonka material showed that ritual evulsion of upper central front teeth was performed in South Australia for at least 7000 years.

Survival of the Roonka population under conditions similar to those faced by paleolithic hunters and gatherers for such a long time is the best proof of a functioning society fully adapted to the given environment.

Acknowledgments

Grateful acknowledgment is made to the South Australian Museum, Adelaide, to the Australian Institute for Aboriginal Studies, Canberra, and to the Institute of Hygiene and Epidemiology, Prague, for their support, and to Lloyd Chilman for the photograph in Figure 2. One of the authors (M.P.) expresses his thanks to the Smithsonian Institution, which enabled him to study human osteological material in the collections of the U.S. Museum of Natural History in Washington, D.C. Further support to G.L.P. was provided by the Australian Research Grant Scheme, Sir Mark Mitchel Foundation, Potter Foundation, Sunshine Foundation, and Utah Foundation. All specimens illustrated herein are courtesy of the South Australia Museum.

The authors are grateful to Colin Cook, Chairman, Gerard Community Council, and Valerie Power, Community Adviser, Point Macleay Community Council, for their support to the Roonka research project and interest in its results.

Literature cited

Acsádi, G., and M.J. Nemeskéri. 1970. *History of Human Life Span and Mortality.* Budapest: Akadémiai Kiadó.

Brothwell, D.R. 1963. *Digging up Bones.* London: British Museum of Natural History.

Campbell, A.H., and M. Prokopec. 1984. Antiquity of Tooth Avulsion in Australia. *Artefact,* 8:3–9.

Campbell, T.D. 1925. *Dentition and Palate of the Australian Aboriginal.* Adelaide, Australia: Hassell Press.

Hrdlicka, A. 1935. Ear Exostoses. *Smithsonian Miscellaneous Collections,* 93:1–100.

______. 1947. *Practical Anthropometry,* T.D. Stewart, ed. Philadelphia: Wistar Institute of Anatomy and Biology.

Krogman, W.M. 1962. *The Human Skeleton in Forensic Medicine.* Springfield, Ill.: Charles C Thomas.

Larnach, S.L., and L. Freedman. 1964. Sex Determination of Aboriginal Crania from Coastal New South Wales, Australia. *Records of the Australian Museum,* 26:295–308.

Martin, R., and K. Saller. 1957. *Lehrbuch der Anthropologie in systematischer Darstellung.* Stuttgart, Germany: G. Fischer Verlag.

Pounder, D.J., M. Prokopec, and G.L. Pretty. 1983. A Probable Case of Euthanasia amongst Prehistoric Aborigines at Roonka, South Australia. *Forensic Science International,* 23:99–108.

Pretty, G.L. 1977. The Culture Chronology of the Roonka Flat. A Preliminary Consideration. In R.V.S. Wright, ed., *Stone Tools as Cultural Markers: Change, Evolution and Complexity,* 228–331. Canberra: Australian Institute for Aboriginal Studies.

Prokopec, M. 1979. Demographical and Morphological Aspects of the Roonka Population. *Archaeology and Physical Anthropology in Oceania,* 14:11–26.

______. 1987. Fber die Rekonstruktion des Gesichtsausdruckes alter Slaven. *Acta Musei Nationalis Pragae.* Line B, Natural Sciences, 43:203–205. Tables 9–12.

Prokopec, M., D. Simpson, L. Morris, and G.L. Pretty. 1985. Craniosynostosis in a Prehistoric Aboriginal Skull: A Case Report. *Ossa,* 9–11:111–118.

Roehlin, D.G. 1965. *Diseases of Ancient Man.* Moscow: Nauka.

Sandison, A.T. 1973. Disease Changes in Australian Aboriginal Skeletons. *Australian Institute of Aboriginal Studies Newsletter,* 3:20–22.

Simpson, D., M. Prokopec, L. Morris, and G.L. Pretty. 1983–1984. Prehistoric Craniostenosis: A Case Report. *Records of the Adelaide Children's Hospital,* 3:163–168.

Sjøvold, T. 1981. Arpas Anatomical Method for Face Reconstruction. *Ossa,* 7:203–204.

Smith, P., M. Prokopec, and G.L. Pretty. 1988. Dentition of a Prehistoric Population from Roonka Flat, South Australia. *Archaeology in Oceania,* 23:31–36.

Stewart, T.D. 1952. *Hrdlicka's Practical Anthropometry.* Philadelphia: Wistar Institute of Anatomy and Biology.

Stloukal, M., and H. Hanáková. 1971. Anthropology of an Early Mediaeval Burial Site in Abraham. *Acta Musei Nationalis,* 27B:3.

Trotter, M., and G.C. Gleser. 1952. Estimation of Stature from Long Bones of American Whites and Negroes. *American Journal of Physical Anthropology,* 10:463–514.

SUMMARY OF AUDIENCE DISCUSSION: The tooth crowding demonstrated is interesting because Dr. Corruccini's work in India suggests a decreased frequency of tooth crowding in populations consuming a diet requiring vigorous chewing, the frequency rising later following the introduction of a softer diet. No such pattern, however, was demonstrable in this population. The two cranii revealing changes suggestive of treponematosis failed to demonstrate periostitis of the tibia or other long bones.

Tuberculosis

Tuberculosis in the Americas: Current perspectives

Jane E. Buikstra and Sloan Williams

> Histologic studies of the hydrated lungs showed a large amount of fibrous tissue in the right apex. Dr. Garcia-Frias concluded the combination of spine and lung disease showed that tuberculosis is the most likely diagnosis, and the present writer agrees, although other conditions cannot be ruled out.
>
> —Morse 1961:497

> The case presented herein should conclusively end this dispute and remove doubt that tuberculosis did exist in the Department of Ica in southern Peru, South America, hundreds of years before the coming of any European to the Americas.
>
> —Allison et al. 1973:985

Rehydrated soft tissue from South American mummies has proved crucial in the ongoing debate concerning the presence of a "tuberculosis-like" pathology in the prehistoric Americas. In the 1961 review cited above, Morse, while skeptical of North American skeletal examples attributed to tuberculosis, was willing to accept tuberculosis as "the most likely diagnosis" for mummified Peruvian remains reported by Garcia-Frias in 1940. Even though he found the tissue evidence convincing, Morse (1961:497) was led to question the archeological context for these materials and thus concluded that a pre-Columbian attribution was not secure.

Further investigations of mummified soft tissue, reported by Allison and co-workers in 1973 and 1981, provided convincing histologic *and* contextual data. The 1973 report described acid-fast bacilli, Pott's disease, and a psoas abscess in the remains of a Nazca child, dating to approximately A.D. 700, and thus established with certainty the presence of a tuberculosis-like pathology in the prehistoric Americas. Even Morse was led to alter his stance, remarking that although he generally agreed with Allison and co-workers concerning the diagnosis, "there should have been many more cases of suspect tuberculosis than have been found to date" (1978:53).

Recent studies of pre-Columbian tuberculosis, as indicated in Table 1, are rapidly providing the "many more cases" called for by Morse. Most examples cited in this survey are descriptive reports of skeletal lesions from North American series, including isolated cases as well as profiles from larger samples. Both isolated examples and frequency data are important in establishing the probable antiquity and distribution of the pathology, although for issues relating to community health and quality of life, the large-scale series are most useful. In South America, Allison and co-workers (1981, 1984) have documented additional cases from Peru and Chile. Surveys of large Andean skeletal samples to establish lesion form and distribution in a manner suitable for comparison with North American data sets have, however, yet to occur. The ultimate goal of this paper is, therefore, to establish lesion patterning within a large prehistoric Peruvian skeletal sample and thus provide a data base for comparison with North American examples.

Related topics addressed within the past few years include the persistent question of origins. The argument that tuberculosis could only have developed in the context of domestic animals as hosts (Cockburn 1963) has proved unconvincing in the North American example. The possibility that atypical mycobacteria should be implicated has been raised (Clark et al. 1987; Eisenberg 1986; Klepinger 1982) and will be discussed in detail below.

A novel approach, recently applied to the study of prehistoric tuberculosis, is the development of mathematical models for the spread of disease. Although this strategy has a lengthy history within the medical sciences (Grigg 1958; Waaler et al. 1962), it has been developed only recently within paleoepidemiology (McGrath 1986, 1988; Milner 1980). As this form of investigation holds promise for establishing expectations and comparabilities across time and space, it also will be reviewed here.

TABLE 1. Recently reported cases of skeletal tuberculosis from North America

Site	*Location*	*Period*	*Pathology*	*Reference*
Moundville	Alabama	Miss. A.D. 1050-1550	Vert: M 25-29.9, 2F 30-39.9; ribs: 2 juv, 8 Y-M adults (13/564)[a]	Powell 1988
Irene Md.	Georgia, north of Savannah	Miss. A.D. 1100-1400	2 juv, 4 y ad, 4 40-50 yr F:M 2:1 (10/265)[a]	Powell 1989
Parkin	Cross Co., AR	Late Miss.	F 35-40 yr, F 17-25 yr (2/16)	Murray 1985
Daw's Is.	Beaufort Co., SC	3300-3700 B.C.	M 30-35 yr	Rathbun et al. 1980
Turpin site	SW Ohio	A.D. 1125-1425 Ft. Ancient	F 20-25 yr, 2M 35+ yr, 2F 16-18 yr, F adult (5/291)	Katzenberg 1977; Perzigian and Widmer 1979; Widmer and Perzigian 1981
Arnold	Cumberland, TN	~A.D. 1200	F y adult	Widmer and Perzigian 1981
Averbuch	Nashville, TN	A.D. 1275-1400	M:F 2:1	Eisenberg 1986; Kelley and Eisenberg 1987
Norris Farm #36	Cent. Illinois	Oneota ~A.D. 1300	(32/264)	Milner et al. 1988
Kane Mounds	Am. Bottom	Miss. A.D. 1150-1250	F y ad, F m ad	Milner 1982
Ball Site	Lake Ontario; Iroquois ossuary	A.D. 1590-1600	±11 yr (1/6)	Melbye 1983
Uxbridge (ossuary)	Ontario	A.D. 1490±80	1 3-5 yr, 2 6-16 yr, 5 17-25 yr, 18 ad	Pfeiffer 1984
Woodlawn site	SE Saskatchewan	A.D. 1080±139	F ±45 yr	Walker 1983
Jamestown Mounds	N Dakota	A.D. 930±70	M 35-45 yr	Williams and Snortland-Coles 1986
Pueblo Bonito	NW New Mexico	A.D. 828-1130	±9 yr	Morse 1969; El-Najjar 1979; Ortner and Putschar 1981
Chavez Pass	N Arizona	A.D. 900-1100	2M 20-30 yr, F 25-35 yr(?) M 40-50 yr (?)	El-Najjar 1979
AZ-J-549	NE Arizona	A.D. 875-975	F 16-18 yr	Sumner 1985
Point of Pines	SW Arizona	A.D. 1285-1450	F y ad	Micozzi and Kelley 1985
Near La7602	Tocito, NM	A.D. 900-1300	4-5 yr	Fink 1985

a. Figures include only those individuals with significant numbers of observable ribs and/or vertebrae (Powell pers. comm.).

Modeling expectations for prehistory

McGrath (1986,1988) has developed a simulation approach to modeling expectations for the spread of tuberculosis within prehistoric communities. She chose as a basis for her analyses a diachronic sequence of three paleopopulations from west-central Illinois: Middle Woodland (150 B.C.–A.D. 400), Late Woodland/Emergent Mississippian (A.D. 400–1050), and Mississippian (A.D. 1050–1150). This study area was selected for its abundant archeological data, as well as the fact that late prehistoric skeletal samples (Buikstra 1977; Buikstra and Cook 1978,1981) show evidence of tuberculosis-like pathology. Estimates of regional population aggregation and disease transmissibility are based upon current archeological wisdom concerning population size and settlement distributions. As emphasized by McGrath, the goal was to demonstrate the value of simulation for paleoepidemiologic study with the expectation that additional investigations of regional prehistory will necessitate redefinition of basic parameters (McGrath 1986,1988).

McGrath's stochastic adaptation of the Reed-Frost model generates epidemic disease curves based upon specified assumptions concerning the behavior of tuberculosis in recent human groups. Communities were modeled either as small, stable units arranged linearly along the Illinois River (Middle and Late Woodland) or as scattered farmsteads (Mississippian). Regular interaction occurred between neighboring communities only, the Late Woodland contact pattern including more groups than the Middle Woodland example. Twice a year Middle Woodland communities converged on a local "ceremonial center"; Mississippian groups traveled twice a year to Cahokia, a large urban complex to the south. McGrath's estimated population parameters are presented in Table 2 (after McGrath 1988:489). Two levels of regional population numbers and settlement size are developed for each model.

TABLE 2. McGrath's (1988:489) population models

Run	*Pop. size*[a]	*Pop. density*	*Settlement size*	*Effective pop.*
MW1	4635	1.60	35	1540
MW2	1345	0.46	10	440
LW1	15855	5.37	120	1080[b] /480[c]
LW2	2655	0.71	20	180[b] /80[c]
M1, 2	2168	0.71	30	45045, 12045
M3, 4	1088	0.35	15	44022, 11022

a. Base population size is used to generate settlement size. Settlement size is multiplied by number of sites, then number of introduced cases of tuberculosis is added to get final population size. Middle and Late Woodland models have 15 introduced cases; Mississippian models have 8 introduced cases.

b. Effective population size for upland communities.

c. Effective population size for valley communities.

McGrath's simulation tests the spread of tuberculosis within the hypothetical region over a 100-year period. Age-specific mortality rates are developed based on contemporary expectations for tuberculosis and a life table constructed from archeological data. Disease prevalence, the infectious proportion of the population, and mortality patterning are modeled. In all cases, with the exception of the second Late Woodland (LW2) model, the simulated populations experience severe, drastic disease stress and become extinct within the 100-year period. In the LW2 example, the pathogen becomes extinct. Again, with the exception of LW2, all groups show evidence of high disease prevalence—100% for Mississippian and 30–40% for the first Late Woodland and both Middle Woodland models.

McGrath (1988:494) concludes that the key variable influencing the fate of these simulated populations is effective population size:

> Effective population size appears to be more important than all the other factors that influence disease occurrence. In other words, regardless of group size, number of neighbors, population age structure, or regional population size it appears to be effective population size that determines the course of the epidemic. Regional population size and group size seem to affect the *speed* with which the disease is spread and population declines, but effective population size determines *whether* decline occurs at all.

McGrath further concludes that "a critical value of effective population size that permits both the host population and the pathogenic organism to survive" exists somewhere in the range between 180 and 440 individuals (McGrath 1988:494). This statement holds implications for paleodemographic reconstructions in situations where an ancient tuberculosis-like disease is documented. Her work also underscores the importance of social and cultural factors that influence population interaction in disease transmission.

Tuberculosis-like lesions in the Mississippian skeletal series from west-central Illinois have, however, been amply documented (Buikstra 1977; Buikstra and Cook 1978,1981). Obviously either McGrath's model is misspecified or the disease entity reflected in the osseous record was not behaving in the same manner as modern tuberculosis caused by *Mycobacterium tuberculosis*. Changing temporal and geographic expressions of disease caused by *M. tuberculosis* have, however, been described (e.g., Dubos 1965; Grigg

1958). We therefore may simply be documenting a flexible host-parasite relationship. An alternative suggestion is the possibility that the prehistoric tuberculosis-like pathology described in Illinois and elsewhere in the Americas was caused by a pathogen other than *M. tuberculosis* (Buikstra 1981; Clark et al. 1987; Eisenberg 1986; Klepinger 1982; McGrath 1986,1988).

Atypical/environmental mycobacteria

Recently, Clark and co-workers (1987) have underscored the importance of considering mycobacteria other than *M. tuberculosis* when assessing the impact and origins of the tuberculosis-like lesions of prehistoric tissues in the Americas.

> What is known about the ecology of mycobacterial disease raises the possibilities that pre-Columbian "tuberculosis" was caused (1) by *M. tuberculosis* but in a population immunized by exposure to environmental ("atypical") mycobacteria; (2) by *M. tuberculosis* but a strain of low virulence; (3) by *M. bovis* transmitted by wild animals (e.g., butchering and tanning skins of infected bison), with infection resulting in self-limiting disease and long-lasting immunity; and/or (4) by one or more of the environmental mycobacterial species. (Clark et al. 1987:51)

In general, individuals who commented on this manuscript commended the authors for emphasizing the dynamic nature of host-parasite relationships. The possibility that the environmental mycobacteria are heavily implicated in late prehistoric populations from the Americas is, however, questioned. Katzenberg (1987:52) and Klepinger (1987:52), for instance, emphasize that the prevalence of observed tuberculosis-like pathology reported for late prehistoric populations in North America is consistent with an infectious disease spread by host-to-host transmission. The environmental pathogens are rarely, if ever, transferred between humans (Lincoln and Gilbert 1972; Sommers 1979; Wolinsky 1979).

Steinbock (1987:56) points out that the calcified Ghon complexes reported by Allison and co-workers (1981) for two prehistoric and one colonial period South American remains are simply not consistent with disease caused by environmental mycobacteria. Even though the environmental mycobacteria could have produced the acid-fast reaction noted by Allison and co-workers (1973), he considers the calcified Ghon complex specific to tuberculosis.

Kelley and Eisenberg have suggested that the skeletal lesions produced by the environmental mycobacteria are "essentially identical" to those produced by *M. tuberculosis* (1987:94). A similar argument is offered by Clark et al. (1987:48–49). This generalization would appear, however, open to debate. In fact, the sources cited by Kelley and Eisenberg in support of this statement are either silent (Good 1980) or rather ambiguous concerning the form and distribution of skeletal lesions. Wolinsky (1974:645) notes that "several cases of *M. kansasii* disease of the bones and joints are on record" but does not provide a comparison of the skeletal lesions characteristic of tuberculosis and those caused by the environmental pathogens. In more recent work, Wolinsky (1979) reviews a number of cases of both disseminated and localized infections caused by the atypical mycobacteria. In so doing, he emphasizes occupational trauma as a major factor influencing the distribution of skeletal lesions. Deep hand infection is emphasized, with fewer cases reported for the wrist, hip, knee, spine, and calcaneum. This review indicates that while the form taken by specific skeletal lesions may resemble tuberculosis, neither lesion location nor age-specific patterning mirrors that expected for tuberculosis (Wolinsky 1979).

In fact, when case studies are reviewed, it appears that both age-specific patterning and intraindividual lesion distributions for environmental mycobacterial infection differ from tuberculosis (Ellis 1974; Halla et al. 1979; Halpern and Nagel 1978; Lakhanpal et al. 1980). Vertebrae may be affected, but there is no convincing evidence of a tuberculosis-like predilection. Lakhanpal and co-workers, for instance, emphasize that the clinical, radiologic, and histologic appearance of the lesions caused by *M. kansasii* is distinctive from that of tuberculosis. "It seems as if the basic pathology of lesions caused by these organisms is different from the one caused by *Mycobacterium tuberculosis*" (Lakhanpal et al. 1980:473).

Lincoln and Gilbert (1972:697) point out that "disease of only one area of the skeletal system was reported infrequently" in their survey of children suffering from disease caused by acid-fast bacilli other than *M. tuberculosis* and *M. bovis*. They conclude that the disseminated mycobacterioses attributed to the atypical forms most closely resemble a "malignant type of reticuloendotheliosis" (Lincoln and Gilbert 1972:708), not tuberculosis.

For individual lesions, however, bony involvement in disease caused by environmental mycobacteria could mimic expectations for osseous tuberculosis (e.g., a 14-year-old male with lumbar vertebral and sacroiliac involvement; Ellis 1974). Even so, it is difficult to disagree with Steinbock's comment: "It is inconceivable that one of these forms [of environmental mycobacteria] could be the pathogen for pre-Columbian tuberculosis" (1987:56).

The geographic distribution and balanced gender ratios observed in North American prehistoric samples would also argue against acquisition through butchering or other occupations that would place an individual at risk for environmental mycobacteria, as Clark et al. (1987) have argued. Thus, although it certainly is possible that these pathogens may have caused a few of the lesions reported in prehistoric series and they may likewise have influenced the expression of disease in certain individuals, it is unlikely that they can be seriously implicated in the vast majority of prehistoric examples.

The distribution and prevalence of the tuberculosis-like pathology recognized in the Americas during pre-Columbian times may, however, have been influenced by the environmental mycobacteria. In this light, it is instructive to note that when the larger skeletal series represented in Table 1 are considered, relatively low population frequencies for tuberculosis occur in the southeastern United States, at Irene Mound and Moundville. Recent laboratory surveys of isolate distribution and frequency for the nontuberculous mycobacteria describe the Southeast as having high isolate frequencies for *M. avium, M. fortuitum, M. kansasii,* and *M. scrofulaceum* (Falkinham et al. 1980; Good 1980; Good and Snider 1982). In reaction to the Clark et al. (1987) argument, Powell has implicated cross-immunity due to the presence of environmental mycobacteria to explain the "low visibility" of tuberculosis at Moundville (Powell 1988:180). Although incomplete archeological recovery may be cited in the Moundville example, the possibility that the distribution of atypical mycobacteria may have influenced the prevalence of prehistoric tuberculosis-like pathology should be considered in studies of ancient disease in the New World.

Clark et al. (1987) call attention to the presence of less virulent strains of tuberculosis that occur in India ("south Indian variant"), in other parts of south and southeast Asia, and in Africa. Such geographic variability underscores the flexibility of the pathogen-host relationship and is a topic worth considering further. It would be of interest, for instance, to discover the degree to which osseous lesions develop in these nonvirulent, pervasive forms. Certainly extensive skeletal involvement has been reported for victims of tuberculosis in India (Ganguli 1963). It would also be appropriate to implement McGrath's simulation approach using variables specified according to the parameters common to the less virulent forms.

North American model

The recent case studies of the North American tuberculosis-like pathology reported in Table 1 expand the earlier summary presented by Buikstra (1977:326). Although certain examples fit expectations more tightly than others, it appears that there is now sound evidence for disease in eastern United States population centers such as the Cumberland Basin, the central Mississippi valley, and southern Ontario. Typical skeletal expressions of tuberculosis-like pathology are described in multiple individuals from several sites, including Norris Farm #36, Schild Mississippian Cemetery, Moundville, Averbuch, and in the Ontario ossuaries reported by Hartney (1981) and Pfeiffer (1984). With the exception of an isolated lesion in a thoracic vertebra from the Serpent Mounds (Anderson 1968), a single case from an Archaic shell mound (Rathbun et al. 1980), and an Illinois example from uncertain context reported by Morse (1969:502) and Ortner and Putschar (1981:173), these skeletal series postdate A.D. 1000. Although uneven sex ratios occur in certain contexts, both males and females, as well as all age groups, are represented. In the Schild and the Averbuch cases, the classic Pott's disease occurs in young adults. Healed cases, as at Irene Mound (Powell 1990), tend to be found in older individuals; disease active at the time of death is associated with juveniles and younger adults.

Plains and the Southwest examples have increased in recent years. Although the sample size is small, they nevertheless suggest a slightly earlier presence than in the eastern examples. Only one, however, clearly predates A.D. 1000: the Kayenta adolescent reported by Sumner (1985).

The frequency data for the North American skeletal series is somewhat enigmatic when viewed from the perspective of modern clinical samples. When the larger, best preserved, and well-excavated series reported in Table 1 and in Buikstra (1977) are considered, percentage values range from the Moundville (5.2%), Irene Mound (5.7%) and Schild Cemetery (6.7%) figures to 12.1% for the Norris Farm #36 site. (The Averbuch frequency may also be as high as 6.1%, but this is difficult to interpret since Kelley and Eisenberg (1987) apparently include individuals with periosteal remodeling in long bones in the absence of other pathognomic lesions.) The oft-cited figures of 5–7% bony involvement in modern hospital samples (Steinbock 1976:175,1987:55), even tempered by Kelley and Micozzi's (1984) observations in the Hamann-Todd collection, suggest that virtually every member of these late prehistoric communities had primary exposure to tuberculosis, as suggested by Klepinger (1987:52). In situations of extreme social and biological stress, such as that reported by Milner et al. (1988) for the Norris Farm #36 population, prevalence of chronic destructive disease appears to have been extreme.

South America

Allison and co-workers (1973; 1981) have reported a series of 12 remains from Peru and Chile that they consider to be expressions of tuberculosis. Of these, three (1981, cases 4,5 and 9) could be postcontact. The diagnosis of the cranial granuloma in case 11 (Allison et al. 1981:51) is sufficiently problematic that it will also be excluded from discussion here. Of the remaining eight prehistoric examples, four show skeletal involvement, three without confirming soft tissue pathology. The remaining four are diagnosed based upon healed Ghon complexes (cases 2 and 8) and cavitary pulmonary disease in association with acid-fast bacilli (cases 6 and 7).

The examples with skeletal involvement include one juvenile 8–10 years of age, two males: one listed as 41+ years of age and another "adult," and an isolated female 40 years old. Those diagnosed through soft tissue include the 8–10-year-old juvenile with Pott's disease, a 14-year-old female, a male adult, and two females: 50 and 56+ years old. Thus, there are two juveniles and four middle–old adults. Individuals dying within the young adult years are conspicuously absent

TABLE 3. Age distributions for tubercular and nontubercular remains from the Estuquina site

Age range	*No. in total sample (%)*	*No. in group with >5 vertebrae (%)*	*No. in affected sample (%)*
b-9.9 y	151.00 (36)	90.00 (39)	5.00 (14)
10-19.9 y	59.00 (14)	36.25 (16)	3.00 (8)
20-29.9 y	63.00 (15)	36.75 (16)	71.50 (31)
30-39.9 y	45.25 (11)	21.00 (9)	4.20 (11)
40-49.9 y	45.50 (11)	23.00 (10)	4.90 (13)
50+ y	50.25 (12)	26.00 (11)	8.40 (23)
	414.00	233.00	37.00

TABLE 4. Age distributions for tubercular and nontubercular remains from the Estuquina site

Age Range	*No. in total sample (%)*	*No. with lesions other than primary rib involvement (%)*	*No. with primary rib involvement (%)*[a]
b-9.9 y	151.00 (36)	3.00 (16.7)	2.00 (10.5)
10-19.9 y	59.00 (14)	1.00 (5.6)	2.00 (10.5)
20-29.9 y	63.00 (15)	7.00 (38.9)	4.50 (23.7)
30-39.9 y	45.25 (11)	3.00 (16.7)	1.20 (6.3)
40-49.9 y	45.50 (11)	1.00 (5.6)	3.90 (20.5)
50+ y	50.25 (12)	3.00 (16.7)	5.40 (28.4)
	414.00	18.00	19.00

a. One example of periosteal reaction on internal aspect of sternum (M6-3269a) included here.

from this sample, thus contrasting with the pattern presented by the larger North American series.

An isolated case of diffuse Pott's disease dated to 160 B.C. is the earliest example, followed by three adults from the Caserones site, dated to A.D. 290. These four examples are 1500 to more than 2000 years old, much more ancient than Kelley and Micozzi imply in their discussion of the same material: ". . . these cases are all within about the last 1,000 years" (Kelley and Micozzi 1984:385). Clearly, the evidence from South America suggests an earlier manifestation of the disease, though with a somewhat different age-specific expression than that found in North America.

Estuquina series

The Estuquina site dates to approximately A.D. 1350 and is situated in the Moquegua valley at approximately 1000 m above mean sea level (Rice 1989). Located near the present-day town of Moquegua, in southern Peru, the site is situated on a ridge top and is fortified by stone walls. Three separate cemetery areas have been identified, with burials also occurring within domestic structures. Samples of both intact and disturbed tombs have been excavated, resulting in 414 individuals from 244 tombs. Tomb contents and cemetery organization have been described in detail by Williams et al. (1989).

The demographic structure of the sample is presented in Table 3, along with the distribution of individuals showing skeletal evidence of tuberculosis-like pathology. For comparative purposes, we also indicate the numbers of individuals with five or more observable thoracic and lumbar vertebrae. Individuals presenting lesions are reported in detail in Appendix 1. Table 4 differentiates those individuals with rib involvement from those with other tuberculosis-like lesions. In the latter group, vertebral involvement is typical for adults, with accompanying sacroiliac and paravertebral rib lesions in extreme cases. Also included are juveniles that present periostitic remodeling of the ilia in a manner similar to that reported for the Schild series (Buikstra and Cook 1978, 1981).

The list of individuals with rib lesions (Table 4) includes both those cases which present only periosteal remodeling and those displaying primarily destructive foci. Resorptive rib lesions are the form commonly referenced in the medical literature (Rechtman 1929; Johnson and Rothstein 1952; Tatelman and Drouillard 1953), while periosteal reaction is emphasized by Kelley and Micozzi (1984) in their discussion of the Hamann-Todd series.

Kelley and Micozzi (1984) argue that subtle periostitis on the internal aspects of ribs may be associated with pulmonary tuberculosis. They cite data from individuals in the Hamann-Todd collection who were diagnosed as either suffering from pulmonary tuberculosis or "tuberculosis." Most of their cases present periosteal reaction only, although in a few instances, more extensive, sharply circumscribed foci are reported. Kelley and Micozzi argue that these lesions develop as a result of extension from the lungs and pleura, citing clinical sources in support of this assertion. "That tubercle bacilli are capable of extending from the lung or pleura into the ribs has been well documented clinically (e.g. Rechtman 1929; Johnson and Rothstein 1952; Jaffe 1972; and Anderson 1976)."

In fact, the two case studies in this reference list very clearly describe tubercular rib lesions which are attributed to hematogenous spread, *not* extension from the lung or pleura. The Rechtman (1929) article describes an infant whose 7th rib involvement developed after an earlier focus within the synovium of the knee. Hematogenous spread to the rib is posited. Johnson and Rothstein report three cases of rib involvement and conclude, "While the exact pathogenesis of these lesions is not certain, they are probably hematogenous in origin, differing in no way from tuberculous involvement of other bones. There is no evidence in any of our cases of extension from adjacent organs" (1952:880). Although Jaffe (1972:1002) does state that proximity and extension explain rib lesions, it is clear that the two primary sources do not so argue. It should also be noted that examples of extension from paravertebral abscesses *into* the lung are commonly reported, as are abscesses on the external aspect of the thorax (Hodgson et al. 1969; Yau and Hodgson 1968; Jaffe 1972; Johnson and Rothstein 1952; Rechtman 1929).

Most clinical experience with rib tuberculosis follows the pattern described by Tatelman and Drouillard (1953). These authors report four types of tuberculosis of the rib: (1) costovertebral, (2) costochondral, (3) isolated body lesion, and (4) multiple cystic foci. In each case, destructive processes constitute the primary symptom, with the majority of the lesions involving the body. These resorptive foci follow the typical pattern for bone tuberculosis and are clearly different from the mild periostitic response described by Kelley and Micozzi (1984). Certainly, the features described by Kelley and Micozzi may indeed reflect tuberculosis. Persons engaged in paleoepidemiological study should, however, also be aware of the patterns typically taken by the resorptive foci reported in the medical literature.

Although Kelley and Micozzi (1984:386) correctly indicate that tuberculosis is the most commonly observed *inflammatory* condition in the ribs, a differential diagnosis of rib lesions attributable to tuberculosis must consider other forms of pathology. For instance, Tatelman and Drouillard report that "tuberculosis is second only to metastatic malignancy as a cause of destructive lesions of the ribs" (1953:923). Thus, it is clear that metastatic processes must be considered in developing a differential diagnosis of tuberculosis-like lesions in ribs.

In discussing differential diagnosis, Kelley and Micozzi (1984:386) state: "Conditions to be considered in the differential diagnosis of skeletal tuberculosis are actinomycosis, typhoid, pyogenic osteomyelitis and syphilis (Sinoff and Segal 1975)." Sinoff and Segal's article, entitled "Tuberculous osteomyelitis of the ribs: a case report," however, makes it clear that their primary concern is with manifestations in the rib, rather than "skeletal tuberculosis" in general, as suggested by Kelley and Micozzi. The full quotation from Sinoff and Segal reads: "The single most important differential diagnosis (in rib tuberculosis) is metastatic carcinoma, but other possible diseases include: (i) actinomycosis, (ii) typhoid or paratyphoid osteomyelitis, (iii) pyogenic osteomyelitis and (iv) syphilis" (1975:866). Thus, Sinoff and Segal are focusing upon tuberculous manifestations of the rib and emphasizing metastatic carcinoma.

Kelley and Micozzi (1984:386) follow their listing of diseases appropriate for differential diagnosis in "skeletal tuberculosis"—actinomycosis, typhoid, pyogenic osteomyelitis, and syphilis—with the statement, "However, none of these conditions commonly affects the ribs." This conclusion is remarkable since Sinoff and Segal (1975) are clearly concentrating upon forms of disease *affecting the rib*. And in fact, one of the conditions most likely to produce just the type of proliferative response described by Kelley and Micozzi is actinomycosis. Tatelman and Drouilard (1953:932) describe rib involvement in actinomycosis through direct extension from lung and pleural involvement, emphasizing productive changes due to this condition. A classic description derives from Edeiken (1981:792): "When the thoracic wall is affected, the ribs may show destruction, although they usually react with periosteal new bone formation and become thick . . . which, although not diagnostic of actinomycosis, is most suggestive." Clearly, actinomycosis and perhaps other related disease processes must be considered when periostitic reaction is observed on ancient ribs. Thus, while Kelley and Micozzi may be correct in attributing the mild periostitis observed in the Hamann-Todd series to tuberculosis, it is clear that conditions other than tuberculosis can produce periosteal reaction on ribs.

The age profiles for the total Estuquina skeletal series and the subsample with more than five observable thoracic/lumbar vertebrae (Table 3) do not differ significantly (Kolmogorov-Smirnov test, $D_{m,n} = 0.06$; $D_{crit} = 0.111$; $p > .05$). For the purposes of further testing, the affected individuals are compared with the vertebrally observable subsample. The two subgroups, rib and nonrib, indicated in Table 4 do not differ significantly in age structure ($D_{m,n} = 92$; $D_{crit} = 142$; $p > .05$). The total affected sample does, however, differ significantly in mortality experience from the larger Estuquina skeletal series ($D_{m,n} = 0.33$; $D_{crit} = 0.29$; $p < .01$). This difference is influenced primarily by the elevated numbers of affected individuals in the young adult and elderly adult years. The former include active cases, while the latter comprise the more extreme and healed examples. Low visibility and perhaps low prevalence of bony involvement likely affect the figures for young juveniles.

These results indicate that young adults are overrepresented in the affected sample, as they were not in the series reported by Allison et al. (1981). This apparent elevation of young adult morbidity, whether the full series or only that with more than five thoracic or lumbar vertebrae present is used for comparison, is expected for a tuberculosis-like pathology. The pattern is characteristic of the larger North American samples reported in Table 1.

An unusual feature of the Estuquina series is, however, an apparent skewed sex ratio. Although males are overrepresented in the full sample (99 males, 71 females), the affected adult sample presents a decidedly more extreme bias (19 males, 7 females). When the tubercular sample is subdivided by area of involvement (Table 4), we find rib lesions in 8 males and 4 females, while among the remainder, 12 males and only 3 females are affected. Two of the three females with classic Pott's disease are older individuals with extreme degeneration of the spine (M6-1021a, M6-5390). A chi-square comparison indicates that sex ratio of affected individuals is significantly different from the total sample ($\chi^2 = 4.346$, $p = .037$). Partitioning the affected group by lesion location demonstrates that the individuals presenting rib involvement are not so biased toward males as those presenting classic Pott's disease. The comparison for those with rib lesions yields a χ^2 of 0.377 ($p = .539$). The value for the more "classic" examples is 3.204 ($p < .073$).

Summary and conclusions

Recent years have seen several new and productive approaches to issues relating to the tuberculosis-like pathology present among prehistoric human remains in the Americas. The simulation modeling developed by McGrath suggests that a pathogen behaving much like modern *M. tuberculosis* could not have been maintained within certain North American prehistoric groups. In the face of skeletal evidence documenting the presence of the disease in west-central Illinois, we must consider the possibility that the pathogen responsible was not *M. tuberculosis* and that we are documenting a host pathogen relationship not common in recent history. In this case, McGrath's model based upon disease experience in modern human groups would have been misspecified.

Clark et al. (1987) have postulated an important role for the "environmental mycobacteria" in explaining the expression of apparent tubercular disease. While we agree that in isolated cases, the possibility that skeletal lesions resulting from the atypical mycobacteria may mimic those caused by *M. tuberculosis*, it is unlikely that the environmental pathogens were responsible for many of the tuberculosis-like lesions described for late prehistoric series. Certainly, as at Moundville, it is possible that disease prevalence was influenced by the presence of environmental mycobacteria. Further investigations of geographic distributions for the mycobacteria and prevalence figures for ancient disease may, therefore, prove instructive.

Finally, we have described patterning for a tuberculosis-like pathology in a large series of skeletons from southern Peru. Although prior studies of Andean remains had described an age-specific lesion pattern different from that reported in North America, the Estuquina site series shows strong similarity to their northern contemporaries. The strong sex bias in the sample, however, remains problematic.

In closing we would like to take issue with Clark et al.'s (1987:58) assertion that "from the anthropological perspective, differential diagnosis is unproductive, at least when adaptation and evolution are the primary foci." We *must* engage in differential diagnosis, if we are to represent fully and accurately the health status of prior human groups. Otherwise we risk confusing the degenerative results of occupational stress with the products of trauma, the treponematoses with fungal infections, the presence of widespread, epidemic disease with the impact of environmental pathogens, and the impact of nutritional imbalance with the results of infectious processes. Without concern for differentiating disease forms, we will at best obfuscate and at worst misrepresent the course of human history. Differential diagnosis is no more an empty exercise than theorizing without considering contradictory empirical evidence. The study of human adaptation in the past requires scientific methodology, including both theory and data-based tests. As illustrated by the current controversies surrounding prehistoric "tuberculosis" in the Americas, differential diagnosis plays a crucial role in this process.

Acknowledgments

The research reported here was funded by the National Science Foundation (BNS87-17590), the Center for Latin American Studies, and the Lichtstern Fund of the Anthropology Department at the University of Chicago. The content and organization of the document has benefited immeasurably from the criticism of Mary Powell and Lisa Leuschner.

Appendix. Individuals with tuberculosis-like pathology from Estuquina site

*M6-181: Sex unknown, 25–30 years.*Resorptive areas on internal aspect of ribs.

M6-336a: Male 39+ years. At time of death remodeling was active in lumbar region. L1 only slightly affected, with resorption on inferior and lateral aspects of body. Much of this area does *not* show sclerotic emargination. Superior aspect of L4 has a ca. 5-mm cavitation with sclerotic emargination that appears to have recently "broken through" to surface. Disk spaces in lumbar region and at L5–S1 have been crossed by process. Anterior aspect of L2–S1 bodies also present periosteal proliferation.

M6-771: Child 2–3.25 years. Slight amount of light, pinpoint cribra orbitalia is observable. Porotic hyperostosis appears on occipital, near lambdoid suture. All deciduous teeth show dark staining on buccal aspect near CEJ. Linear enamel hypoplasia present in all incisors.

Limb long bones show porosity, especially at metaphyses, as well as remodeling in areas of tendon/ligament attachment. Both ilia present unusual porosity on external surfaces. Sternum very porous, especially the manubrium.

Ribs show sclerotic raised areas on internal aspect of bodies of right ribs 9–12, esp. 11. It is this pathology that has drawn M6-771 into the affected group, along with periosteal remodeling on external aspects of ilia. Remodeling of limb long bones is subtle, not fulminating onion-skin expansion noted in others from the Estuquina sample.

Well-integrated remodeled diaphyseal bone and dental pigmentation suggest long-standing health problems for this individual.

M6-1002: Male 17–19 years. 7th thoracic unit shows a multilocular resorptive area, without much obvious sclerotic emargination.

M6-1021a: Female 45+. Right scapula and humerus have configuration that could be the result of a tubercular process. If so, onset occurred before individual was fully (skeletally) mature since the disease seems to have caused resorption and dislocation of humerus head, which is fused to glenoid fossa of scapula. Glenoid fossa surrounding this area shows focal lesions ca. 5–7 mm in diameter, with sclerotic emargination.

Spinal column heavily involved. There is an oval cavity on C6, 1.1 × 0.5 cm in diameter, into which C5 fits. Epiphyseal ring of C6 has formed a buttressing osteophyte, again suggesting long-term pathology. This strut was sufficiently efficient that there was little apparent loss of effective body height.

T3: superior surface of body has collapsed into an internal lesion, which displays sclerotic emargination.

T4 shows a small focal resorption on right side of body, with epiphyseal ring gone. Body height loss, leading to scoliosis, apparent.

T5/T6: circumferential porosity and remodeling on both bodies.

T7: erosive circumferential lesions, mostly small (ca. 2–3 mm) foci. Slightly larger resorptive areas occur within neural canal, extending onto pedicles. This is the only pedicle involvement observed in M6-1021a.

T8 shows a central lesion with the main opening inferiorly directed, although a superior perforation also occurs. Multilocular sclerotic emargination.

T9–T10 fused through bodies only, with disk space gone. Erosive circumferential remodeling on each body.

T11: circumferential erosion present with one resorptive focus at midline.

T12 relatively intact.

L1–L4 ankylosed kyphosed mass, fused through neural arches. Body of L3 entirely gone, with a few residual osteophytes. A lesion was observed in body of L1, in addition to circumferential erosion under anterior longitudinal ligament. L1–L2 fused through arch only, as are L3–L4. L4 has massive erosive area under anterior longitudinal ligament.

L5 fused to sacrum. Extensive zygapophysial arthritis is probably a secondary result of disease process.

M6-1025: 5.5–7.5 months. Main involvement is in right hip. Erosion of acetabulum with onion-skin proliferation extending over all of internal and external surfaces. Right femur shows heavy remodeling in area of greater trochanter that extends to linea aspera. Unfortunately, this region is poorly preserved.

M6-1183a: Male 35–39 years. L5 and S1 are affected. L5 shows evidence of an active disease process eroding inferior surface of body. Sclerotic reaction clearly present, with bony reactive processes active at time of death. Trabeculae coarsened. Neural arches, i.e., zygapophysis, affected by osteoarthritic change secondary to disease. S1 presents a complementary lesion pattern.

M6-1183b: Male 50+. Remodeling observed on internal aspects of ribs. Surface well integrated; costal groove obliterated.

M6-1215: 12–15 years. Remodeling evident at sternal ends of ribs.

M6-1223: 5–7 months. An infant with fiberbone periostitis beginning to develop on internal aspects of both ilia, r > l. Long bones show evidence of periosteal remodeling. Both internal and external surfaces of cranial vault are slightly porous.

M6-1557: Sex unknown, adult. Periostitis observed on internal aspect of ribs.

M6-1610a: Male 45–50 years. Periostitis observed on internal aspect of ribs.

M6-1616a: Male 35–45 years. Internal surface of ribs shows evidence of integrated periosteal reaction.

M6-2256a: Male 30–40 years. 9th and 10th thoracic elements are affected. This may be an osteomyelitic process since no other vertebrae are affected, but will include it as a possible example of tuberculosis.

T9: much of body destroyed by disease process. Superficial new bone formation is present. Only approximately 2/3 of superior surface is intact, as is right side. Superficial remodeling present on external aspect of vertebral body. Sclerotic reaction of trabeculae bone has smoothed interior "surface," although two oval reaction area remnants remain.

T10: body height maintained on left side. Sclerotic emargination of oval lesions evident in body.

Kyphosis due to complementary destruction of these two vertebrae could have led to an acute angulation of as much as 30°.

M6-2279b: Male 20–21 years. L5 (only lumbar vertebra fully observable) shows extensive remodeling on superior surface of body and multiloculated lesions. Body height reduced. Lesions extend into pedicles. Other lumbar fragments suggest no additional units affected.

M6-2297a: Male 27–30 years. Involvement of T9–T11 which present multiloculated erosive areas. No loss of vertebral body height, no arch involvement. Destructive foci extending across disk space apparent at levels of T9–T10, T10–T11. Sclerotic emargination of resorptive regions is characteristic.

M6-2330: 1–2 years. Inferior surfaces of centra of two lumbar vertebrae present extensive remodeling and multiple small, coalesced resorptive areas. In addition, sternal extremity of three left ribs present expansive periosteal remodeling.

M6-3198a: Male 20–21 years. L5 (only lumbar vertebra fully observable) shows extensive remodeling on superior surface of body. Multiloculated lesions apparent, and extend into pedicles. Body height diminished. Other lumbar fragments suggest no additional units affected.

M6-3205: Male 27–30 years. Periostitis observed in internal aspect of ribs.

M6-3215: Male 25–30 years. Heavily remodeled spinal column and lower portion of body. Long bones porous, with some periosteal reaction. Weight-bearing joint surfaces somewhat porous. Tarsals in general are light in weight, and appear expanded and porous. Metatarsal bodies show periostitic reaction. Auricular surface of right innominate heavily eroded, with some sclerotic emargination. Remnants of oval lesions evident.

Circumferential remodeling evident on T3–T11. T4 shows some porosity that may be post mortem. Part of inferior body has collapsed into internal lesion. Sclerotic emargination is obvious.

T7: anterior portion of body eroded and absent.

T8: left side of body eroded and absent. Focal lesions evident, as is sclerotic emargination.

T9: 1.8 × 1.3 cm resorptive space present on right side of body. This feature leads into internal, smaller focal resorption. Body has collapsed with little proliferative reaction.

Ribs: at least three left and five right show expansion on internal aspect of neck. Proliferative reaction has added up to 2 mm of new bone. Both sclerotic and active fiberbone reaction is evident from mid to lower thorax. These bodies are expanded internally such that costal grooves are indistinct.

M6-3248a: Female 25–26 years. Evidence of remodeled periostitis on internal aspect of ribs.

M6-3269a: Sex unknown, adult. Periosteal reaction obvious on internal aspect of manubrium.

M6-3299: Male 18–19 years. Periostitis observed on internal aspect of ribs and on anterior surface of L3.

M6-3644a: Female 25–26 years. Remains were recovered from a collar tomb that contained a minimum number of five individuals, including three adults. Two adjacent (T9/T10) lower thoracic vertebrae exhibit massive resorptive lesions within the bodies. On superior surface of T9, three coalesced large foci are visible. Body height maintained on right side, but destroyed on left. Only anterior and right side of T10 body present, showing circumferential bone proliferation. Erosive lesions have destroyed interior portion of body. Inferior aspect had apparently fused to T11 (not recovered). Right postzygapophysis and inferior surface of a 2d or 3d lumbar vertebra from this set of commingled remains also show extensive resorption.

M6-4165: Male 27–30 years. Solitary lesion apparent on left side of L4 body, ca. 2 cm in diameter, that perforates to left side. Modest amount of proliferative bone around lesions. Osteophytes extend to L3. L3 not affected.

M6-4176: Male 22–24 years. Right sacroiliac articulation heavily eroded by disease process, which is somewhat obscured by postmortem change. A lesion on articular surface has penetrated external surface. Periosteal reaction evident on opposite external face. Reaction evident on proximal portion of right femoral diaphysis.

M6-4213a: Female 50+ years. Ribs present internal expansion of bone well integrated at time of death. No costal groove observable.

M6-4256: Male 45+ years. T10–T11 affected by erosive process that has produced extensive loss of body height on inferior aspect of T10 and superior aspect of T11. A kyphosis of ca. 30° was produced. Sclerotic emargination evident on all lesions. Pedicles affected on both vertebrae.

At least five left and two right ribs show periosteal reaction on their vertebral aspects, extending across bodies past angles. Costal grooves obscured. In that these are lower ribs, the reaction is probably related to degenerative process observed in vertebrae.

M6-4268: Female 22–24 years. Periostitis observed on internal aspect of ribs.

M6-5390: Female 50+ years. This individual shows characteristic and extensive skeletal changes.

Right ilium ankylosed to sacrum.

Vertebral involvement extensive. Initial focus of disease process was in bodies of T10–T12, with subsequent extension to L1–T9, kyphosis (ca. 90°) and ankylosis. Lesions occur adjacent to disks in L5 and T9–T12. L2, T3, T4, T5, and T6 present oval resorptive areas posteriorly, adjacent to foramina for basivertebral veins. Circumferential resorption occurs in T3–T8 and L3. The most extensive neural arch involvement is in L2, with pedicles affected in T4–T7, T9–T12, and L3. Transverse processes show pathological resorptive areas in T5–T7.

Bones of feet somewhat porous, with notable periostitis on lateral aspect of calcanei. There are two sets of fused foot phalanges.

Most ribs, nine left, eight right, have arthritic articular facets in addition to periostitis located on their necks. Most inferior ribs are remodeled externally.

M6-5428: Male 50+ years. Healed periosteal reaction evident on internal aspect of ribs.

M6-5838a: Female 50+ years. Periostitis on internal surface of rib.

M6-5859: Male 35+ years. Periostitis noted on internal aspects of ribs.

M6-6464: Male 35–39 years. L1 and L3 both have resorptive lesions, with anterior surface of L3 and posterior surface of L1 being affected. Right acetabulum and caput femoris present trabecular exposure following premortem cortical resorption. Left scapula also has a large channel extended vertically through glenoid fossa.

M6-8210a: Sex unknown, middle-old adult. Healed periosteal reaction on internal aspect of ribs.

M6-9334: Male 39–44 years. Small resorptive area on superior surface of rib.

M6-99407b: 2.5–3.0 years. Ribs expanded, as are long bones and ilia. Involvement of ribs emphasized.

Literature cited

Allison, M.J. 1984. Paleopathology in Peruvian and Chilean Populations. In M.N. Cohen and G.J. Armelagos, eds., *Paleopathology at the Origins of Agriculture*, 515–529. New York: Academic Press.

Allison, M.J., E. Gerszten, J. Munizaga, C.A. Santoro, and D. Mendoza. 1981. Tuberculosis in Pre-Columbian Andean Populations. In J.E. Buikstra, ed., Prehistoric Tuberculosis in the Americas. *Northwestern University Archeological Program Scientific Papers*, 5:49–51. Evanston, Ill.: Northwestern University.

Allison, M.J., D. Mendoza, and A. Pezzia. 1973. Documentation of a Case of Tuberculosis in Pre-Columbian America. *American Review of Respiratory Diseases*, 107:985–991.

Anderson, J.E. 1968. The Serpent Mounds Site Physical Anthropology. *Royal Ontario Museum, Art Archaeology Occasional Paper*, 11.

Anderson, J.R., ed. 1976. *Muir's Textbook of Pathology*, 10th edition. Yearbook Med. Pubs., Inc., 810–849.

Buikstra, J.E. 1977. Differential Diagnosis: An Epidemiological Model. *Yearbook of Physical Anthropology 1976*, 20:316–328.

Buikstra, J.E. 1981. Introduction. In J.E. Buikstra, ed. Prehistoric Tuberculosis in the Americas. *Northwestern University Archeological Program Scientific Papers*, 5:1–23. Evanston, Ill.: Northwestern University.

Buikstra, J.E., and D.C. Cook. 1978. Pre-Columbian Tuberculosis: An Epidemiological Approach. *Medical College of Virginia Quarterly*, 14:32–44.

______. 1981. Pre-Columbian Tuberculosis in West-Central Illinois: Prehistoric Disease in Biocultural Perspective. In J.E. Buikstra, ed., Prehistoric Tuberculosis in the Americas. *Northwestern University Archeological Program Scientific Papers*, 5:115–139. Evanston, Ill.: Northwestern University.

Clark, G.A., M.A. Kelley, J.M. Grange, and C. Hill. 1987. The Evolution of Mycobacterial Disease in Human Populations: A Reevaluation. *Current Anthropology*, 28:45–62.

Cockburn, T.A. 1963. *The Evolution and Eradication of Infectious Diseases*. Baltimore: Johns Hopkins University Press. Dubos, R. 1965. *Man Adapting*. New Haven, Conn.: Yale University Press.

Edeiken, J. 1981. *Roentgen Diagnosis of Diseases of Bone*. 3d edition. Baltimore: Williams and Wilkins.

Eisenberg, L.E. 1986. Adaptation in a "Marginal" Mississippian Population from Middle Tennessee: Biocultural Insights from Paleopathology. Ph.D. dissertation, Department of Anthropology, New York University. University Microfilms, Ann Arbor.

Ellis, W. 1974. Multiple Bone Lesions Caused by Avian-Battey Mycobacteria. *Journal of Bone and Joint Surgery*, 56B:323–326.

El-Najjar, M.Y. 1979. Human Treponematosis and Tuberculosis: Evidence from the New World. *American Journal of Physical Anthropology*, 51:599–618.

Falkinham, J.O., B.C. Parker, and H. Gruft. 1980. Epidemiology of Infection by Nontuberculous Mycobacteria. *American Review of Respiratory Diseases*, 121:931–937.

Fink, T.M. 1985. Tuberculosis and Anemia in a Pueblo II–III (ca. A.D. 900–1300) Anasazi Child from New Mexico. In C.F. Merbs and R.J. Miller, eds., *Health and Disease in the Prehistoric Southwest*, 359–379. Tempe: Arizona State University.

Ganguli, P.K. 1963. *Radiology of Bone and Joint Tuberculosis with Special Reference to Tropical Countries*. New York: Asia Publishing House.

Garcia-Frias, J.E. 1940. La Tuberculosis en los Antiquos Peruanos. *Actualidad Med. Purana*, 5:274.

Good, R.C. 1980. Isolation of Nontuberculous Mycobacteria in the United States. *Journal of Infectious Diseases*, 142:779–783.

Good, R.C., and D.E. Snider. 1982. Isolation of Nontuberculous Mycobacteria in the United States, 1980. *Journal of Infectious Diseases*, 146:829–833.

Grigg, E.R.N. 1958. The Arcana of Tuberculosis. *American Review of Tuberculosis and Pulmonary Disease*, 78:151–172.

Halla, J.T., J.S. Gould, and J.G. Hardin. 1979. Chronic Tenosynovial Hand Infection. In *Mycobacterium Terra, Arthritis and Rheumatism*, 22:1386–1390.

Halpern A.A., and D.A. Nagel. 1978. *Mycobacterium fortuitum* Infections. *Clinical Orthopedics and Related Research*, 136:248–252.

Hartney, P.G. 1981. Tuberculosis Lesions in a Prehistoric Population Sample from Southern Ontario. In J.E. Buikstra, ed., Prehistoric Tuberculosis in the Americas. *Northwestern University Archeological Program Scientific Papers*, 5:141–160. Evanston, Ill.: Northwestern University.

Hodgson, A.R., W. Wong, and A. Yau. 1969. *X-ray Appearances of Tuberculosis of the Spine*. Springfield, Ill.: Charles C Thomas.

Jaffe, H.L. 1972. *Metabolic, Degenerative, and Inflammatory Diseases of Bones and Joints*. Philadelphia: Lea and Febiger.

Johnson, J., and E. Rothstein. 1952. Tuberculosis of the Rib. *The Journal of Bone and Joint Surgery*, 34A:878–882.

Katzenberg, M.A. 1977. An Investigation of Spinal Disease in a Midwest Aboriginal Population. *Yearbook of Physical Anthropology*, 20:349–355.

______. 1987. Comment in G.A. Clark et al., 51–52.

Kelley, M.A., and L.E. Eisenberg. 1987. Blastomycosis and Tuberculosis in Early American Indians: A Biocultural View. *Midcontinental Journal of Archaeology*, 12:89–116.

Kelley, M.A., and M.S. Micozzi. 1984. Rib Lesions and Chronic Pulmonary Tuberculosis. *American Journal of Physical Anthropology*, 65:381–386.

Klepinger, L.L. 1982. Tuberculosis in the New World: More Possibilities, Probabilities, and Predictions. *American Journal of Physical Anthropology*, 57:203.

______. 1987. Comment in G.A. Clark et al., 52–53.

Lakhanpal U.P., H. Singh, and P.C. Sen. 1980. *Mycobacterium kansasii* and Osteoarticular Lesions. *Acta Orthopedica Scandinavica*, 51:471–473.

Lincoln, E.M., and L.A. Gilbert. 1972. Disease in Children due to Mycobacteria other than *Mycobacterium tuberculosis*. *American Review of Respiratory Disease*, 105:683–714.

McGrath, J.W. 1986. A Computer Simulation of the Spread of Tuberculosis in Prehistoric Populations of the Lower Illinois River Valley. Ph.D. dissertation, Northwestern University, Evanston, Ill.

______. 1988. Social Networks of Disease Spread in the Lower Illinois Valley: A Simulation Approach. *American Journal of Physical Anthroplogy*, 105:683–714.

Melbye, J. 1983. The People of the Ball Site. *Ontario Archaeology*, 40:15–36.

Micozzi, M.S., and M.A. Kelley. 1985. Evidence for Pre-Columbian Tuberculosis at the Point of Pines Site, Arizona: Skeletal Pathology in the Sacroiliac Region. In C.F. Merbs and R.J. Miller, eds., Health and Disease in the Prehistoric Southwest. *Anthropological Research Papers*, 34:347–358. Tempe: Arizona State University.

Milner, G.R. 1980. Epidemic Disease in the Post-Contact Southeast: A Reprisal. *Midcontinental Journal of Archeology*, 5:39–56.

______. 1982. Measuring Prehistoric Levels of Health: A Study of Mississippian Period Skeletal Remains from the American Bottom, Illinois. Ph.D. dissertation, Northwestern University, Evanston, Ill.

Milner, G.R., V.G. Smith, and E. Anderson. 1988. Conflict, Mortality, and Community Health in an Illinois Oneota Population. Revision of a paper presented at the conference entitled Between Bands and States: Sedentism, Subsistence, and Interaction in Small-Scale Societies. Center for Archaeological Investigations, Southern Illinois University, Carbondale, Ill.

Morse, D. 1961. Prehistoric Tuberculosis in America. *American Review of Respiratory Disease*, 83:489–504.

______. 1969. American Disease in the Midwest. 1st edition. *Illinois State Museum, Report of Investigations*, 15.

______. 1978. Ancient Disease in the Midwest. 2d edition. *Illinois State Museum, Report of Investigations*, 15.

Murray, K.A. 1985. Bioarcheology of the Parkin Site. Honor thesis on file at J. William Fulbright College of Arts and Sciences, University of Arkansas.

Ortner, D.J., and W.G.J. Putschar. 1981. Identification of Pathological Conditions in Human Skeletal Remains. *Smithsonian Contributions to Anthropology*, 28. Washington, D.C.: Smithsonian Institution Press.

Perzigian, A.J., and L. Widmer. 1979. Evidence for Tuberculosis in a Prehistoric Population. *Journal of the American Medical Association*, 241:2643–2646.

Pfeiffer, S. 1984. Paleopathology in an Iroquoian Ossuary, with Special Reference to Tuberculosis. *American Journal of Physical Anthropology*, 65:181–189.

Powell, M.L. 1988. *Status and Health in Prehistory: A Case Study of the Moundville Chiefdom*. Washington, D.C.: Smithsonian Institution Press.

———. 1990. On the Eve of the Conquest: Life and Death at Irene Mound, Georgia. In C.S. Larsen, ed., The Archaeology of Santa Catalina de Guale 2. Biocultural Interpretations of a Population in Transition, 26–35. *Anthropological Papers of the American Museum of Natural History*.

Rathbun, T.A., J. Sexton, and J. Michie. 1980. Disease Patterns in a Formative Period South Carolina Costal Population. *Tennessee Anthropological Association, Miscellaneous Paper*, 552–74.

Rechtman, A.M. 1929. Tuberculous Osteitis with Pathologic Resection of Seventh Rib. *The Journal of Bone and Joint Surgery*, 11:557–559.

Rice, D. 1989. The Osmore Drainage: Altitudinal Zonation, Economic Complementarity, and Vertical Control in the Southern Pacific Watershed. In D. Rice and C. Stanish, eds., *Ecology, Settlement and History in the Osmore Drainage*. Cambridge, BAR International Series S545.

Sinoff, C.L., and I. Segal. 1975. Tuberculous Osteomyelitis of the Rib. *South African Medical Journal*, 49:865–866.

Sommers, H.M. 1979. Disease due to Mycobacteria Other Than *Mycobacteria tuberculosis*. In G.P. Youmans, ed., *Tuberculosis*, 306–403. Philadelphia: W.V. Saunders.

Steinbock, R.T. 1976. *Paleopathological Diagnosis and Interpretation: Bone Diseases in Ancient Human Populations*. Springfield, Ill.: Charles C Thomas.

———. 1987. Comment in G.A. Clark et al., 55–57.

Sumner, D.R. 1985. A Probable Case of Prehistoric Tuberculosis from Northeastern Arizona. In C.F. Merbs and R.J. Miller, eds., Health and Disease in the Prehistoric Southwest. *Anthropological Research Papers*, 34:340–346. Tempe: Arizona State University.

Tatelman, M., and E.J.P. Drouillard. 1953. Tuberculosis of the Ribs. *The American Journal of Roentgenology*, 70:923–935.

Waaler, H., A. Geser, and S. Andersen. 1962. The Use of Mathematical Models in the Study of the Epidemiology of Tuberculosis. *American Journal of Public Health*, 52:1002–1013.

Walker, E.G. 1983. The Woodlawn Site: A Case for Interregional Disease Transmission in the Late Prehistoric Period. *Canadian Journal of Archaeology*, 7:49–59.

Widmer, L., and A.J. Perzigian. 1981. The Ecology and Etiology of Skeletal Lesions in Late Prehistoric Populations of Eastern North America. In J.E. Buikstra, ed., Prehistoric Tuberculosis in the Americas. *Northwestern University Archeological Program Scientific Papers*, 5:99–113. Evanston, Ill.: Northwestern University.

Williams, J.A., and S. Snortland-Coles 1986. Pre-Contact Tuberculosis in a Plains Woodland Mortuary. *Plains Anthropologist*, 114:249–252.

Williams, S.R., J.E. Buikstra, N.R. Clark, M.C. Lozada Cerna, and E. Torres. 1989. Mortuary Site Excavations and Skeletal Biology in the Osmore Project. In D. Rice and C. Stanish, eds., *Ecology, Settlement and History in the Osmore Drainage*, 329–346. Cambridge, BAR International Series S545.

Wolinsky, E. 1974. Nontuberculous Mycobacterial Infections of Man. *Medical Clinics of North America*, 58:639–648.

———. 1979. Non-Tuberculosis *Mycobacteria* and Associated Diseases. *American Review of Respiratory Disease*, 119:107–159.

Yau, A.C.M.C., and A.R. Hodgson. 1968. Penetration of the Lung by the Paravertebral Abscess in Tuberculosis of the Spine. *The Journal of Bone and Joint Surgery*, 50A:243–254.

SUMMARY OF AUDIENCE DISCUSSION: Several audience members expressed concern about drawing conclusions about etiological agents for human disease on the basis of theoretical mathematical models. Some pointed out that disease rates did not always correlate with population size, such as the high rate of tuberculosis in the small Bedouin groups. It was noted, however, that the author was identifying the larger population size with which certain smaller, more isolated groups came into contact during their periodic rituals.

Endemic treponematosis and tuberculosis in the prehistoric southeastern United States: Biological costs of chronic endemic disease

Mary Lucas Powell

The importance of infectious disease as a selective force in human adaptation has been argued eloquently during the past quarter century (Alland 1970; Armelagos et al. 1978; Buikstra 1981; Buikstra and Cook 1980; Burnet and White 1972; Cockburn 1973; McNeal 1976; Ortner and Putschar 1981). Many recent assessments of health in prehistoric populations have treated only in general terms the observed prevalence of nonspecific periostitis, osteitis, and osteomyelitis, with little or no consideration of specific infectious diseases. The most commonly reported nonspecific lesion, periostitis, may represent illness caused by endogenous bacterial and viral infections, certain endemic (and more rarely, epidemic) infectious diseases, nutritional disorders, trauma, or a variety of congenital syndromes (Greenfield 1980; Jaffe 1972). Its mere presence in an individual or a population reported without an epidemiological context is of minor significance in evaluation of the biological costs of infectious disease experience.

Differential diagnosis of specific infectious diseases requires familiarity with the pathogenesis and epidemiology of the diseases in question. Certain infectious diseases such as treponematosis and tuberculosis produce both pathognomonic skeletal pathology (e.g., caries sicca in the former, extensive vertebral destruction in the latter) and nonspecific response. Ortner and Putschar (1981:105) note that "infectious conditions affecting the skeleton tend to be subacute, chronic diseases and may not be the immediate cause of death." Because bone lesions typically occur relatively late in the progress of disease subsequent to considerable soft tissue involvement, their presence is indicative of relatively long-term immune response. Some chronic diseases typically produce abundant skeletal morbidity yet rarely result in death, because of the nature of their pathophysiological effects, while others may produce high mortality but few cases of skeletal involvement (Hackett 1951; Robbins and Cotran 1980). This paper outlines briefly the different biological costs of two chronic infectious diseases with distinctively different patterns of skeletal involvement and mortal impact, endemic treponematosis and tuberculosis, that have been recently identified in skeletal samples from late prehistoric American Indian populations in Alabama and Georgia.

Endemic treponematosis and tuberculosis

Yaws, endemic syphilis, and venereal syphilis are considered to be closely related disease entities because of the similarity of their causal organisms and the morphology of their lesions of skin and bone (Grin 1953; Hackett 1976; Hudson 1958; Turner and Hollander 1957). As regards levels of morbidity and mortality, however, the first two are radically different from their more dangerous relative, being both more widely prevalent and more benign because of their mode of transmission and their pathophysiological effects. They are typically contracted in early childhood through direct contact with infectious skin lesions rather than through venereal transmission. Prevalence levels in endemic regions approach 100%, and lesions occur in approximately 50% to 75% of late secondary and tertiary cases, resulting from hyperallergic response to superinfection by the sensitized hosts (Hackett 1951). Both congenital transmission and invasion of vital organ systems are rare, in contrast to the well-documented effects of venereal syphilis (Grin 1956; Murray et al. 1956). These diseases do not noticeably dampen fertility, and exert a negligible impact upon mortality except indirectly through secondary bacterial or mycotic infection of skin lesions.

In his comprehensive study of endemic yaws in Uganda (1951), C.J. Hackett sought to document the complete range of bone lesions observed radiographically in patients diagnosed by serological analysis. He noted in his introduction, "It is not the differential diagnosis of the changes present in one patient, but of those . . . in a whole population that is being considered." Hackett found that while some patients displayed "classic" bone lesions characteristic of late secondary and tertiary yaws (sabre shins, polydactylitis, and os-

TABLE 1. Tuberculosis and endemic treponematosis: comparison of morbid and mortal effects

	Tuberculosis	*Treponematosis*
	EPIDEMIOLOGY	
Pathogen	*Mycobacterium tuberculosis*	*Treponema pallidum*, *T. pertenue*
Mode of infection	Respiration, ingestion	Skin lesions
Modal age at exposure	Childhood	Childhood
Modal age at onset of disease	Late adolescence, early adulthood	Childhood
Duration of infectious state of patient	Decades (with latent periods)	5–10 years
	PATHOLOGY	
Initial lesions	Lungs, hilar lymph nodes	Mucocutaneous tissues
Subsequent lesions	Any organ system	Mucocutaneous tissue, bone
Prevalence of disease in endemic contexts	10–50% of exposed individuals	Virtually 100% of exposed individuals
Prevalence of skeletal involvement	3–15% of cases	50–75% of cases
Predominant skeletal response	Major: osteolytic Minor: osteoblastic	Major: osteoblastic Minor: osteolytic
Skeletal regions typically affected	Spinal column, hip and knee joints, ribs, sternum	Tibia, fibula, humerus, radius, ulna, clavicle, cranial vault, nasopalatal region
Potential for mortal effect	Moderate to high	Low

SOURCES: Hackett 1951, Hoeprich 1977, Hudson 1958, Kelley and Micozzi 1984, Myers 1951, Ortner and Putschar 1981, Robbins and Cotran 1980

teolytic lesions of the external cranial vault and nasopalatal region), many more exhibited minor nonspecific bone reactions (periostitis of long bone shafts, particularly the tibia, fibula, ulna, radius and clavicle). Subsequent radiographic examination of these patients indicated long-term persistence of bone changes after the disappearance of the clinical symptoms.

Commenting upon the high level of moderate skeletal morbidity without associated mortality, Hackett concluded that "it is improbable that septic infection of the bones is responsible for the changes seen. Untreated septic infection of the extent necessary to produce the wide spread changes seen in some cases would be accompanied by grave general symptoms and high mortality; whereas the patients showing these bone lesions were not severely ill, although they suffered considerable discomfort" (1951:13). Studies of yaws in other populations (Grin 1956) and of endemic syphilis in Bosnia (Grin 1953) and in southern Africa (Murray et al. 1956) present similar pictures of the biological costs of these diseases.

Tuberculosis is a chronic infectious disease caused by the gram-negative *Mycobacterium tuberculosis*. Clinical studies indicate that in endemic contexts, most people are infected in infancy or childhood, but more than half of the exposed but otherwise healthy individuals may never develop clinical disease (Myers 1951). Individuals with poor immune response may develop primary lesions within the lungs and hilar lymph nodes. If death does not ensue during the primary infection, the invading pathogens may be encapsulated by calcified tissue. This response halts immediate progression of the disease, but the organisms remain viable for decades (Robbins and Cotran 1980). Localized foci may rupture and spread mycobacteria via direct or hematogenous dissemination throughout the body, affecting all types of tissue including bone. Reinfection from active cases or reactivation of latent foci because of severe systemic stress may produce acute symptoms later in life (Hoeprich 1977).

In chronic tuberculosis overstimulation of immune responses in sensitized tissues may result in such proliferation of granulomatous tissue within the lungs that pulmonary function is compromised and death follows. Tuberculosis was a major cause of death in children, adolescents and young adults before the development of effective surgical and antibiotic therapy and was responsible for 260 deaths per 100,000 residents in Germany in 1892 (Ortner and Putschar 1981:142). Mortality from tuberculosis is particularly high in populations under severe stress from malnutrition, overcrowded and unsanitary living conditions, other diseases, and psychosocial stress (Hrdlicka 1909; Hoeprich 1977; Myers 1951). Under more healthful conditions, successful repair of tissue destruction permits extended survival of the host, an outcome incidentally in the pathogen's favor as it promotes subsequent infection of other hosts.

Table 1 summarizes the contrasting morbid and mortal effects of endemic treponematosis and tuberculosis. These differing patterns have important implications for paleopathological studies of the two diseases, for the following reasons. Tuberculosis is less "visible" than endemic treponematosis in skeletal series. In older museum collections, spinal elements tend to be less well represented than long bones because vertebrae are more prone to postmortem destruction and because in many field situations they were less systematically collected. In the Moundville series, for example, fewer than 40% of the individuals were represented by thoracic and/or lumbar vertebrae, the most common sites of tubercular bone lesions. By contrast, more than 70% were represented by the postcranial bones most characteristically affected by treponemal infection.

The nature of the lesions produced by the different diseases also plays a role in affecting favorably or unfavorably the chances for postmortem preservation. The osteolytic lesions characteristic of tuberculosis destroy bone tissue and weaken the fabric of affected skeletal elements. The osteoblastic lesions characteristic of endemic treponematosis produce additional bone, thickening the cortex of affected long bones and rendering them more resistant to dissolution.

Materials and methods

The first population sample discussed in this paper was excavated from the prehistoric American Indian community of Moundville located on the Black Warrior River some 13 miles southeast of Tuscaloosa in west central Alabama. It represents the Mississippian occupation of the site, which lasted from A.D. 1050 to 1550. By the mid-14th century, an estimated 3000 individuals were concentrated within the protective palisade and in small "suburban" clusters located nearby. The subtropical climate and the easily cultivated, fertile soils encouraged the development of a sophisticated subsistence regimen combining maize, squash, and beans with a wide variety of plentiful wild plant foods, game, and fish (Peebles 1978).

More than 1500 burials were excavated at Moundville between 1929 and 1941 by the Alabama State Museum of Natural History, and are presently curated at the Laboratory for Human Osteology at the University of Alabama in Tuscaloosa. From this large series, 564 skeletal individuals were selected on the basis of preservation and archeological provenience for investigation of the social and biological dimensions of health (Powell 1988).

The second population sample represents a late prehistoric community at the Irene Mound site, located near the mouth of the Savannah River on the Atlantic coast. This occupation was contemporaneous with Moundville, spanning three centuries (A.D. 1110–1400) during the Savannah and early Irene phases of the local Mississippian cultural tradition. Subsistence and other aspects of life in this smaller community were similar to those noted for Moundville. Continuous archeological excavations at the site from 1939 to 1940 supported by federal relief funds recovered 265 skeletal individuals, presently curated at the National Museum of Natural History, Smithsonian Institution, in Washington, D.C. The data reported here were collected as part of a general assessment of health at Irene Mound (Powell 1990).

For each series, all available bones were examined for macroscopic evidence of skeletal pathology. Observed lesions were classified as osteoblastic or osteolytic in morphology, as active or quiescent at the time of death, and according to their extent of involvement. The differential diagnoses of treponematosis and tuberculosis were based on identification of pathognomonic lesions and comparisons of the patterns of associated nonspecific skeletal pathology (Figure 1).

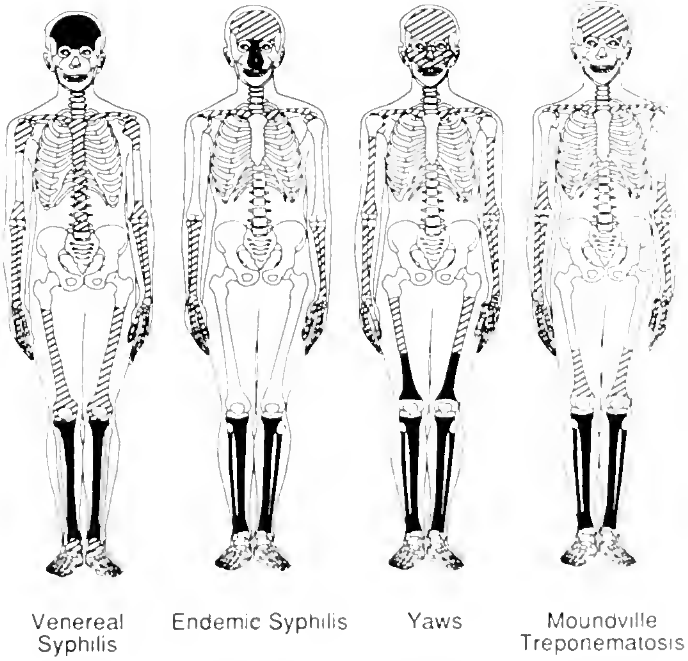


FIGURE 1. Distribution of skeletal lesions in four treponemal syndromes. Solid shaded areas are those most frequently affected; hatched areas are less often involved. Figures showing syphilis and yaws after Steinbock 1976.

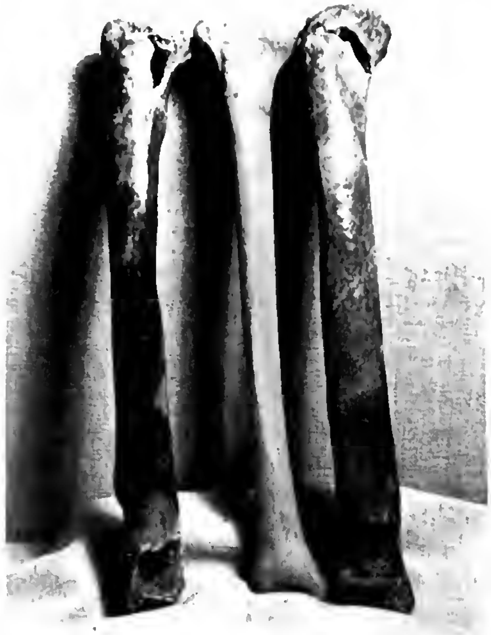


FIGURE 2. Irene Mound, NMNH 385528, treponematosis, sabre shins (shown with non-pathological tibia, center, for comparison).

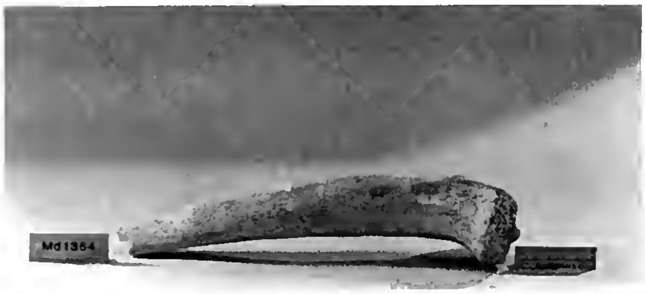


FIGURE 3. Moundville, tibia.

Results

A diagnosis of endemic treponematosis was initially suggested in both series by the frequent appearance of localized or extensive periostitis on the shafts of the tibia, fibula, radius and ulna. The thickness and degree of remodeling of this new bone suggested the recurrent episodes of periosteal inflammation described by Hackett (1951) during the late secondary and tertiary stages of yaws. Many tibiae display areas of localized apposition along the anterior crest, a region subject to the frequent minor trauma noted by Hackett as an exacerbating factor in soft tissue lesion formation. Others show more severe extensive pathological involvement, illustrated by cases from Irene Mound (Figure 2) and Moundville (Figure 3) that resemble the deformity known in modern treponematosis as "sabre shins."

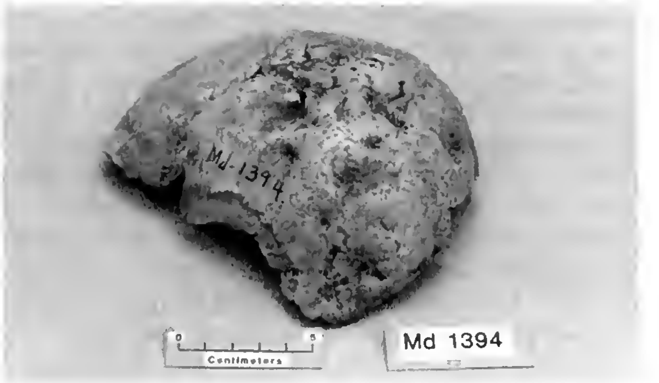


FIGURE 4. Moundville, cranial lesions.

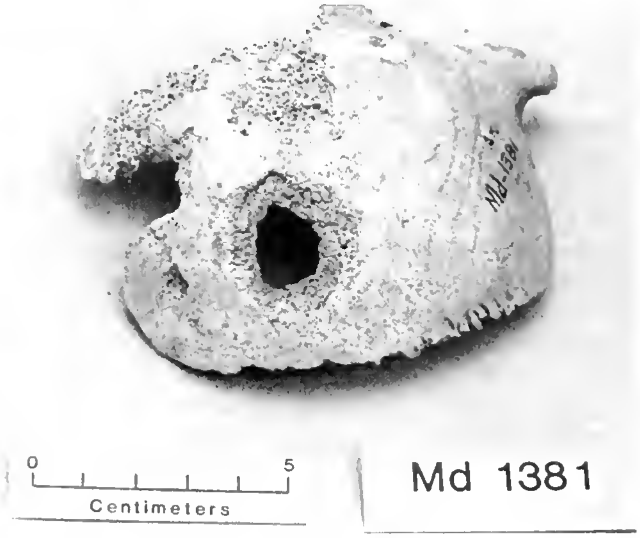


FIGURE 5. Moundville, cranial lesion in frontal.

Other lesions suggestive of late-stage treponemal disease appear as small, circular depressions on the outer cranial vault, seen here in an adult case from Moundville (Figure 4). The gummatous ulcers that often develop in yaws and endemic syphilis frequently infect bone lying close beneath the skin. Their particular pattern of tissue destruction and healing results in the pathognomonic osteolytic lesions known as "caries sicca" (Hackett 1976). The cranial lesions seen in these series are neither large nor extensive, and show considerable remodeling before death. The single exception is a large, penetrating, frontal lesion (Figure 5) from a young Moundville woman who probably died from superinfection. Posterior vault lesions (Figure 6) often show more clearly than frontal lesions the characteristic stellate configuration of the healed scar.

The mucocutaneous and osseous tissues of the nasal and oral cavities are also common sites of treponemal pathology. Osteolytic lesions penetrated the palate and maxilla of a

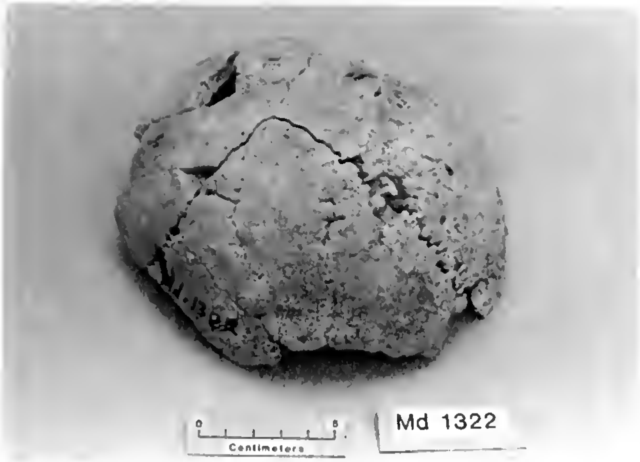


FIGURE 6. Moundville, posterior cranial vault, stellate lesions.

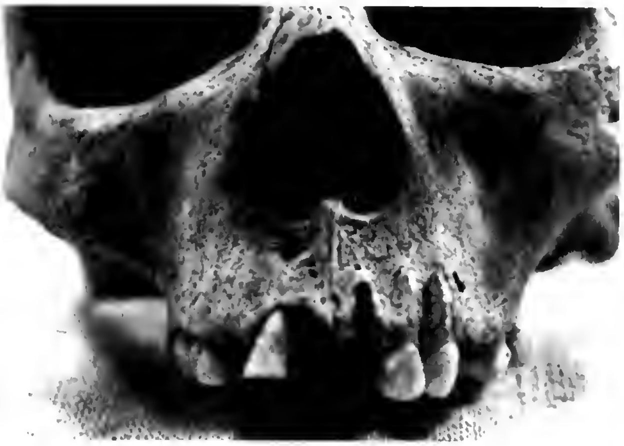


FIGURE 7. Irene Mound, NMNH 385540, treponematosis, right nasal margin remodeling.

young adult female from Irene Mound (Figure 7), who also displays remodeled frontal lesions. The right border of her nasal aperture shows extensive remodeling. Destruction of facial structures of this sort is known as "gangosa," a Spanish word referring to the harsh nasal quality of the victim's voice (Hudson 1958). Because of their pre-Columbian provenience, no historical descriptions of the Moundville or Irene Mound populations exist. However, in 1709 the Englishman John Lawson described ailments that resemble endemic treponematosis among the Santee Indians some 200 miles to the north of Irene Mound. He wrote, ". . . they have a sort of Rheumatism or Burning of the Limbs, which tortures them grievously, at which times their legs are so hot, that they employ the young People continually to pour water down them" (1709:223). Lawson also noted "another Distemper, which is, in some respects, like the Pox, but is attended with no Gonorrhea. This not seldom bereaves them of their Nose." (1709:223). The Santee made a clear distinction between pre-Contact and post-Contact diseases, leading Lawson to comment ". . . the Natives of America have for many Ages (by their own Confession) been afflicted with a Distemper much like the *Lues Venerea* which hath all the Symptions of the Pox, being different in this only: for I never could learn, that this Country-Distemper, or Yawes, is begun or continued with a Gonorrhea. . . . I have known mercurial Unguents and remedies work a Cure, following the same methods as in the Pox" (1709:18).

The "Rheumatism" and "nocturnal pains in the limbs" described to Lawson by the Santee correspond well to the episodes of ostalgia (deep bone pain) that afflict late secondary and tertiary cases of yaws and endemic syphilis. The ulceration and loss of nasal structures, the absence of urethral discharge ("gonorrhea"), the responsiveness of the skin lesions to "mercurial Unguents and Remedies," and the essentially self-limiting nature of the disease are also prominent characteristics of endemic treponematoses.

In both series, the demographic profiles of individuals displaying skeletal pathology diagnostic or suggestive of endemic treponemal disease closely matched the demographic profiles of the series as a whole. Skeletal evidence of the disease was age-accumulative: older adults were more likely to bear lesions than were younger adults, adolescents, or children. The great majority of the observed lesions were well remodeled, indicating that the disease was not active around the time of death.

Lawson commented that the Santee "are wholly Strangers to . . . the Phthisick," a term referring to pulmonary tuberculosis (Jaffe 1972:955). Although it may have been absent in that population, tuberculosis is evidenced by a variety of skeletal lesions at Moundville and at Irene Mound. Of the ten individuals from Moundville with bone lesions diagnostic of tuberculosis, only one displays "classic" vertebral destruction. This young man died in his late twenties, a decade short of the average adult male age at death. Virtually his entire spine from T3 downward to his sacrum shows pathological involvement (Figure 8). The bodies of six thoracic vertebrae have been destroyed, producing the anterior kyphosis characteristic of spinal tuberculosis or Pott's disease. Numerous large round osteolytic lesions with smooth margins appear in the bodies of several lower thoracic and lumbar vertebrae. As compensation for the loss of bone mass, the remaining portions of several vertebrae have fused to provide support for the thorax. Ribs 6 through 10 on both sides display small shallow osteolytic lesions and poorly remodeled periostitis on their pleural aspects. Their necks and heads are considerably distorted, with the same combination of destructive and proliferative reaction. No other postcranial tuberculous lesions were noted, and the skull is unfortunately absent. The extensive remodeling evident in all areas of pathological involvement indicates survival for some considerable length of time despite severe deformity, as has been abundantly documented in modern clinical cases (Myers 1951).

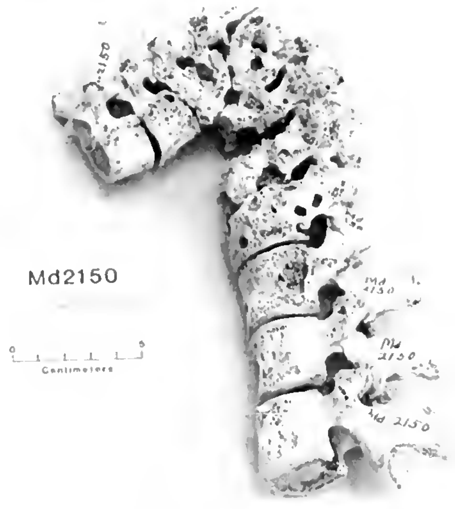


FIGURE 8. Moundville, spinal tuberculosis.

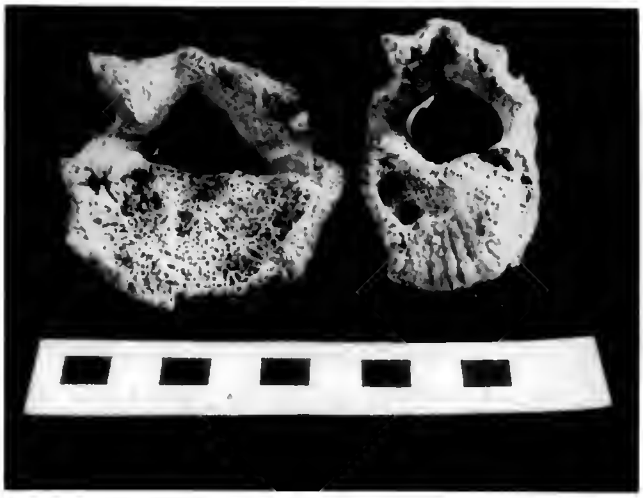


FIGURE 9. Irene Mound, NMNH 385562, lumbar vertebrae, TB lesions.

Two mature women from Moundville each bore osteolytic lesions on a single lumbar vertebrae, but such isolated destruction suggests a diagnosis of blastomycosis rather than tuberculosis (Chick 1971). No cases with tuberculous lesions of the hip, knee, or cranium were observed. However, seven adults and two subadults displayed rib lesions closely matching those described from clinical cases in the Hamann-Todd collection by Kelley and Micozzi (1984) as characteristic of chronic pulmonary tuberculosis. The two subadults (ages birth to 6 months and 7 to 8 years) display extensive, diffuse, unremodeled periostitis on the pleural aspects of multiple right and left ribs. The seven adults were evenly distributed through the third, fourth, and fifth decades of life, and all displayed lesions at least partially remodeled at death. Women and men were equally represented. This age and sex distribution of adult cases matches that reported in the Hamann-Todd study.

In the Irene Mound series, three cases bear osteolytic lesions in thoracic or lumbar vertebrae suggestive of spinal tuberculosis. L2 through L5 of a young woman (Figure 9) display extensive shallow lesions on the paradiscal surfaces. T3 and T4 of a second young woman bear deep centrally located osteolytic lesions, and L2 through L5 of a young man exhibit widespread circumferential lesions. One older woman displayed extensive destruction of her left sacroiliac auricular surface with minimal remodeling (Figure 10). Two adults displayed periostitis on the pleural aspect of the scapula, and the pleural aspect of the sternum was also similarly affected in one case.

As in the Moundville series, the most abundant extravertebral tuberculous lesions were those affecting the pleural aspects of the ribs. These lesions appeared in 8 of the 10 cases. In one case focal osteolytic lesions were surrounded by osteoblastic response. Seven of the 8 displayed localized or diffuse periostitis, illustrated here by the most extreme case (Figure 11), the young female adult with multiple lumbar paradiscal lesions.

The mean age at death of the 10 Irene Mound cases with tuberculous vertebral, pelvic, and rib lesions was 29.5 years, five years below the modal sample age of 34.9 years. One young adolescent, one older adolescent, and four adults aged 20 to 24.9 years constituted the younger set of cases. Approximately 15 years separated the oldest of these cases from the youngest of the four remaining cases, all in their early to late 40s. Female prevalence is double that for males, in contrast to the equal representation of the sexes at Moundville.

Conclusions

Analyses of archeological data on subsistence and skeletal indicators of nutritional quality at Moundville and Irene Mound suggest that both populations enjoyed diets adequate to promote vigorous immune response to infectious disease. The rank-stratified social organization of the Moundville chiefdom may have created certain dietary differences between elite and non-elite segments of the population, but a broad range examination of the social dimensions of health and disease at the paramount site revealed no significant differences in dental disease or skeletal pathology (Powell 1988). The nature and degree of social differentiation at Irene Mound have not yet been delineated, but significant rank-determined, dietary inequalities seem improbable (D.G. Anderson, pers. comm. 1987). Good nutrition is a less critical factor in resistance to endemic treponematosis than in re-

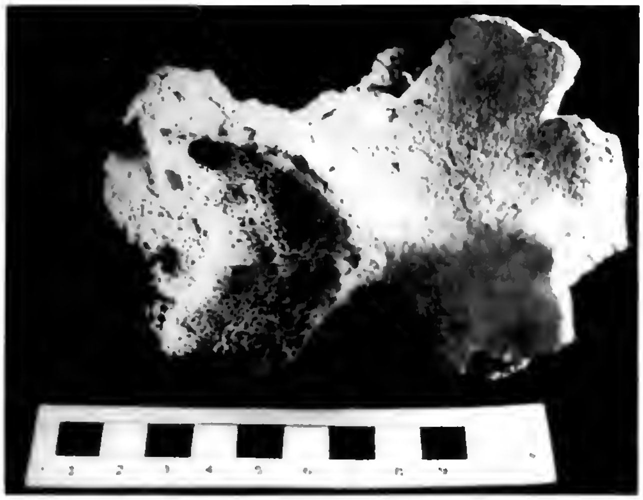


FIGURE 10. Irene Mound, NMNH 385411, SI joint lesion, TB.

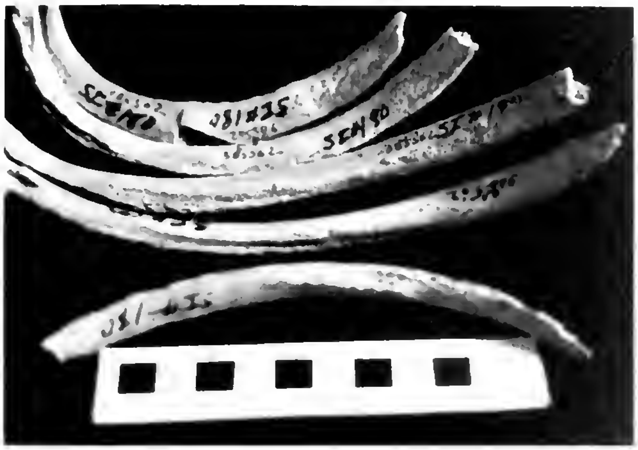


FIGURE 11. Irene Mound, NMNH 355562, ribs, pleural periostitis.

sistance to tuberculosis (Hackett 1951; Myers 1951), but its importance to nonspecific mechanisms safeguarding health (e.g. phagocyte production) should not be forgotten.

The human remains excavated from these sites some fifty years ago and carefully curated as part of systematic museum collections in Alabama and Washington, D.C., provide convincing diagnostic evidence of the presence of two chronic endemic infectious diseases. Analysis of the patterns of the associated bone lesions indicates to some degree the breadth of their prevalence within the populations, respecting neither demographic parameters nor exalted social rank. But the skeletal record alone cannot convey the full extent of the biological costs of these insidious and persistent forms of illness, the toll levied upon each successive generation in terms of death, discomfort, deformity, decreased energy, and general debilitation of resistance to other stresses. Each disease followed its own distinctive trajectory from childhood infection through subsequent episodes of illness throughout adult life, the one more blatantly disfiguring and the other more subtly lethal. The presence of each made the presence of the other a greater burden to the unfortunate individuals who were doubly afflicted and to the populations in general. Endemic treponematosis was no doubt regarded as one of life's regular nuisances, while tuberculosis was a rarer but far more serious matter.

An eminent paleopathologist of the author's acquaintance continually adjures his students to "remember that a dog may have both ticks and fleas," and that differential diagnosis should always be sensitive to the possibility of multiple pathological conditions simultaneously affecting an individual. To apply this same analogy again in a somewhat different manner, the skeletal lesions of endemic treponematosis and tuberculosis may be likened to the parasites visible on a dog's body. Their simple presence in terms of the amount of blood consumed, the burden of their collective weight, and the minor irritation of their bites does not reflect directly the magnitude of their potential impact upon the dog's health. Hidden within them may be agents that produce serious illness or death, depending upon the circumstances surrounding host and parasites.

In similar fashion, the degree of skeletal pathology observed does not equal directly the biological costs of the diseases in question, either in terms of numbers of individuals who were ill or the range of possible symptoms. Clinical studies identify numerous cases that would be invisible in the archeological record. What we actually see in the bones is not the entirety of disease that did exist, but the evidence alerts us to the presence of diseases whose true costs may be inferred from the modern epidemiological and clinical literature.

In the face of growing political pressure for reburial of human skeletal remains in the United States and elsewhere, paleopathologists should feel a particular obligation to bring to bear our most sophisticated theoretical and methodological capabilities to investigations of the dimensions of health in the prehistoric past. Differential diagnosis provides a powerful tool for conducting such analyses, and to ignore its full potential is to deliberately limit the contributions of our research.

Acknowledgments

This paper is the result of numerous discussions with Donald J. Ortner, my advisor during a Postdoctoral Fellowship at the Smithsonian Institution, about the problems and potential of interpretation of skeletal disease. I have benefited greatly from his sophisticated insights into critical issues, and he is coauthor in fact, if not in name. Misinterpretations of the data, however, are solely my own.

Literature cited

Alland, A. Jr. 1970. *Adaptation in Cultural Evolution: An Approach to Medical Anthropology.* New York: Columbia University Press.

Armelagos, G.J., A. Goodman, and K.H. Jacobs. 1978. The Ecological Perspective in Disease. In M.H. Logan and E.E. Hunt Jr., eds., *Health and the Human Condition: Perspectives on Medical Anthropology,* 71–84. North Scituate, Mass.: Duxbury Press.

Buikstra, J.E., ed. 1981. Prehistoric Tuberculosis in the Americas. *Northwestern University Archaeological Program, Scientific Papers,* 5. Evanston, Ill.: Northwestern University.

Buikstra, J.E., and D.C. Cook. 1980. Paleopathology: An American Account. *Yearbook of Physical Anthropology,* 20:316–328.

Burnet, Sir M., and D.O. White. 1972. *Natural History of Infectious Disease.* Cambridge, U.K.: Cambridge University Press.

Chick, E.W. 1971. North American Blastomycosis. In R.D. Baker, ed., *Human Infections with Fungi, Actinomycetes and Algae,* 465–506. New York: Springer-Verlag.

Cockburn, A. 1973. *Infectious Diseases, Their Evolution and Eradication.* 2d edition. Springfield, Ill.: Charles C Thomas.

Greenfield, G.B. 1980. *Radiology of Bone Diseases.* 3d edition. Philadelphia: J.B. Lippincott.

Grin, G.I. 1953. Epidemiology and Control of Endemic Syphilis: Report on a Mass-Treatment Campaign in Bosnia. *World Health Organization Monograph,* 11. Geneva.

______. 1956. Endemic Syphilis and Yaws. *Bulletin of the World Health Organization,* 15:959–973.

Hackett, C.J. 1951. *Bone Lesions of Yaws in Uganda.* Oxford: Blackwell Scientific Publications.

______. 1976. *Diagnostic Criteria of Syphilis, Yaws and Treponarid (Treponematoses) and of Some Other Diseases in Dry Bones.* Berlin: SpringerVerlag.

Hoeprich, P.D., ed. 1977. *Infectious Diseases, a Modern Treatise on Infectious Processes.* 2d edition. New York: Harper and Row.

Hrdlicka, A. 1909. Tuberculosis among Certain Indian Tribes of the United States. *Bureau of American Ethnology Bulletin,* 42.

Hudson, E.H. 1958. *Non-Venereal Syphilis, a Sociological and Medical Study of Bejel.* Edinburgh: E. and S. Livingstone.

Jaffe, H.L. 1972. *Metabolic, Degenerative, and Inflammatory Disease of Bones and Joints.* Philadelphia: Lea and Febiger.

Kelley, M.A., and M.S. Micozzi. 1984. Rib Lesions in Chronic Pulmonary Tuberculosis. *American Journal of Physical Anthropology,* 65:381–386.

Lawson, J. 1709. *A New Voyage to Carolina.* London.

McNeill, W.H. 1976. *Plagues and Peoples.* New York: Anchor Press.

Murray, J.F., A.M. Merriweather, and M.L. Freedman. 1956. Endemic Syphilis in the Bakwena Reserve of the Bechuanaland Protectorate. *Bulletin of the World Health Organization,* 15:975–1039.

Myers, J.A. 1951. *Tuberculosis among Children and Adults.* 3d edition. Springfield, Ill.: Charles C Thomas.

Ortner, D.J., and W.G.J. Putschar. 1981. Identification of Pathological Conditions in Human Skeletal Remains. *Smithsonian Contributions to Anthropology,* 28. Washington, D.C.: Smithsonian Institution Press.

Peebles, C.H. 1978. Determinants of Settlement Size and Location in the Moundville Phase. In B.D. Smith, ed., *Mississippian Settlement Patterns.* New York: Academic Press.

Powell, M.L. 1988. *Status and Health in Prehistory: A Case Study of the Moundville Chiefdom.* Washington, D.C.: Smithsonian Institution Press.

______. 1990. On the Eve of the Conquest: Life and Death at Irene Mound, Georgia. In C.S. Larsen, ed., The Archaeology of Mission Santa Catalina de Guale: 2. Biocultural Interpretations of a Population in Transition, 26–35. *Anthropological Papers of the American Museum of Natural History.*

Robbins, S.L., and R.S. Cotran. 1980. *Pathologic Basis of Disease.* 2d edition. Philadelphia: W.B. Saunders.

Steinbock, R.T. 1976. *Paleopathological Diagnosis and Interpretation.* Springfield, Ill.: Charles C Thomas.

Turner, T.B., and D.H. Hollander. 1957. *Biology of the Treponematoses.* Geneva: World Health Organization.

SUMMARY OF AUDIENCE DISCUSSION: The absence of skull involvement by tuberculosis in this study is consistent with the paucity of reported cases both in ancient and in modern populations. Prior to the availability of effective chemotherapy, tuberculous meningitis certainly was not rare, and the primarily cortical response which one would expect ought to be easily separable from the "caries sicca" lesion characteristic of cranial treponematosis. It is conceivable tuberculous periostitis is present more frequently than reported but overlooked because the lesions may be small, of nonspecific structure and difficult to see in an intact skull because of their endocranial position. The rapid course of tuberculous meningitis in modern populations also probably provides insufficient time for development of the osseous reaction. Since a slower course would imply the development of greater resistance to the tubercle bacillus, it is unlikely that its course was any slower in antiquity.

Vertebral tuberculosis in ancient Egypt and Nubia

Eugen Strouhal

It has become a widely accepted view that diagnosis of tuberculosis from archeologically excavated human skeletal remains is not easy, but that a reasonably reliable prediction can be made if the spine is involved (Haneveld 1980:2). Vertebral tuberculosis (spondylitis tuberculotica) was first described by Sir Percival Pott, a surgeon at St. Bartholomew's Hospital in London, in 1779 (Pott 1790) and named *malum Potti* in honor of him. It deserves to be studied by modern paleopathologists also 200 years later. Its pathological anatomy is usually very characteristic, making it possible to distinguish it from other spinal diseases.

While modern clinical reports indicate that skeletal tuberculosis as a rule occurs as a complication secondary to either pulmonary or intestinal tuberculosis in about 1% of such patients, its incidence was considerably higher during the preantibiotic era: between 5 and 7% (Steinbock 1976:175; Zimmerman and Kelley 1982:103). In the same period the spine was involved in 25–50% of skeletal tuberculosis cases (Steinbock 1976:176). This means that cases of skeletal tuberculosis represent about 1–3.5% of the total number of people who were infected by tuberculosis.

It is encouraging that the acid-fast bacterium *Mycobacterium tuberculosis (bacillus Kochi)* was recently demonstrated in a Peruvian mummified child of the seventh century A.D. Nasca culture (Allison et al. 1973) as well as in the vertebrae and pulmonary blood of an Egyptian mummified child from Thebes West, dated 1000–400 years B.C. (Zimmerman 1977,1979). Nevertheless, bacteriological techniques can be used only exceptionally in excavated material and the agent of the disease is not always preserved in it. For current studies on the incidence of tuberculosis in ancient populations, however, macroscopic and radiological diagnosis of cases of vertebral tuberculosis may still be used as convenient and technically simple methods. The presence of vertebral tuberculosis has to be considered at the same time as evidence for the presence of other tuberculotic forms, such as pulmonary, intestinal or glandular, without, however, revealing their specific frequencies (Grmek 1983:265).

Moreover, the description of each newly detected case adds some new knowledge to the morphology, extent, and course of the disease. This applies to two cases recently found in a Middle Kingdom burial in Egypt (case no. 1) and in a Christian period cemetery in Nubia (case no. 2). They are of interest because of their considerable extent and contrasting developmental phases, pointing to different social implications.

Case number 1

ARCHEOLOGICAL BACKGROUND

An isolated secondary burial of Middle Kingdom dating was found in the 5th dynasty Pyramid Temple of King Raneferef at the royal cemetery at Abusir by the mission of the Czechoslovak Institute of Egyptology, Charles University, Prague, in 1984. The burial of a male called Khuyankh (Hwy'nh) (900/1/84) was placed at the bottom of a rectangular shaft excavated in the northern half of room AA-Ec, situated in the NE corner of the *hut-nemet* section of the temple. The lower part of the shaft was lined with sun-dried bricks delimiting the burial chamber, oriented with its longer axis roughly N-S. As no traces of roofing were detected, it seems probable that after insertion of the coffins the shaft was simply filled up with sand.

The dead individual was provided with two coffins. The external, box-shaped one was decorated on its outer surface by inscriptions, and on its interior sides by further inscriptions, coffin texts, and pictures of offerings. The internal, anthropoid coffin was lying on its left side inside the external coffin with the head end to the north. The skeleton, 155 cm long, was in an extended position at the bottom of the inner coffin with the skull turned with its face down.

In the inner coffin two faience beads and the head of a wooden stick were found. South of the foot end of the outer coffin a low chest was placed, divided by two boards into four compartments with remnants of organic matter and wrappings on their bottom. A large, globular storage vessel stood in the NE corner of the burial chamber.

EMBALMING METHOD

Several black spots could be observed on the skull. Bones of the lower extremities (except the missing femora) were stained with a dark, almost violet color. Both are traces of resin used to smear the wrappings during embalming. Some textile fragments with pieces of stiffened resin were found among the bones and fragments of wood of the inner coffin.

Remnants of dried brain were still present in the cerebral cavity. The nasal skeleton was found intact. Both these findings attest that brain removal was not performed, being reserved only for persons of royal or high official rank during the Middle Kingdom times (Strouhal 1986:145).

AGE

Cranial sutures showed a progressive state of obliteration (C3, S2 + 3, L1 + 2). The complete dentition (with only crowns of right upper C and left upper I 1 and 2 broken off) was considerably abraded, ranging from points of exposed dentine on lower M3 to complete removal of the crowns on upper left C and upper right premolars, but without pulp exposure. A caries lesion was present on the left lower M2, destroying the distal half of the crown and, through involvement of the pulp, causing a great, longitudinally oriented oval cyst opening on the outer aspect of the alveolar process (13 × 7 mm, depth 7 mm). Resorption of the alveolar process was of medium degree according to the scale of Brothwell (1963:150). Considerable deposits of dental calculus were accumulated on the upper left molars and on all lower premolars both buccally and lingually, and on the lower molars lingually. Medium large deposits were present on the upper right molars lingually (according to the scale of Brothwell).

All epiphyseal and apophyseal fissures of the postcranial skeleton were completely fused. Pubic symphysis relief resembled phase 8 of Todd (1920) pointing to the range of 39–44 years. No synostosis of the sternal parts occurred. Only beginnings of lipping on the left humeral head and a rugged surface of the tubercula minora could be observed. The femora were missing. Slight osteophytic outgrowths were present on the patellae, and some of medium size on the calcanei. No arthritic changes were apparent in any joint. The grade of vertebral osteophytosis in sections not directly involved in the pathological changes to be described (C1–7, T1–8, L2–5, S1) was mostly of medium degree (osteophytes more than 3 mm long but not bridging) or slight (osteophytes less than 3 mm). Spondylarthrosis could be detected in joints T6–7 and T7–8 and osteochondrosis of intervertebral discs between C5–6 and C6–7, both probably related to the main pathology of the spine.

According to the described features the individual died between 40 and 50 years of age.

SEX

In spite of only a slightly developed glabella (Broca 2) and supraorbital arches (Eickstedt 2) as well as of nasofrontal transition on a medium curved arch, most other secondary sexual features pointed to the male sex. There was a slightly oblique forehead, a medium protuberantia occipitalis externa (Broca 2), a medium thick and long mastoid process with a deep incisura mastoidea, a medium thick upper orbital margin, slightly to medium developed marginal process, a well-developed muscular relief of the nuchal and mandibular regions, a slightly averted mandibular angle, and a broad square chin. In addition moderately developed apophyses of the pelvis, a medium deep and large ischiatic notch, absence of a preauricular sulcus, a big oval foramen obturatum, a sagittally narrow pelvic inlet, an outstanding pubic tubercle, a flaring lower aspect of the pubic bone, an acute subpubic angle (50°), and a low ischio-pubic index (left 65.0, right 61.3, according to Thieme and Schull 1957:269) all indicate the male sex unequivocally.

GENERAL PHYSICAL FEATURES

The body build was moderately robust, with musculature developed to a medium degree on the lower limbs and slightly less developed musculature on the upper limbs. Stature was reconstructed according to the tables of Trotter and Gleser (1952) for American Negroes, which were found to fit better to proportions of the Nubians (Strouhal and Jungwirth 1984:119–122) as well as Egyptians (Robins 1983:17–20), and appeared high (171.4 cm).

SPINAL PATHOLOGY

The vertebral column viewed from the sides was strongly bent in the lower thoracic third (Figures 1,2). The bodies of five vertebrae—T9 to L1—were completely merged and their neural arches, intervertebral joints, and ventral portions of the interspinous ligaments (the last except T10–11, Figures 3–5) were also fused. While the body of L1 retained its individual shape, bodies of the last four thoracic vertebrae had almost disappeared and their remnants joined into an uniform, wedge-shaped formation (anterior height 2 mm, posterior height 36 mm) merging with the wedge-shaped body of L1 (anterior height 7 mm, posterior height 20 mm). The radiogram (Figure 6) revealed a regular, strain-conditioned trabecular structure without remnants of residual cavities or intervertebral spaces. The right side of the formation was covered by a thin layer of newly formed bone with fine, radial ridges on its surface. The left side was strengthened by a thickly ossified lateral longitudinal ligament, encompassing also the 10th costovertebral joint.

Vertebra T8 also had a wedge-shaped body (anterior height 5 mm, posterior height 20 mm) and it was firmly fused with the mentioned formation in intervertebral joints as well as by

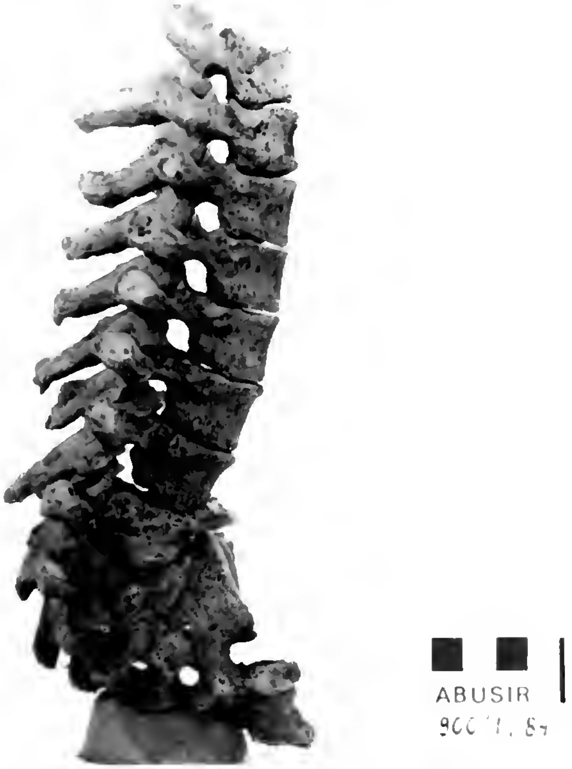


FIGURE 1. Case no. 1. Thoracic spine with L1 showing strongly arched deformity of T8–L1, right lateral view.

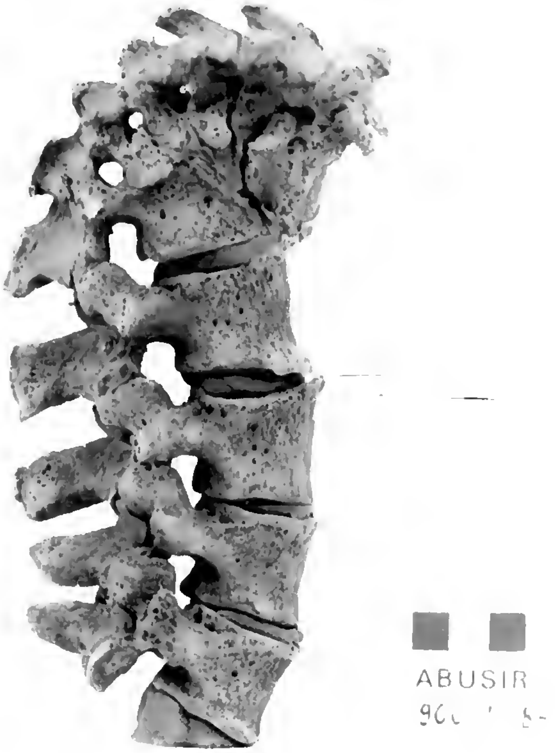


FIGURE 2. Case no. 1. Lower third of thoracic and lumbar spine showing arched deformity of T8–L1, right lateral view.

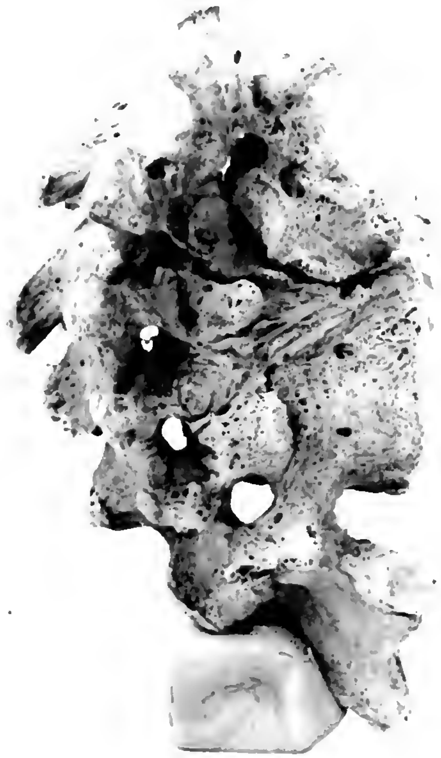


FIGURE 3. Case no. 1. Wedge-shaped T8 and fused mass of bodies T9–12 united with L1, right lateral view.

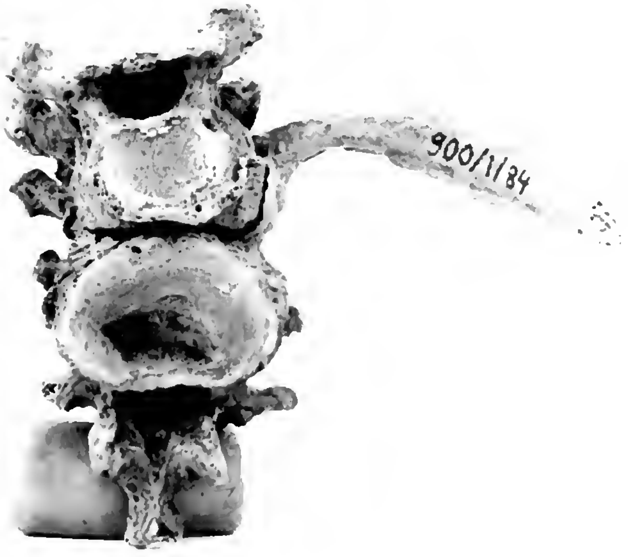


FIGURE 4. Case no. 1. Owing to a bend of 130°, upper terminal plate of T8 and lower terminal plate of L1 with a deep depression are visible in frontal view.

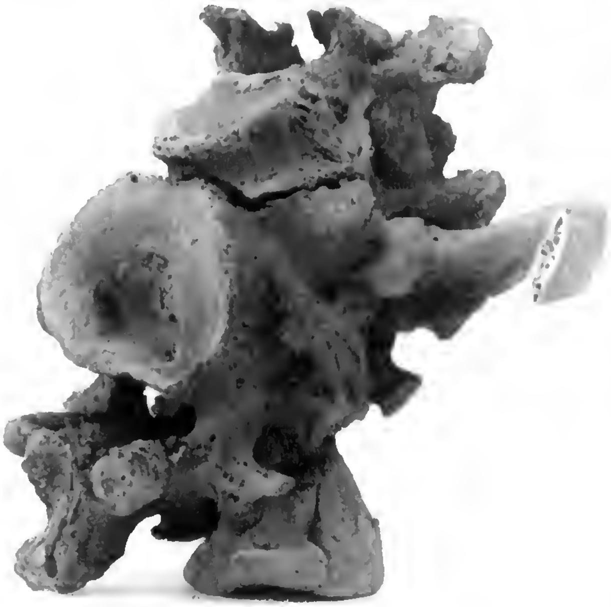


FIGURE 5. Case no. 1. Deformity covered by ossified left lateral longitudinal ligament, encompassing 10th costovertebral joint.

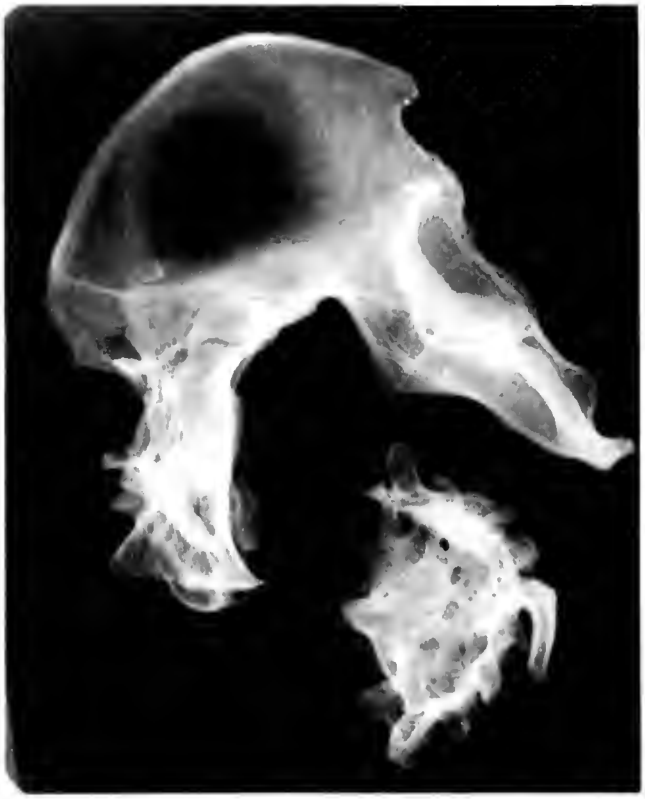


FIGURE 6. Case no. 1. Radiogram of right sacroiliac ankylosis *(above)*, axial projection, and fused vertebrae T8–L1 *(below)*, lateral projection.

ossification of the interspinal ligament. The intervertebral space T8–9 was preserved (height 2 mm) and there was also a wavy chink dividing the ossified left lateral longitudinal ligament. The right side of the body of T8 was bare.

Intervertebral openings and the spinal canal did not show any reduction that could have caused neurological problems.

The course of the vertebral column was deformed both in the frontal and sagittal planes. There was a slight, S-shaped scoliosis, sinistroconvex in the lumbar and lower thoracic sections, dextroconvex in the upper thoracic half.

The kyphotic angulation reaching 130° appeared more important. To compensate for this protuberance, the remaining, healthy lumbar vertebrae showed increased anterior heights compared with their posterior heights (L2 36/26, L3 34/30, L4 32/28, L5 32/26 mm). At the same time, the physiological thoracic kyphosis was reversed into a compensatory lordosis, in the section not involved in the pathology, by a similar increase of anterior heights compared with the posterior ones (T3 21/19, T4 20/18, T5 22/20, T6 23/20, T7 22/21 mm). The remaining correction of the upright posture must have been achieved by hyperlordosis of the cervical spine and dorsal flexion of the head. Also with these compensatory adaptations, the position of the lower thoracic spine was almost horizontal, causing deformation of the thorax and heavy pressure on thoracic and abdominal internal organs. The spinal deformation lowered substantially the living stature of the man. The difference between the value calculated according to lengths of long bones and the measurement of the body length in situ was 16.4 cm. In spite of these changes, the hunchback was able to walk (possibly with the help of a stick, whose head was put into his inner coffin) and work.

The described changes could, by the unnatural twist of the spine, also have caused deep, oval depressions on terminal plates of the neighboring vertebrae L1 + 2 and L2 + 3, signs of the prolapse of the nucleus pulposus of the intervertebral discs (Figures 4,5). Osteochondrosis of the intervertebral discs C5–6 and C6–7 and spondylarthrotic changes in the joints T6–7 and T8–9 may also have been associated with the adaptive changes of the gravity of the thoracic spine.

SACROILIAC SYNOSTOSIS

The right sacroiliac joint was ankylosed by means of a thick layer (up to 4 mm) of newly formed bone covering the anterior half of the upper margin and the upper half of the anterior margin of the facies auriculares (Figures 6,7). The remaining margins and the inner space were free. The left sacroiliac joint presented osteophytic lipping on the margins of both facies auriculares (2–5 mm). These changes could have been adaptive, strengthening the basis of the deformed spine.

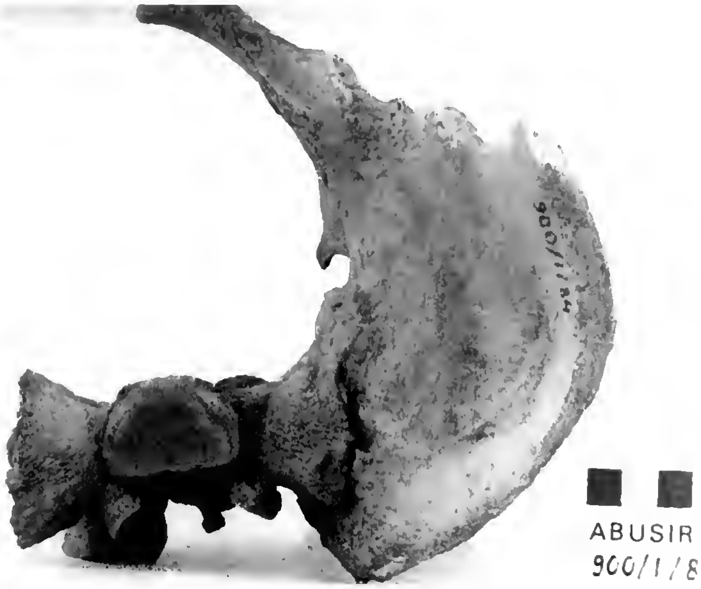


FIGURE 7. Case no. 1. Synostosis of part of right sacroiliac joint, cranial view.

OSTEOMA ON THE RIGHT FIBULA

A longitudinally oriented oval, roundish protrusion (18 × 10 mm, elevated 4 mm) was situated at the medial aspect of the distal end of the right fibula. Its surface was covered by compact bone, smooth or slightly uneven.

CONGENITAL ANOMALIES

A bilateral small foramen arcuatum atlantis (diameter 1 mm) was combined with bilateral, anteriorly open foramina transversalia atlantis. The left foramen transversale epistrophei was also open laterally and there was an anomalously small opening (diameter 2 mm) at the site of the right foramen transversale.

DIAGNOSIS OF THE SPINAL PATHOLOGY.

Features considered to be characteristic for spinal tuberculosis in current paleopathological literature have been compared for both cases in Table 1. Leaving aside age and sex, there are 20 features of which the majority fit for case no. 1. There are, however, seven features which disagree with the scheme and need to be explained.

The extent of the pathology usually involves two to four vertebrae (Manchester 1983:40; Zimmerman and Kelley 1982:105), but occasional cases may involve considerably more (examples are quoted by Zimmerman and Kelley 1982:105), as shown by our case. Lack of the progressive erosion of the circumferential surface of vertebral bodies betrayed the termination of the activity of the disease. The same applied for the absence of a recent central abscess cavity in any of the vertebrae. In the course of the process, which was limited only to vertebral bodies, no changes leading to narrowing of the intervertebral openings or of the neural canal occurred. Paravertebral abscesses, derived from original abscesses in vertebral bodies T8–L1, apparently did not affect any bony surface to leave observable changes. Not every patient with tuberculosis must be emaciated. Our case indicates a successful course of the disease thanks to the extraordinary resistance of the host.

The strong kyphotic curvature observed in our case, instead of an angular nick, also can be reconciled with the diagnosis of spinal tuberculosis. It was the result of summation of six lesser angular kyphoses which gradually developed after evacuation of the abscesses and pathological fractures of bodies of the afflicted vertebrae.

COURSE OF THE DISEASE

The infection must have begun early in childhood as evidenced by the adaptive greater increase of anterior heights of the healthy vertebrae. It is well known that before the development of an effective therapy, vertebral tuberculosis developed during the first decade of their life in 50–70% of tuberculous children and usually appeared 9 months to 2 years after the primary infection (Ulrich-Bochsler et al. 1982: 1322). Other authors also stress the onset in early childhood, mostly before 7 years of age (Ortner and Putschar 1981:145).

From the beginning of the disease in his first decade of life the afflicted man lived 30–50 more years, during which complete healing occurred except for the preservation of the deformity of the spine by firm fusion of the remnants of the involved vertebral bodies. It seems highly probable that during this long period his immune response succeeded in subduing also other possible manifestations of *bacillus Kochi* in his other organs.

TABLE 1. Comparison of characteristic features of spinal tuberculosis of cases 1 and 2

Feature	*Case no. 1*	*Case no. 2*
Age at death	40–50 yr	22–24 yr
Sex	male	male
Thoracolumbar localization	+ (T8–L1)	+ (T1–L5)
Extent	6 vertebrae	17 vertebrae
Progressive erosion of circumferential aspect of body	–	+
Central abscess cavity within vertebral body	–	+ (T11)
Wedge-shaped vertebral body	+ (T8–L1)	+ (T11, L1)
Complete healing of body without residual cavity	+ (T8–L1)	+ (L1)
Fusion of vertebrae with destruction of intervertebral discs	+	–
Noninvolvement of neural arch and its processes	+	–
Secondary arthritis and ankylosis of intervertebral joints	+	+
Ossification of interspinous ligaments	+	–
Ossification of lateral longitudinal ligaments	+ (left)	–
Apposition of new-formed bone	+ (right)	–
Fusion of costovertebral joints	+ (left 10th)	–
Compensatory growth of healthy vertebral bodies	+	–
Narrowed intervertebral openings	–	+ (right T9–10)
Narrowed neural canal	–	–
Kyphosis	+ (strong bend)	+ (double angle)
Scoliosis	+ (S shaped)	+ (dextroconvex)
Evidence of paravertebral abscess	–	+ (bilateral)
Corporal emaciation caused by advanced tuberculosis	–	–

DIFFERENTIAL DIAGNOSIS

Most other diseases that can cause a similar picture, listed by Steinbock (1976:176,179), are systemic, and leave traces also in other skeletal locations, which were not found in our case. The possibility of a healed compression fracture, however, has to be considered, although usually only a single vertebra is involved. Its areas of predilection are the cervical and upper thoracic spine. Destruction of the vertebral body is less extensive than observed in our case (Ortner and Putschar 1981:149). We may add that on radiograms a diagonal break may be detected and that the intervertebral spaces usually remain intact.

SOCIAL IMPLICATIONS

Our patient very probably must have been nursed during the initial period of his disease in childhood. After healing was completed and the deformity of his spine became fixed, he was able to be self-sufficient and lead a normal way of life. It could have been precisely because of his curious external appearance that he was considered a "sacred" person, and buried in the area of the Pyramid Temple of Raneferef. Tuberculosis was not connected with his death.

Case number 2

ARCHEOLOGICAL BACKGROUND

In tomb no. 87 of cemetery K situated on the slope of the left Nile bank west of the fortified settlement at Nag'el-Scheima (area of recent Sayala, 130 km south of Aswan), an adult skeleton was lying stretched out in the supine position with head to the west and arms alongside the body. There were no funeral offerings in the tomb. Together with other burials of cemetery K, serving the common people of the fortified settlement, the tomb dates from the Christian period (6th–11th century A.D.).

EMBALMING METHOD

No traces of an attempt to preserve the body by mummification were found.

AGE

All cranial sutures were still open except for beginning of fusion in the lateral thirds of the coronal suture (C3). In the mostly preserved dentition (only upper right I2 and C and lower left I1 were missing and both upper I 1, upper left C and lower right I1 showed broken-off crowns) we found fully erupted third molars and only the beginnings of attrition of the dentine (points) in most of the teeth. At the same time, both lower M2 were already lost and their alveoli closed. The crown of the lower right M1 was devoured by a caries leaving in situ only roots surrounded by a periapical abscess cavity. Both lower M3 showed occlusal caries, greater right (diameter 3 mm) than left (diameter 1 mm). Premature incidence of caries, already beginning in deciduous dentition, is a common feature of the population of Sayala during the Christian period. Resorption of the alveolar process was only slight according to the scale of Brothwell (1963:150) with incipient atrophy of the alveolar process around both lost lower M2. Beginnings of deposition of dental calculus could be observed.

Most of the epiphyseal and apophyseal fissures were completely closed, except between bodies S1 and S2 of the sacrum and on the medial ends of the clavicles. Moreover, traces of recent epiphyseal or apophyseal fusion could be detected on terminal plates of the cervical vertebrae, between other sacral bodies, along the cristal and ischiatic apophyses, on humeral and femoral heads as well as on the distal ends of radii, ulnae and fibulae. The pubic symphysis showed phase 3 of Todd (1920) having the range of 22–24 years. Parts of the sternum were not fused. No age-dependent changes could be found on the proximal ends of both humeri and femora, except some rugged relief medially on the crista tuberculi maoris, caused by muscular action. There was no arthritis, no patellar and only beginning calcaneal osteophytes. On vertebral bodies whose margins were not eroded by the pathological process to be described (C1–T4, L2–5, S1) no lipping was present except slight beginnings on C4 and C5. Also the intervertebral joints were intact except in the region mostly involved in the pathology, where an arthrosis of T9–10 and synostosis T10–11 developed.

The described features agree with a young adult age of 22–24 years.

SEX

In spite of an only slightly developed glabella (Broca 2) and supraorbital arches (Eickstedt 2) as well as a nasofrontal transition in an only slightly concave arch and a very feeble protuberantia occipitalis externa (Broca 1), other features pointed to the male side. There was a smoothly arched forehead, a thick and long mastoid process, a small and shallow incisura mastoidea, situated within the mass of the process, a moderately thick orbital margin, a slightly to moderately developed processus marginalis, a slightly to moderately developed nuchal, but a medium to strongly marked mandibular muscular relief with a large eversion of the mandibular angle. Also moderately large pelvic apophyses, a deep and narrow ischiatic notch, absence of a preauricular sulcus, an oval, moderately large pelvic inlet, an outstanding tuberculum pubicum, a flaring lower aspect of the pubic bone, a rather acute subpubic angle (60°) and a low ischiopubic index (left 64.0, right 63.6, according to Thieme and Schull 1957:269) argued unequivocally for the male sex.

GENERAL PHYSICAL FEATURES

The body build was moderately robust to robust with well-developed muscular insertions. Stature, reconstructed according to tables of Trotter and Gleser (1952) for American Negroes, was medium (166.1 cm).

SPINAL PATHOLOGY

In contrast with case no. 1, the whole thoracic and lumbar spine showed a lytic process with inhibition of new bone regeneration, progressing from above downward in the thoracic and from below upward in the lumbar section with maximum changes in T10–L1, resulting in a double angular kyphosis (Figures 8–13). Multiple small, larger, and big confluent cavities were characteristically localized on the circumferential aspects of the vertebral bodies, portraying the hematogenous spread of the infection via the paravertebral plexus and anterior longitudinal ligament (Zimmerman and Kelley 1982:105).

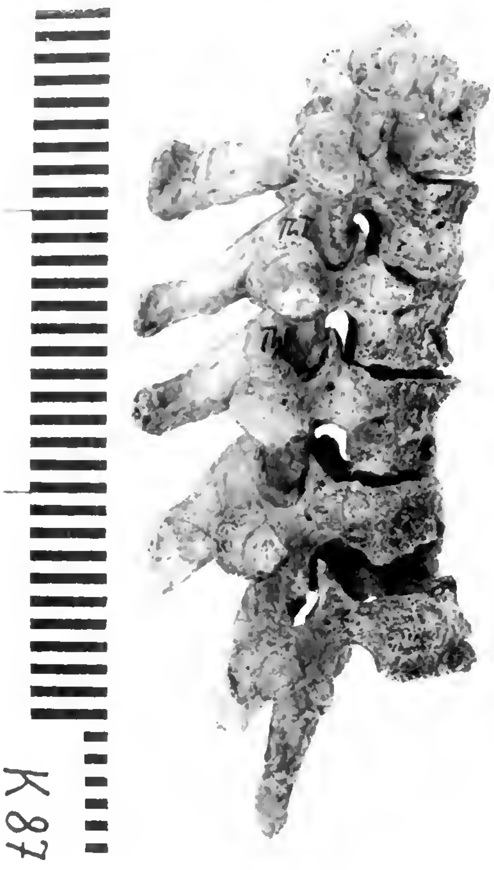
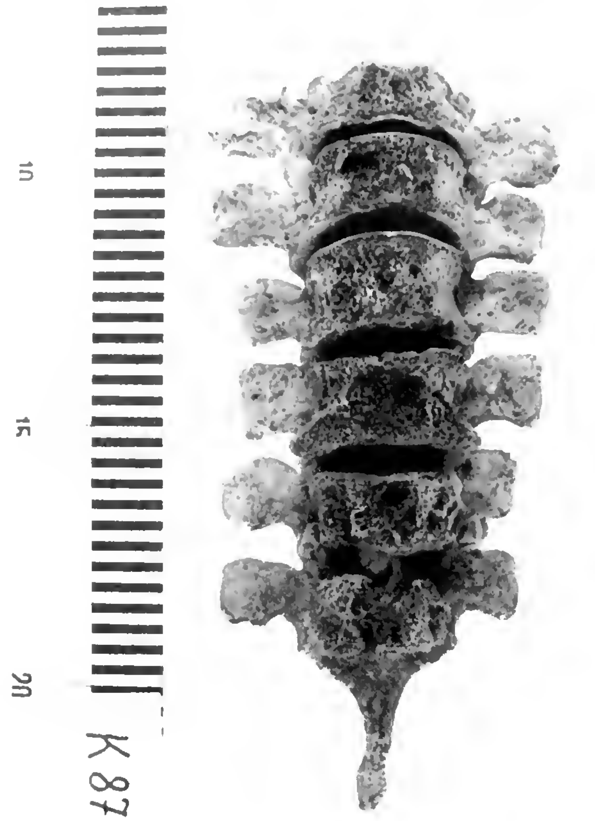


FIGURE 8. Case no. 2. Upper thoracic spine with lytic changes on vertebral bodies, right lateral view.

The cervical spine was devoid of pathological changes. The upper third of the thoracic spine showed eroded pits on the vertebral bodies, whose original shape had been mostly preserved. In the middle part of the thoracic spine the destruction by confluent foci reached such an extent that only stumps of vertebral bodies remained. They had bizarre forms of columns bordering deep cavities and perforations. The process did not penetrate into the neural arch and no changes were present on intervertebral joints.

The lower part of the thoracic spine was maximally afflicted by the pathology. The lytic process removed the whole body of vertebra T10 and eroded the anterior parts of the pediculi arcus vertebrae. The anteroposterior axis of the vertebra had been rotated 70°, and consequently the spinous process of T9 became the salient point of an angular kyphosis (90°). Intervertebral joints T9–10 were enlarged and eroded



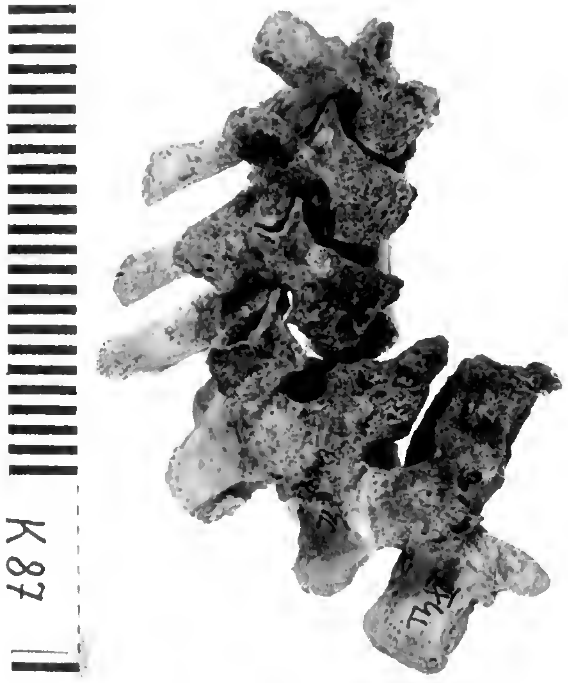


FIGURE 10. Case no. 2. Lower thoracic spine with lytic changes and angular deformity of T10–12, right lateral view.

FIGURE 9 *(left)*. Case no. 2. Upper thoracic spine with lytic changes on vertebral bodies, frontal view.

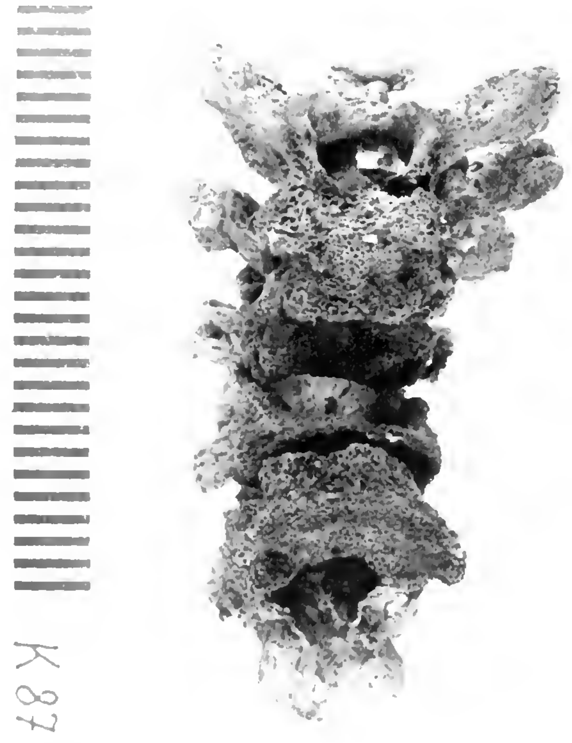


FIGURE 11. Case no. 2. Lower thoracic spine with lytic changes, completely destroyed body T10, and wedge-shaped body T11, frontal view.

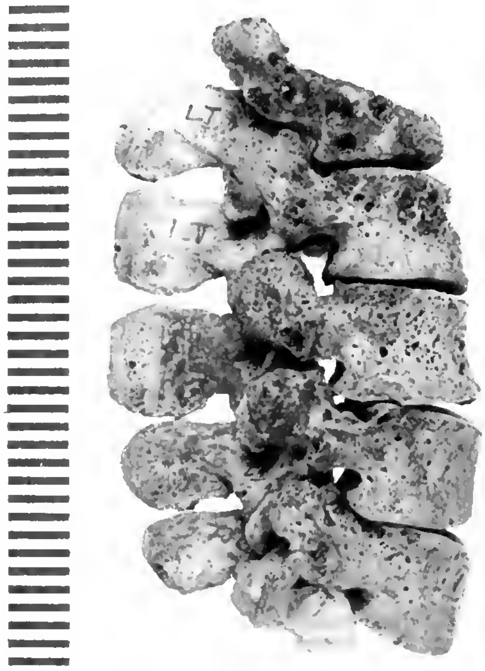


FIGURE 12. Case no. 2. Lumbar spine with wedge-shaped L1, lytic cavities in body L2, and more or less pitting in bodies L3–5, right lateral view.

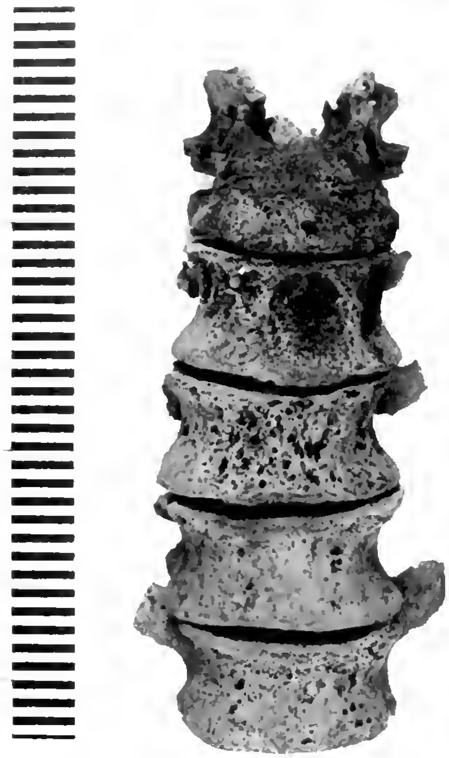


FIGURE 13. Case no. 2. Lumbar spine with wedge-shaped L1, lytic cavities in body L2, and more or less pitting in bodies L3–5, frontal view.

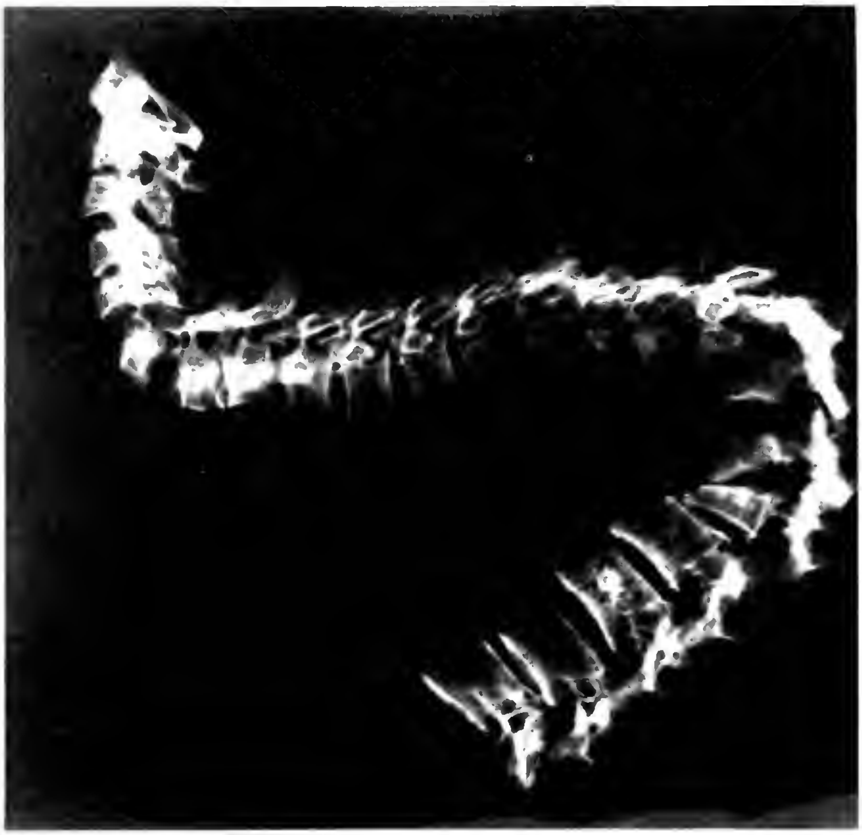


FIGURE 14. Case no. 2. Radiogram of spine showing lytic changes and double angular kyphosis, which together equals 140°, lateral projection.

(spondylarthritis), while joints T10–11 were completely fused (synostosis). These changes were secondary to unusually heavy strain.

The wedge-shaped vertebra T11 (anterior height 4 mm, posterior height 20 mm) showed in its center a large abscess cavity, bordered by lateral pillars of the remaining spongiosa, thicker right (8–11 mm) than left (5–6 mm). The body of this vertebra filled the empty space in the place of the destroyed body T10 by rotation of the anteroposterior axis of the vertebra including 90° with the axis of T9. Articular facets of the intervertebral joint T11–12 were enlarged but not eroded.

Vertebra T12 retained its original shape, but its whole surface had been eroded by several large and partly merging cavities. Small saucer-shaped erosions penetrated also into both pediculi arcus vertebrae. Intervertebral joints T12–L1 did not show any change.

Another wedge-shaped vertebra L1 (anterior height 3 mm, posterior height 21 mm) revealed that the lytic process continued after the evacuation of the abscess and healing of the pathologically fractured vertebral body. There were several small or medium cavities and erosions on its surface (upper, right lateral, and lower margin). Even both pediculi arcus vertebrae were perforated from above (5 × 7 mm) downward (1 × 1 mm) and the anterolateral side of the right processus articularis superior showed two tiny "borings." Owing to the wedge shape of L1, its spinous process was the top of another angular kyphosis of the spine (50°).

Vertebra L2 retained its original shape, but several large cavities penetrated its surface (anteriorly left of the midline, on the left and right anterolateral aspect) together with smaller pits and erosions.

The following vertebrae L3–5 showed a decreasing quantity of pitting, on a few places merging into larger defects. Here we could observe well the progress of the described process. All lumbar intervertebral joints were intact.

The axis of the spine deviated in the sense of a dextroconvex scoliosis of the lower thoracic section. More important, however, was the double kyphosis in the thoracolumbar transition reaching collectively an incredible 140° angle. The deformation of the vertebral column can be seen on the radiogram (Figure 14) together with the extent of erosion and destruction of the individual vertebrae. Because of absence of a compensatory growth of the vertebrae in the sense of an adaptive lordosis, the thoracic spine starting at the thoracolumbar bend in vivo was sinking from above backward to downward and forward in a very oblique position.

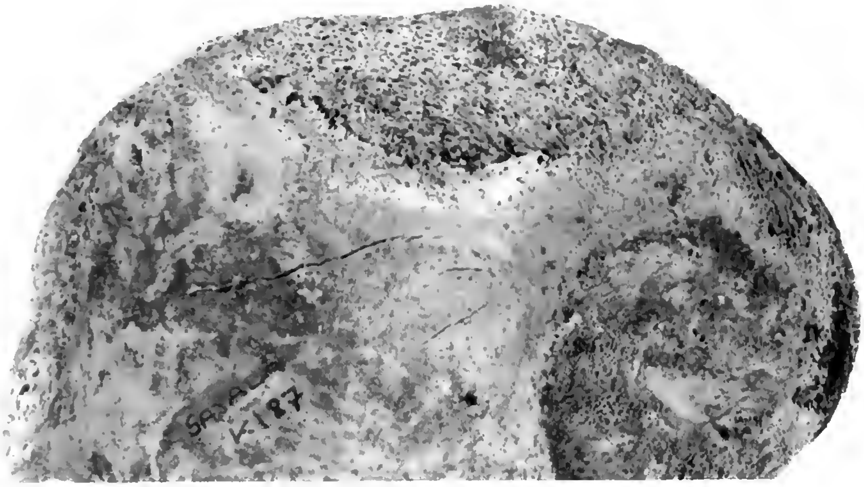


FIGURE 15. Case no. 2. Eroded area with new bone overgrowth on inner side of right iliac wing, frontal view.

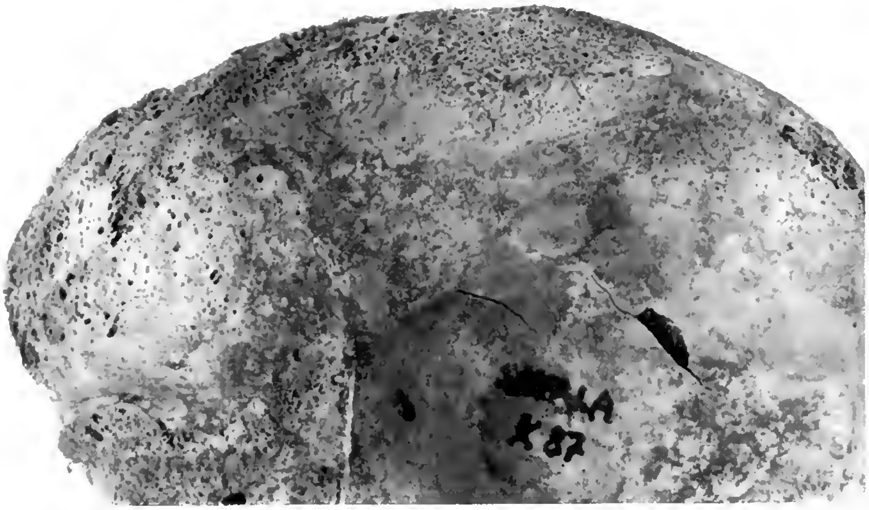


FIGURE 16. Case no. 2. Eroded area with new bone overgrowth on inner side of left iliac wing, frontal view.

The deformed thorax and the abdomen were very much compressed into the narrow space between the lumbar and thoracic spine, making the function of internal organs extremely difficult. The only possibility to maintain the quasi-horizontal position of the head was an exaggerated cervical and atlanto-occipital hyperlordosis.

DEMARCATED ILIAC PERIOSTITIS

Unusual findings on the upper parts of the inner aspects of both wings of the iliac bones were almost certainly connected with the described spinal pathology. There were crescent-shaped segments of eroded compact bone, revealing the thick spongiotic bone. On the lower borders of the erosions and in a few places inside the erosions there were spiculae, lippings and islets of newly formed, compact bone (Figures 15,16).

On the right iliac bone the defect began on the crista iliaca 67 mm anterior to the spina iliaca posterior superior and extended with a caudally convex border (maximum distance from the crista iliaca 27 mm) to a point 84 mm posterior to the spina iliaca anterior superior. The erosion encompassed also the crista iliaca between the points mentioned.

On the left iliac bone the extent of the defect was narrower and shorter than on the right one. It began about 70 mm anterior to the spina iliaca posterior superior and extended with a wavy, inferior border (maximum distance from the crista iliaca 19 mm) to a point 95 mm posterior to the spina iliaca anterior superior.

The described changes may be considered reactive to temporary adherent "cold" abscesses, descending paravertebrally on the posterior side of the large and small psoas muscles.

DIAGNOSIS

Comparing characteristic features of tuberculosis in case no. 2 with case no. 1 (Table 1), we find differences in more than half of them. Case no. 1 findings indicated a long duration with prevalence of adaptive changes and healing. In contrast to this, case no. 2 showed a relatively short-term and active, predominantly lytic process with very limited healing.

At the same time, with half of the features there seems to be a disagreement with the usual diagnostic set for tuberculosis. This applies first to the extraordinarily great extent of the process, involving altogether 17 vertebrae with major changes in four of them. From the recent literature two cases with similarly elevated numbers of afflicted vertebrae can be quoted. The first one from Oberweil bei Buhren a.d. Aare (Switzerland), dated 7th–8th century A.D., was that of an 18–23-year-old woman afflicted by a destructive-reparative process involving 13 vertebrae, C7 and all thoracic with maximum changes between T4 and T6 (Ulrich-Bochsler et al. 1982:1318–1319). A juvenile male aged about 15 years from Arene Candide Cave (Liguria, Italy), dated beginning of the 4th millennium B.C., showed resorptive lesions in eight vertebrae (T9–L4) with a maximum of destruction between T10 and L1 (Formicola et al. 1987:3). Cases with a great extent of the process most probably reflect the great virulence of infection and the limited resistance of the host.

In agreement with that we found the uncommon involvement of anterior sections of the neural arch (pedicles and articular processes) which characterizes very severe cases (Zimmerman and Kelley 1982:105).

Because the process was relatively rapid and destruction dominated production of new bone, there was not enough time and resistance for the development of fused vertebral bodies, ossification of interspinous or lateral longitudinal ligaments, apposition of new-formed bone, or fusion of costovertebral joints. Steady progression of the destruction over healing was revealed especially in L1 whose "healed" wedge shape was eroded anew by suppuration.

The process did not narrow the neural canal, but did involve a single intervertebral opening. Absence of compensatory growth of vertebrae was connected with the later beginning of the disease and lack of emaciation together with its relatively short duration.

COURSE OF THE DISEASE

Our young man caught the disease after the end of his growth period, since otherwise adaptive increase of anterior heights of vertebrae would have appeared. With regard to his age at death, there is a span of about seven to nine years in which the process could occur. It seems, however, probable that it lasted for a far shorter period. Most probably the body of T10 was first destroyed, later an abscess involved the left L1, and finally another abscess was evacuated from T11, leaving there remnants of its cavity. More than a single extension into psoas abscesses is attested by the bilateral development of the demarcated iliac periostitis. The patient did not develop enough resistance and the infection spread both in the cranial and caudal directions gradually involving more and more vertebrae.

Due to the severity of the infection and the extreme grade of deformation of the trunk we may be almost sure that the patient died as the result of it or from some of its complications. The mortality of tuberculosis in the preantibiotic era was 50% and most often occurred in the first two decades of life. Today it is about 5% and confined to an adult or senile age (Ulrich-Bochsler 1982:1322).

DIFFERENTIAL DIAGNOSIS

The absence of pathological changes in the remaining skeleton, revealed macroscopically and by the x-rays, as well as the evidence of the psoas abscesses are strong arguments against other diagnostic possibilities (Steinbock 1976:176, 179). The multiplicity of lytic foci in such a great number of vertebral bodies could be produced by blastomycosis, but in it the lesions are almost only destructive, with a punched-out appearance and with little new bone growth. It involves also neural arches and, beside the spine, affects ribs and other bones (Zimmerman and Kelley 1982:89). In pyogenic osteomyelitis of the spine such a great extent and massive destruction of several vertebral bodies leading to an angular kyphosis and paravertebral abscesses are uncommon (Ortner and Putschar 1981:149). The pus, penetrating bone cortex, leads to new bone formation with irregular thickening and cloacae in the involucrum (Zimmerman and Kelley 1982:93–94).

SOCIAL IMPLICATIONS

The rapid progression and severity of the process forced the patient to lie in his bed and be nursed by his family or neighbors. The exaggerated deformity of the spine with extreme compression of internal organs prevented standing, walking and working. He became fully dependent on social support. This state did not last, however, for a long time. The short course of the disease is reflected in the preservation of his relatively robust and muscular body.

Literary data on vertebral tuberculosis in Egypt and Nubia

Since it is a disease of at least neolithic antiquity, associated with drinking milk of cows infected by the bovine type of *Mycobacterium tuberculosis*, from which the human type developed later by microevolution (Manchester 1983:39–40), the occurrence of tuberculosis in ancient Egypt and Nubia may well be expected. Milking there became very important as expressed in the widespread cult of the cow-goddess Hathor, and there was a high population density in crowded villages and towns which enabled close contact of people and easy transmission of the agent.

Three different sources of information, scattered in the literature, may be consulted for a survey of the presence of vertebral tuberculosis: iconography, excavated skeletal remains, and mummies.

ICONOGRAPHY

Several statues and drawings of hunchbacks may be found in Egyptian art (Morse et al. 1964:525–528; Morse 1967:263; Baud 1978:pl.16). Their evidence is, however, ambiguous. Diagnosis of vertebral tuberculosis can be based only on the shape of the kyphosis. If it is angular, the probability of tuberculous origin is great. If it is arched, other diseases such as rachitis, Scheuermann's disease, osteoporosis, and bad body posture also must be taken into account.

Better information may be gained in plastic art. The diagnosis of vertebral tuberculosis is suggested in an allegedly predynastic clay statuette of an emaciated man with a thoracic angular kyphosis found inside a bowl (Morse et al. 1964:524–525), a possibly predynastic standing, ivory figure showing protrusion of the back and chest (Morse et al. 1964:525), a probably Archaic statuette of a squatting, bearded man with a thoracic angular protuberance and an angular protrusion of the chest (Jonckheere 1948), and a wooden statue from the Cairo Museum with an angular kyphosis on the transition between upper and middle third of the thoracic spine (Ghalioungui and Dawakhly 1965:20, fig.64).

In drawings, evaluation is more complicated, because the hunchback can also be factitious due to artistic ineptitude and stylistic convention. In Egyptian pictures, human shoulders traditionally were represented in frontal view and if the artist failed to place one of the arms correctly the protruding shoulder may resemble a strongly arched hunch, situated high on the spine. This can be the case of the representation of the "deformed" gardener from the New Kingdom tomb of Ipuy (Davies 1927:pl.XXIX), of the relief of the priest Ankhutus from the false door of his Old Kingdom tomb (Mogensen 1930:90,pl.XCV), as well as of the relief of a servant leading two dogs in the Ti's Vth dynasty tomb (Steindorff 1913: pl.115). Only the relief of a serving girl from tomb no. 45 at Giza, dated IVth dynasty, shows a strongly arched thoracic protuberance (Vandier 1964:fig.2), and the painting of an attendant from a XIIth dynasty tomb at Beni Hasan demonstrated a protruding, arched hump at the cervicothoracic transition, both suggesting tuberculous origin.

SKELETAL REMAINS

Altogether, 30 cases of vertebral tuberculosis were gathered in the last reviews of tuberculosis in ancient Egypt by Morse et al. (1964:529–540) and Morse (1967:263–268). There are 13 cases from the Upper Egyptian site at Nagada with "typical pathological changes to be considered as tuberculosis and seven of them . . . quite typical" (Morse 1967:268). Unfortunately, their dating is problematic. While some are predynastic (B 107, T 52), others may come from dynastic times up to 1400 B.C. In six cases (B 107, 60, 1003, D 5, R, and Q) the diagnosis of tuberculosis seems to be less probable, and other causes (such as acute osteomyelitis or healed compressed fracture) were also taken into consideration (Morse et al. 1964:535–539).

Of the nine cases described by Derry (1938:1) and summarized in a table by Morse (1967:264) only two were from Egypt, one found in an aged woman from Saqqara dated as early as 3300 B.C., and the other in an old woman from Deir el-Bahri dated 1500 B.C. Seven other cases come from Nubia, most of which are dated 3000 B.C., and in case no. 1, 2000 B.C. Four adult men, two adult women and a nine-year-old boy were afflicted. Epidemiologically interesting are two graves, each containing remains of two tuberculous individuals (a man with a woman in tomb no. 314, and a man with a boy in tomb no. 452).

A series of six unfortunately isolated Nubian vertebral specimens were described by Morse et al. (1964:531–534) and summarized by Morse (1967:265). One of them listed under catalog no. 182 E, together with vertebrae of two other persons, is identical with Derry's case no. 1, being a lumbar vertebra with a large smoothly delimited cavity more suggestive of a bone cyst or osteolytic neoplasm (Morse et al. 1964:533,fig.9 above right) and should be excluded.

Another case of vertebral tuberculosis was described by Smith (1927:24) and quoted by Morse et al. (1964:534), but insufficiently quoted by Morse (1967:265,267). In the incomplete remains of Pa-Ra'messu, the son of Seti I and brother of Ramesses II, aged 26–30 years, a mass consisting of six thoracic vertebrae (probably T4–9) with parts of corresponding ribs was found, all firmly ankylosed together. The bodies of the middle vertebrae had collapsed causing a right-angle bend between probably T6–7. Smith associated with this process the slenderness and gracility of the left leg compared with the right one.

The case described by Watermann (1960:170–171) has been misquoted by Morse et al. (1964:531) and Morse (1967:264) as originating at Giza and excavated by Abu Bakr. In reality it was found in a prehistoric grave and stored in the Museum of Helwan. Two fused sections of the spine were apparent, the first between T4 and T7 with wedge-shaped bodies and another between L1 and L5 with a wedge-shaped body L3. In both areas neural arches were massively involved, making the diagnosis of tuberculosis somewhat doubtful (Morse et al. 1964:531).

To this survey we may add another case of a middle-aged male found in shaft no. 42 of the uninscribed mastaba B near the Pyramids of Giza with wedge-shaped vertebrae T10 and

T12 and less compressed T11. In spite of the expressed diagnosis of a "chronic rarefaction" leading to "gradual compression of the . . . vertebrae" (Abadir 1953:85), tuberculous origin of the condition cannot be excluded as well.

MUMMIES

Notwithstanding the fact that large series of Egyptian mummies have been subjected to radiological and other scientific studies in the two recent decades, cases with vertebral tuberculosis remain rare. There is the often-quoted hunchback priest of the god Amun called Nesperehan, detected among 44 well-preserved mummies found by Grebart in 1891 (Smith and Ruffer 1910; Smith and Dawson 1924:156, fig.62; Cave 1939:142). His mummy shows destruction of the lower thoracic and upper lumbar vertebrae causing an angular kyphosis and a large abscess cavity in the area of the right psoas muscle.

In the most probably natural mummy of a 5-year-old child from an intrusive burial (1000–400 B.C.) found in the tomb of Nebwenenef at Thebes, pulmonary and vertebral tuberculosis ending with a fatal hemorrhage was described and tuberculous bacilli proved in the vertebral bones (Zimmerman 1979).

Discussion

Together with our two cases, 6 iconographic, 30 skeletal, and 2 mummy specimens attest with lesser or greater probability the presence of vertebral tuberculosis in ancient Egypt and Nubia. Of the skeletal cases, 19 were from Egypt and 13 from Nubia with no particular preference for either of the two countries. Unfortunately it is not possible to guess how many vertebral columns were examined by anthropologists experienced in pathology and what was the incidence of the disease. The accumulated number of cases discloses, however, that it was by no means negligible.

Our case no. 1 comes from an isolated Middle Kingdom tomb inserted in the ruins of an Old Kingdom temple at Abusir. No data on the frequency of the disease, therefore, can be given.

Our case no. 2 was found in the Christian cemetery at Sayala. The total number of identified individuals was 161. In no other individual of this group could we detect changes pointing to tuberculosis. Thus a rough estimate of incidence would be 0.62%. Because vertebral tuberculosis accounts for 1–3.5% of the incidence of tuberculosis in all its forms, this would mean that 18–62% of people could have been infected by tuberculosis at Sayala. Together with other unknown causes this could explain the extremely low mean age at death of its Christian population (about 18 years).

The scarcity of findings of vertebral tuberculosis in artificially mummified bodies (representing members of the high social stratum that could afford costs of mummification) could well reflect social differences in the incidence of tuberculosis. However, if we consider that some of the skeletal remains could represent disintegrated mummies (as our case no. 1 and possibly also the remains of the royal son Pa-Ra'messu), there would be 3 cases out of the total of 32 belonging to the royal and priestly society. The incidence (9.4%) does not seem to be significantly smaller than the supposed percentage share of the high stratum in the ancient Egyptian society.

In the present state of our knowledge these reflections can be no more than preliminary. Much research should be performed in the future, aiming at establishing reliable data on the incidence of vertebral tuberculosis, the excellent marker for studies of tuberculous infection in different periods, regions, and social layers. They are especially important for reconstruction of the social history of ancient Egypt and Nubia.

Literature cited

Abadir, F. 1953. Skeleton Found in Shaft No. 42 of the Uninscribed Mastaba "B." In A.M. Abu-Bakr, ed., *Excavations at Giza 1949–1950*, 85. Cairo: Government Press.

Allison, M.J., D. Mendoza, and A. Pezzia. 1973. Documentation of a Case of Tuberculosis in Pre-Columbian America. *American Review of Respiratory Diseases*, 107:985–991.

Brothwell, D.R. 1963. *Digging Up Bones*. London: British Museum (Natural History).

Baud, M. 1978. *Le Caractère du Dessin en Egypte Ancienne*. Paris: Librairie d'Amérique et d'Orient.

Cave, E.J.E. 1939. The Evidence for the Incidence of Tuberculosis in Ancient Egypt. *British Journal of Tuberculosis*, 33:142.

Davies, N. de G. 1927. *Two Ramesside Tombs at Thebes*. Robb de Peyster Tytus Memorial Series, vol. 5. New York: Metropolitan Museum of Art.

Derry, D.E. 1938. Pott's Disease in Ancient Egypt. *Medical Press*, 197:1.

Formicola, V., Q. Milanesi, and C. Scarsini. 1987. Evidence of Spinal Tuberculosis at the Beginning of the Fourth Millennium B.C. from Arene Candide. *American Journal of Physical Anthropology*, 72:1–6.

Ghalioungui, P., and Z. Dawakhly. 1965. *Health and Healing in Ancient Egypt*. Cairo: Dar Al-Maaref.

Grmek, M.D. 1983. *Les Maladies à l'Aube de la Civilisation Occidentale*. Paris: Payot.

Haneveld, G.T. 1980. Pott's Disease before Pott. *The Netherlands Journal of Surgery*, 32:2–7.

Jonckheere, F. 1948. Le Bossu des Musees Royaux d'Art et d'Histoire de Bruxelles. *Chronique d'Egypte*, 45–46:24–35.

Manchester, K. 1983. *The Archaeology of Disease*. Bradford, U.K.: University of Bradford Press.

Mogensen, M. 1930. *La Glyptotheque Ny Carlsberg*. La Collection Egyptienne. Copenhagen: Levin and Munksgaard.

Morse, D. 1967. Tuberculosis. In D. Brothwell and A.T. Sandison, eds., *Diseases in Antiquity*, 249–271. Springfield, Ill.: Charles C Thomas.

Morse, D., D.R. Brothwell, and P.J. Ucko. 1964. Tuberculosis in Ancient Egypt. *The American Review of Respiratory Diseases,* 90:524–541.

Ortner, D.J., and W.G.J. Putschar. 1981. Identification of Pathological Conditions in Human Skeletal Remains. *Smithsonian Contributions to Anthropology.* 28. Washington, D.C.: Smithsonian Institution Press.

Pott, P. 1790. Remarks on that Kind of Palsy of the Lower Limbs which is Frequently Found to Accompany a Curvature of the Spine. *The Chirurgical Works of P. Pott,* vol. 3, 409–443. London.

Robins, G. 1983. Natural and Canonical Proportions in Ancient Egyptians. *Gottinger Miszellen,* 61:17–25.

Smith, G.E. 1927. Report on the Bones Found in the Sarcophagus of PaRa'messu (Tomb 5). In R. Engelbach, ed., *Gurob,* 24. London: British School of Archaeology and Quaritch.

Smith, G.E., and W.R. Dawson. 1924. *Egyptian Mummies.* London: Allen and Unwin.

Smith, G.E., and M.A. Ruffer. 1910. Pottsche Krankheit an einer aegyptischen Mumie aus der Zeit der 21 Dynastie. In K. Sudhoff, ed., *Zur historischen Biologie der Krankheitserreger,* vol. 3, 9–16. Leipzig, GDR.

Steinbock, R.T. 1976. *Paleopathological Diagnosis and Interpretation.* Springfield, Ill.: Charles C Thomas.

Steindorff, G. 1913. *Das Grab des Ti,* vol. 2. Leipzig, GDR: J.C. Hinrichs'sche Buchhandlung.

Strouhal, E. 1986. Embalming Excerebration in the Middle Kingdom. In A.R. David, ed., *Science in Egyptology: Proceedings of the Science in Egyptology Symposia,* 141–154. Manchester, U.K.: Manchester University Press.

Strouhal, E., and J. Jungwirth. 1984. *Die anthropologische Untersuchung der C-Gruppen- und Pan-Graber-Skelette aus Sayala, Aegyptisch-Nubien.* Osterreichische Akademie der Wissenschaften, phil.-hist. Klasse, Denkschriften, 176. Band. Vienna: Verlag der Oesterreichischen Akademie der Wissenschaften.

Thieme, F.P., and W. Schull. 1957. Sex Determination from the Skeleton. *Human Biology,* 29:242–273.

Todd, T.W. 1920. Age Change in the Pubic Bone. *American Journal of Physical Anthropology,* 3:285–334.

Trotter, M., and G.C. Gleser. 1952. Estimation of Stature from Long Bones of American Whites and Negroes. *American Journal of Physical Anthropology,* 10:463–514.

Ulrich-Bochsler, S., E. Schaublin, T.B. Zeltner, and G. Glowatzki. 1982. Invalidisierende Wirbelsauleverkrummung an einem Skelettfund aus dem Fruehmittelalter (7./8. bis Anf.9. Jhr). Ein Fall einer wahrscheinlichen Spondylitis tuberculosa. *Schweizerische Medizinische Wochenschrift,* 112:1318–1323.

Vandier, J. 1964. *Manuel d'Archeologie Egyptienne.* Tome IV. Bas Reliefs et Peintures. Scenes de la Vie Quotidienne. Paris: Picard.

Watermann, R. 1960. Palaeopathologische Beobachtungen an altaegyptischen Skeletten und Mumien. *Homo,* 11:167–179.

Zimmerman, M.R. 1977. The Mummies of the Tomb of Nebwenenef: Paleopathology and Archeology. *American Research Center in Egypt,* 14:33–36.

______. 1979. Pulmonary and Osseous Tuberculosis in an Egyptian Mummy. *Bulletin of the New York Academy of Medicine,* 55: 604–608.

Zimmerman, M.R., and M.A. Kelley. 1982. *Atlas of Human Paleopathology.* New York: Praeger.

SUMMARY OF AUDIENCE DISCUSSION: These two cases each demonstrate a very large diseased area involving 8 to 10 vertebrae. In the absence of vertebral body destruction a mycotic etiology might be considered. These, however, are rare in Egypt and the second case does show two foci of vertebral body destruction. The bilateral nature of sacral involvement is unusual, yet cannot be explained as a nontuberculous, mechanical abrasion.

Leprosy

Leprosy and tuberculosis in the Byzantine monasteries of the Judean Desert

Joseph Zias

In his classic work of 1911 on biblical medicine, the German physician J. Preuss noted that there were nearly as many books and treatises on the subject of biblical leprosy as there were on circumcision in the Bible (Preuss 1978). However, despite the plethora of written information on this ancient disease which, according to literary sources, existed as early as 600 B.C., scant archeological evidence of leprosy exists prior to A.D. 1200. In fact, prior to the publication of Preuss's encyclopedic work, there had been only one find from the postbiblical period (A.D. 500) in Nubia (Smith and Dawson 1924). Even today, despite the intense worldwide activities of archeologists and physical anthropologists, no indisputable physical evidence of the disease has been found, with the exception of isolated instances in the United Kingdom, North Africa, and the cemeteries of medieval Danish leprosaria. In Israel, where evidence of man's presence is over one million years old, and where thousands of skeletal remains have been uncovered, evidence of leprosy has eluded us. In fact, S.G. Browne noted in 1975 that not a single case had ever been found in all of the Fertile Crescent.

In the spring of 1983, excavations were carried out at the Judean Desert monastery of Martyrius, which had been founded in the fifth century and destroyed by the Persians in A.D. 614, never to be rebuilt. Here a common grave was discovered in an ancient church floor, dated by inscription to 492. The grave contained the skeletal remains of nine men and one woman, four of whom showed bilateral mutilating changes on the hands and feet which were originally thought to be associated with leprosy (Figure 1). Subsequent study of the material by the author with American and European specialists in paleopathology showed that our original diagnosis of leprosy was incorrect. Despite the widespread involvement of the small bones of the extremities with numerous lytic destructive foci, the concentric absorption of the phalanges, metatarsal and metacarpal bones, which is common in leprosy, was absent. Moreover, three individuals showed ankylosis of the vertebral column and the sacroiliac joint, which is not usually associated with leprosy. The widespread skeletal involvement of the disease with varying degrees of architectural destruction suggests that psoriatic arthritis is the preferred clinical diagnosis rather than leprosy as was earlier reported (Zias

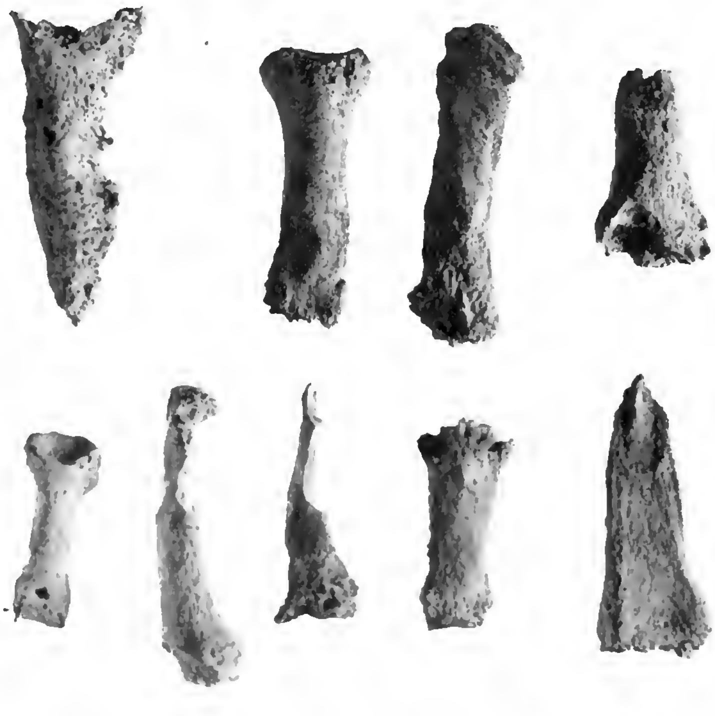


FIGURE 1. Phalanges of the hand. (Courtesy of Israel Antiquities Authority, T. Sagiv)

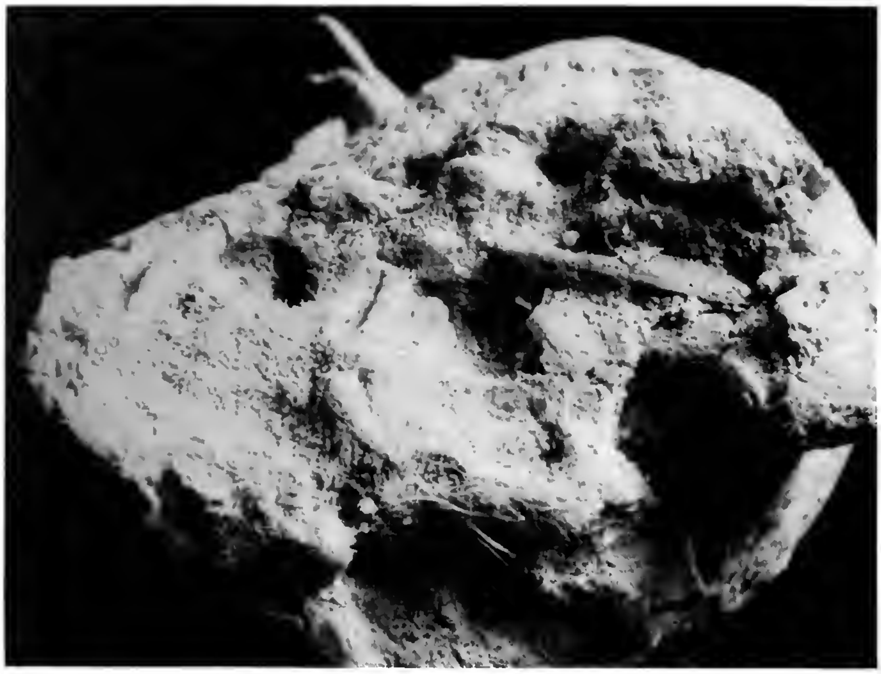


FIGURE 2. Hyena feces showing textiles and human bone. (Courtesy of Department of Antiquities, Ministry of Education and Culture, State of Israel—T. Sagiv)

1985). Since psoriatic arthritis affects both the skeleton and the skin of the individual, it mimics to a certain extent those changes usually associated with leprosy, which may have been the reason for inclusion in the Byzantine monastery. Literary evidence from the Mediterranean area indicates that the Early Church established hospitals for the care of those suffering from leprosy as early as the fourth century A.D. (Avi-Yona 1963). Therefore it seems entirely plausible that these four individuals may have been mistaken as suffering from leprosy and sought refuge in the desert monastery of Saint Martyrius.

Following this discovery, we obtained permission from the Greek Patriarch to survey the few Byzantine monasteries possessing skeletal collections dating from the Persian massacres of A.D. 614. This survey, however, did provide evidence of leprosy in the Holy Land during the seventh century and earlier.

In the summer of 1983 we were notified of the accidental discovery of a mass grave at the Monastery of John the Baptist near the Jordan River where, according to Christian tradition, the "washing of the lepers" took place (Hoade 1981). Most of the grave had been destroyed by a bulldozer; however, we managed to retrieve 34 skeletons which were dated by ^{14}C to approximately A.D. 600. Owing to the unique environmental conditions (400 meters below sea level, minimal humidity and rainfall), preservation was excellent. One interesting aspect of this find is that some of the burial customs, such as placing seeds from the tree *Balanites aegyptiaca* in the hands of the deceased, conform to ancient Egyptian traditions, suggesting that these individuals had traveled for some considerable distance prior to their deaths. Among the human remains were feces (subsequently identified as those of a hyena) which contained fragments of human hair, bone, and remnants of the same cloth found in the grave, suggesting that those buried here had been killed in the Persian massacre of A.D. 614 and had been buried only hours or days later (Figure 2).

Anthropological analysis of the skeletal material showed those specific destructive and erosive changes known to be caused by *Mycobacterium leprae* in the hands, feet and lower extremities. Evidence of tuberculosis, in the form of pleural calcification, was also noted in this collection (Figure 3).

The appearance of tuberculosis and leprosy, both of which are rare in the Holy Land, raises important questions about this site. Although hospitals were frequently associated with monasteries, no literary evidence from this period attests to the existence of a hospital at the Monastery of Saint John. However, the high incidence of pathological material found here suggests that those buried here were seriously ill, perhaps members of a hospital population who may have been banished to the desert. This tradition of banishment is apparently an ancient one, in that the first archeological evidence of leprosy comes from the Dakleh oasis in the Egyptian desert. Here, interred in a Sudanese cemetery dated to about 200 B.C., were the remains of four Caucasian males, all of whom showed stigmata of leprosy (Dzierzykray-Rogalski 1980).

Further survey and research in the Judean Desert monasteries, which predate the Danish leprosaria by some 600 years, should provide an answer to the much-debated question of which diseases in antiquity were included in the generic category leprosy. For example, talmudic evidence of the postbiblical and Byzantine periods suggests that severe nasal disfigurement was considered one of the stigmata of leprosy (Rosen 1982); at this site, 2 of the 34 skeletons showed nasal abnormalities characteristic of neither tuberculosis nor leprosy. The cotton textiles recovered from this site may also shed light on whether those buried here, apparently seriously ill and banished to the desert, had been required to wear distinctive clothing indicative of their health status.

Finally, one of the more intriguing medical questions that may be answered by further study of these monas-

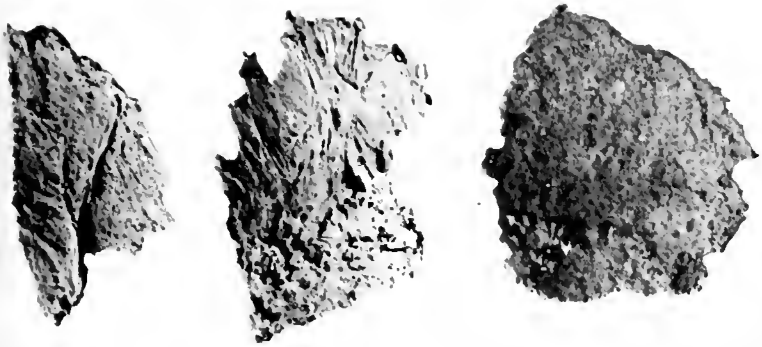


FIGURE 3. Calcified pleura. (Courtesy of Israel Department of Antiquities and Museums—T. Sagiv)

tic sites is that of the paleoepidemiology of leprosy and tuberculosis. Since, apart from this find, tuberculosis and leprosy have rarely been found in the Holy Land, these patients may have come from abroad or may have immigrated while incubating these diseases. One wonders, therefore, what factors may have operated to restrict the diseases to these desert communities. Was social and religious separation between the various communities so complete as to protect the members of one community from a disease prevalent in another? Did those who traveled great distances do so for spiritual reasons, or had they been banished from their home communities? According to Judaic and Christian literary sources of that period, those suffering from these diseases were ostracized. Perhaps they were welcomed by these desert communities, thus explaining the lack of such remains elsewhere in the Holy Land. Further excavations and other studies in the monasteries of the Judean Desert may well provide answers to questions such as those raised here.

Literature cited

Avi-Yona, M. 1963. The Bath of the Lepers at Scythopolis. *Israel Exploration Journal*, 13:325–326.

Browne, S. 1975. Some Aspects of the History of Leprosy: The Leprosy of Yesterday. *Proceedings of the Royal Society of Medicine*, 68.

Dzierzykray-Rogalski, T. 1980. Paleopathology of the Ptolemaic Inhabitants of Dakleh Oasis (Egypt). *Journal of Human Evolution*, 9:71–74.

Hoade, E. 1981. *Guide to the Holy Land*, 501–504. Jerusalem: Franciscan Press.

Preuss, J. 1978. *Biblical and Talmudic Medicine*. New York and London: Sanhedrin Press.

Rosen, Z. 1982. The Healthy and the Diseased Nose in the Bible. Proceedings of the First International Symposium on Medicine in the Bible. *Koroth*, 8:79–85.

Smith, G.E., and W.R. Dawson. 1924. *Egyptian Mummies*. London: Allen and Unwin.

Zias, J. 1985. Leprosy in the Byzantine Monasteries of the Judean Desert. *Koroth*, 9:242–248.

SUMMARY OF AUDIENCE DISCUSSION: The lesions on the anterior surface of the patella are considered occupational (monastery monk) because of their ancient and even modern habit of falling to their knees, rising, then kneeling again and continuing this process daily for hours on end. Since, during kneeling, the patella does not actually make contact with the ground, but suffers substantial stress during the lowering and raising of the body, the lesions may result from that stress. We also found lice, not so easily in the mummy's hair but very easily in the hair combs.

Evidence of disease in ancient Near Eastern texts: Leprosy in the Epilogue to the Code of Hammurapi?

Debra A. Chase

A great deal of caution must be exercised in the discussion of disease in ancient Mesopotamia. In part, this caution is due to the nature of the available sources which, scattered over many centuries, are often fragmentary and filled with lexical difficulties.[1] It is the duty of the philologist and student of the culture to interpret texts using etymologies, and literary and form critical analyses. This textual work is not complete, however, without input from the physical and social sciences (Biggs 1969:103). Over a quarter of a century ago Oppenheim (1962:107) noted that the study of Mesopotamian medicine had been hampered by separation between departments: its interpreters were "either philologists lacking knowledge of medicine, its history, or that of pharmacology, or physicians without adequate linguistic training." It is in the spirit of long-needed interdisciplinary cooperation that I offer for discussion this text from the Epilogue to the Code of Hammurapi, which, I believe, presents a more complete picture of the clinical manifestations of leprosy than any other text from ancient Mesopotamia proposed to date.[2]

Before examination of the passage let me give a brief synopsis of the use of the term in the translations of textual data from Mesopotamia. In my study I have encountered at least four different words that have been translated by "leprosy." There is a fairly straightforward philological explanation for this: the Akkadian loanword *saḫaršuppû* may be traced to Sumerian *SAḪAR.ŠUB.A.*, literally, "covered with dust" (i.e., dustlike, whitish scales) (Oppenheim 1956: 273n.54). In different lexical series *SAḪAR.ŠUB.A.* is equated with the terms *epqu* and *garābu*. Thus, *saḫaršuppû* = *epqu* = *garābu*. Furthermore, *saḫaršuppû* occurs in contexts in which the affliction covers the individual "like a garment" and results in his wandering outside the city walls "like a wild ass on the steppe." The combination of excommunication and "whitish scaly skin"—which was not considered inappropriate for leprosy until Dr. S.G. Browne (1967:190) pointed out its unsuitability in a reply to J.V. Kinnier Wilson (1966:47–58)—contributed to the translation of the Akkadian as "leprosy." The fourth term, *būšānu*, seems to have been translated "leprosy" on the basis of association with symptoms more appropriate to the disease (it affects the mouth, nose, skin); however, other references would militate against such a diagnosis.

It is necessary to underscore that the passage from the Epilogue is not a medical text. It does not set forward symptoms for the sake of diagnosis or prescription of medication. Rather, it is a single curse invoking the goddess Ninkarak, in the context of a series of curses which Hammurapi enjoins the gods to bring upon those who would in some way change his laws. The form—(1) deity name (2) deity epithet (3) curse—is typical of Near Eastern curses for a period of more than a millennium. The diction and poetic devices apparent in the curse reveal a subtle literary skill. I would suggest, however, that behind the imagery lies a very specific disease entity that Ninkarak is called upon to inflict. Such a supposition is supported by the fact that the basic nature of the curse remains the same over time. Although the description of the symptoms is general by modern standards and certainly not pathognomonic, the presence of cutaneous lesions, testicular damage, and suggestion of neuropathy point to a consideration of leprosy as the disease.

The text and translation follow:[3]

50. ^{d}Ninkarak
51. mārat Anim
52. qābiat
53. dumqīya
54. ina Ekur
55. murṣam kabtam
56. asakkam lemnam
57. simmam marṣam
58. ša lā ipaššeḫu
59. asû qerebšu
60. lā ilammadu

60. lā ilammadu
61. ina ṣimdī
62. lā unaḫḫušu
63. kīma nišik mūtim lā innassaḫu
64. ina biniātīšu
65. lišaṣiaššumma
66. adi napištašu
67. ibellû
68. ana eṭlūtīšu
69. liddammam

50. May Ninkarak,
51. the daughter of Anum,
52. who speaks
53. my well-being
54. in Ekur,
65. cause to break out
64. on his limbs,
55. a serious illness,
56. an evil affliction,
57. a grievous sore
58. which does not heal,
59. whose nature the doctor
60. cannot find out,
62. which he cannot soothe
61. with bandages,
63. (which) like the bite of death cannot be removed,
66. so that until his life
67. is extinguished,
69. he keeps on moaning
68. about his virility.

The translation of the curse is straightforward and presents few difficulties for the philologist. In lines 50–54 Ninkarak is identified as the malefactor. Ninkarak, equated with Gula and Nin-Isina, all healing goddesses,[4] is the daughter of Anum, the supreme god of heaven. She is the great physician, *azugallatu rabītu*,[5] who confers life and revives from death.[6] Her role as a divine healer makes her eminently suitable as the perpetrator of an incurable affliction and she is frequently summoned for this role in the curse formulae of certain inscriptions.[7] The curse, set forward in lines 55–69, details the nature of the disease. The discussion that follows includes comments on the range of meaning of the Akkadian diction, together with brief literary critical notes, and the signs and symptoms of leprosy that appear in the text.

Leprosy is a chronic infection caused by *Mycobacterium leprae* which primarily attacks the peripheral nerves and dermis.[8] The clinical manifestations vary widely depending upon the immunological response of the host. Variations of the disease range from tuberculoid leprosy to lepromatous leprosy. Specific diagnostic criteria have been established for these forms, although most individuals will present somewhere along the spectrum between the two poles. Knowledge of the clinical aspects of leprosy provides data relevant to an understanding of Ninkarak's curse.

Line 55: *murṣam kabtam* "a serious illness," literally, "heavy illness." *murṣu* is used generally of physical ailments and with a qualifier (often part of the body) for specific illnesses (*CAD* M/2 224–227).[9]

Line 56: *asakkam lemnam* "an evil (in the sense of "dangerous," "bad") affliction." According to *CAD* A/2 325–327, *asakku* is both a demon and the diseases it causes. As Ninkarak is the subject, the one inflicting the ailment, it is clear that another personality is not involved; but the nature of the ailment (other than evil) cannot be further specified. The figurative aspect of the description is underscored by the fact that the term is not found in medical texts (*CAD* A/2 326b). It is noteworthy that Jacobsen (1946:147) interpreted the Sumerian *A-sag* literally as "the one who smites the arm." Although it has been suggested that this interpretation is "probably a popular etymology" (*CAD* A/2 326b), the association with binding, laming, crippling, or paralysis is particularly suggestive with respect to the diagnosis of lepromatous leprosy in which, in the advanced stage, "peripheral nerve disease leads to widespread neuropathic deformities" (Stein et al. 1983:1423).[10] A more secure reference in this context is the mention of *miqtu* "incurable paralyzation (?)" that is attributed to Gula in a similar curse.[11]

Without treatment "fibrosis of nerves is an inevitable end-result of lepromatous leprosy . . . causing bilateral 'glove and stocking' anaesthesia" (Jopling 1984:17). Edema of the legs, an early symptom of lepromatous leprosy, might be applicable here, but the parallel reference to *miqtu* would suggest a more serious disease process. Furthermore, such edema usually precedes the classical skin lesions (Jopling 1984:19), and in this text, emphasis is clearly placed on skin changes. Because of this emphasis, I am inclined to suggest that the text portrays lepromatous leprosy in which the physical signs in the skin are likely to be noticed first and evidence of damage to nerves occurs late (Jopling 1984:21).[12]

Line 57: *simmam marṣam* "serious (grievous) sore/skin eruption." The synonymous parallelism of these three lines poetically emphasizes the severity of the malady.[13] The final description, *simmam marṣam*, provides the most diagnostic information.

There are many types of *simmu:* The deities Sin and Enlil are described as putting every kind of *simmu* in the land (*AMT* 84,4 ii 9 and 11 cited from *CAD* S 277b–278a). For the most part, *simmu* appears to be associated with the skin (also eye) where it may break out *(waṣû)*, appear *(bašû)*, or heal *(balāṭu)* (*GIG* as *simmu*). It may be infectious (*ARM* 10 130:3,14), and it may be treated with medications or bandages (*CAD* S 276–278).[14]

In this passage from the epilogue Ninkarak is called upon to "bring out" the *simmu*, cause it to "come forth," (perhaps "erupt") (Š of *waṣû*, line 65) on the limbs of the offender. Note that the skin lesions of lepromatous leprosy present a "most variegated clinical spectrum" (Arnold and Fasal 1973:40). The skin manifestations may be "macules, papules, nodules or all three" (Jopling 1984:20). They are multiple with bilateral symmetrical distribution. The face, arms, buttocks and legs are principally involved.[15]

The malady is further qualified by the relative clause beginning in line 58 in which the reader is told: (1) that the illness does not heal (with the denotation of "calm down," "be appeased") (line 58), (2) that the doctor cannot find out its fundamental nature *(qerbum)* (lines 59–60), and (3) that it is not relieved with bandages (denotation of *nâḫu* = abatement of illness [*CAD* N/1 147a]; D of *nâḫu* "to stanch, still, allay" [*CAD* N/1 149a]) (lines 60–61). The verbs *pašāḫu* and *nâḫu* may suggest inflammation and perhaps pain (as does the adjective *marṣam* in line 57). I would suggest that if pain is implied the text associates it with the skin lesions. In this context it is significant that, particularly in the reactional states of leprosy, there may be a fairly rapid change in skin lesions. Note especially, in a severe Type 2 reaction, which occurs almost exclusively in lepromatous leprosy, the lesions become vesicular or bullous and break down (erythema necroticans) (Jopling 1984:72 and plate 19). Fever, nerve pain, periosteal pain, muscle pain, and joint pain may also occur (Jopling 1984:73). The neuritis associated with leprosy would be a source of pain; however, in one study 81% of patients gave no significant history of pain or tenderness in affected nerves (Fritschi 1987:173). Again note that unlike tuberculoid leprosy, where pain may be present from the earliest stages of the disease, in lepromatous leprosy, nerve damage usually occurs late.

The striking simile in line 63 underscores the victim's helplessness, the futility of any efforts to overcome the ailment: *kīma nišik mūtim lā innassaḫu*/ "like the bite of death it cannot be removed."[16] The image is palpable; the *simmu, asakku, murṣu* is apparent on the offender's arms and legs, a constant reminder of impending death, specified in lines 66–67: *adi napištašu ibellû*/ "until his life is extinguished."

Line 68: *ana eṭlūtīšu liddammam*. The verb *damāmu* here means "to moan, mourn." There is some debate over the translation of *eṭlūtīšu*. An *eṭlu* is a man, particularly a young, able-bodied man (*CAD* E 407). The form in line 68 may be interpreted as the nominative plural "men" and as such, the passage has been translated "he will complain to his men" (*CAD* E 409b; see also B 73a, D 60a). Such a translation, however, is ill suited to the context. There is no previous reference to the afflicted one's interactions with others and certainly no meaningful referent for "his men." In light of the context of affliction and general physical degradation, it is preferable to read *eṭlūtu*, "virility";[17] thus, "may he continually moan about his virility." Such a reading is congruent with the tangible imagery of the curse and, as the final blow, acutely conveys the humiliation of the progressive debilitation.

The image here is certainly of physical breakdown; however, the reference may even be more specific and refer to testicular atrophy. "Testicular involvement leading to impotence, sterility and gynecomastia is well documented in lepromatous leprosy" (Pareek and Al-Nozha 1985:49).[18] "Varying degrees of testicular atrophy are likely to occur, particularly if the disease is neglected" (Jopling 1984:31). In the earlier stages the individual is sexually potent but sterile. Impotence and gynecomastia are later developments. Also in Type 2 reaction the testes may become swollen and tender with acute epididymo-orchitis (Jopling 1984:73).[19]

Although not diagnostically conclusive, the symptoms may be interpreted as representative of lepromatous leprosy. Clearly, a chronic skin disease involving numerous skin lesions—specifically located on the arms and legs—is described. The severity of the ailment is reinforced by the repetitive formulation of lines 55–57, the fact that its etiology is unknown by the medical practitioner (as well as the fact that it is inflicted by the "divine" doctor), and that surface treatments are of no avail (Fritschi 1987:173).[20] There is an implication that the disease is progressive in the simile, line 63, and more concretely, in the iterative verb form (*liddammam* Gtn *damāmu*) "he keeps on moaning (line 69) until his life is extinguished" which points to a length of time between recognition of the infection and its conclusion.[21] The possibility of peripheral nerve involvement, common in advanced lepromatous disease, was alluded to previously. Although little diagnostic emphasis can be placed on *asakku* in our text, it is intriguing that the malady *miqtu*, from the root *maqātu* "to fall down," is associated with the goddess's persistent *simmu* in another text.[22] Finally, the reference to the loss of virility (lines 68–69) may be interpreted as the testicular damage that leads to atrophy characteristic of lepromatous leprosy.

End notes

1. A number of summaries on medicine in ancient Mesopotamia are available: Labat 1953; Oppenheim 1962; Reiner 1964; Biggs 1969,1978.

2. The most current proposal of which I am aware is that of Kinnier Wilson (1982:354–357) who suggests that in an Old Babylonian omen text published by Köcher and Oppenheim in 1957, the *pūṣu* "white spots," and *nuqdū* perhaps, "nodules," on the afflicted man be interpreted as dimorphous leprosy. Although I cannot offer another interpretation of the skin marks, it seems to push the data too far to infer leprosy without further descriptive material.

3. Codex Hammurabi Epilogus R XXVIII 50–69 (Tafel 29). Tablet and line numbers are taken from the edition of Borger (1963). The normalizations and translations of the text are my own.

4. See Haussig 1965:78 and Weidner and von Soden 1971:695.

5. See, for example, this epithet in the parallel curses: Borger 1970 (Kudurru SB kol. IV 5–6), 1967:109 (IV 3); King 1912:7 ii 29–31.

6. *RLA* 3 695. Note the hymn of Nebuchadnezzar to Ninkarak in which Nebuchadnezzar appeals to her as a health goddess—for his own health and longevity and that of his children and descendants (von Soden and Falkenstein 1953:#33).

7. See Kudurru (boundary stone) of Melišiḫu VII 14–25 (Scheil 1900:110); Kudurru SB 33 IV 5–9 (Borger 1970); King 1912: no. 8 iv 16, 1 R 70 iv 6 and p.41 7 ii 29–31 and p.79 II iii 10–13.

8. For an introduction to the subject see Braunwald et al. 1987; du Vivier 1986; Fitzpatrick et al. 1987; Jopling 1984; Stein et al. 1983.

9. For example, associated with the goddess Gula but with regard to the heart: *GIG lā pādû ana libbišu li[bši(?) KAR* 111 r.8 cited from *CAD* M/2 226a)/ "May (Gula bring) an illness without pity into his heart."

10. "A diffuse hypesthesia involving the peripheral portion of the extremities is common in advanced lepromatous disease" (Braunwald et al. 1987:635).

11. Kudurru SB 33 IV 7 which Borger translates as "unheilbare Lähmung(?)" (1970:15). See also Goetze (1955:12) who notes that *miqtu*, literally "fall," is used of attacks for various sicknesses and sometimes equated with *bennu*, "epilepsy."

12. Here Jopling clarifies that "nerve damage in lepromatous leprosy . . . is different from the pathological process in the other types of leprosy and is much slower to unfold." While in borderline leprosy "clinical evidence of nerve damage, whether sensory or motor, or both, is likely to antedate skin lesions by months or years" (1984:15). Tuberculoid leprosy patients may present with neural or dermal symptoms or both (1984:34).

13. Note the alliteration (particularly m, ṣ, š, and k) and the inclusio and wordplay, *murṣam–marṣam*.

14. The range of meaning of Akkadian *simmum* has been compared to Greek *elkos* which embraces both notions of wound and ulcer. (Stol 1979:62 n.232, kindly brought to my attention by Professor W.L. Moran).

15. See Jopling 1984:figs. 4,15,18, erythema nodosum leprosum in Type 2 reaction–which occurs almost exclusively in lepromatous leprosy or may be the stage at the patient's presentation (1984:70).

16. The image here has several levels: (1) Death is frequently anthropomorphized as a maw; consequently, the image would be particularly vivid for its indigenous readers/hearers. See also *ina šinnatīšu izāb mūtum*/ "from his (the demon's) teeth flows death." (A 704:16 cited from *CAD* M/2 318a). (2) The symbolic animal of Gula is the dog. (3) Familiarity with an actual animal bite introduces the elements of the unexpected and speed—a bite often cannot be anticipated or prevented—thus heightening the sense of anxiety. (4) The physical image: once the flesh has been pierced, although the teeth may be removed, the evidence of the attack cannot—thus, the omnipresent awareness of injury.

17. *CAD* E 411b. Von Soden (1965:1 266a) who quotes this passage under *eṭlūtu*, defines the word as "Mannheit." Therefore, our form *eṭlūtīšu* = genitive of *eṭlūtu* + 3ms suffix.

18. This study notes earlier reports stating that half of the males with lepromatous leprosy develop testicular atrophy.

19. Note as well that protein and red blood cells occur in the urine. In this context it is most interesting to compare a difficult passage in a parallel curse associated with Gula's persistent *simmu: šarka u dāma/kīma mê lirtammuk*, "so that he may pass light and dark blood like water" (King 1912:7 ii 31; 11 iii 12–13).

20. It is significant that in four other texts Gula is associated with this same incurable affliction —*simmu lazzu*, "a persistent sore/skin eruption" (Scheil 1900:110 [VII 19]; King 1912:41 [7 ii 30]; Borger 1967:109 [IV 4]); and *simma akṣa lazza*, "a terrible, persistent sore" (Borger 1970:15 [IV 6]).

21. "The course of untreated lepromatous leprosy in presulfone days was in general a progressive downhill one, with eventual fatal termination. . . . The disease itself is seldom a direct cause of death, . . . but it so enfeebles the patient in advanced cases that other infections may have a fatal outcome" (Arnold and Fasal 1973:51–52).

22. See note 11.

Literature cited

Works frequently cited have been identified by the following abbreviations:

AMT	R.C. Thompson. *Assyrian Medical Texts*. Oxford 1923.
ARM	*Archives royales de Mari*. Paris 1950ff.
CAD	*The Assyrian Dictionary of the Oriental Institute of the University of Chicago*. Chicago 1956ff.
RLA	E. Ebeling and B. Meissner, eds. *Reallexikon der assyriologie*. Berlin 1928ff.

Arnold, H.L., and P. Fasal. 1973. *Leprosy Diagnosis and Management*. Springfield, Ill.: Charles C Thomas.

Biggs, R. 1969. Medicine in Ancient Mesopotamia. *History of Science*, 8:94–105.

———. 1978. Babylonien. In H. Schipperges, E. Seidler, P.U. Unschuld, eds., *Krankheit, Heilkunst, Heilung*, 91–114. Munich: Verlag Karl Alber.

Borger, R. 1963. *Babylonisch-Assyrische Lesestücke. III Kommentar die Texte in Keilschrift*. Rome: Pontifical Biblical Institute.

———. 1967. *Die Inschriften Asarhaddons Königs von Assyrien*. Osnabrück, Germany: Biblio Verlag.

———. 1970. Vier Grenzsteinurkunden Merodachbaladans I. von Babylonien. *Archiv für Orientforschung*, 23:15 (Kudurru SB 33 kol.IV 5–6).

Braunwald, E., K.J. Isselbacher, R.G. Petersdorf, J.D. Wilson, J.B. Martin, and A.S. Fauci, eds. 1987. *Harrison's Principles of Internal Medicine*. New York: McGraw-Hill.

Fitzpatrick, T.B., A.Z. Eisen, K. Wolff, I.M. Freedberg, and K.F. Austen, eds. 1987. *Dermatology in General Medicine*. New York: McGraw-Hill.

Fritschi, E.P. 1987. Field Detection of Early Neuritis in Leprosy. *Leprosy Review*, 58(1–3):173–77.

Goetze, A. 1955. An Incantation Against Diseases. *Journal of Cuneiform Studies*, 9:8–18.

Haussig, H.W., ed. 1965. *Wörterbuch der Mythologie I Götter und Mythen im Vorderen Orient*. Stuttgart, Germany: Ernst Klett Verlag.

Jacobsen, T. 1946. Sumerian Mythology: A Review Article. *Journal of Near Eastern Studies*, 5.128–152.

Jopling, W.H. 1984. *Handbook of Leprosy*. London: Heinemann Medical Books.

King, L.W. 1912. *Babylonian Boundary Stones*. London.

Kinnier Wilson, J.V. 1966. Leprosy in Ancient Mesopotamia. *Révue d'Assyriologie*, 60:47–58.

______. 1982. Medicine in the Land and Times of the Old Testament. In T. Ishida, ed., *Studies in the Period of David and Solomon and Other Essays*, 337–365. Winona Lake, Ind.: Eisenbrauns.

Labat, R. 1953. La Médecine Babylonienne. In *Les Conférences faite au Palais de la Découverte le 18 Avril 1953*, 5–23. Paris: Université de Paris.

Oppenheim, A.L. 1956. *The Assyrian Dream-Book*. Philadelphia: American Philosophical Society.

______. 1962. Mesopotamian Medicine. *Bulletin of the History of Medicine*, 35(2):97–108.

Pareek, S.S., and M. Al-Nozha. 1985. *Mycobacterium leprae* in Seminal Fluid: A Case Report. *Leprosy Review*, 56:49–50.

Reiner, E. 1964. Medicine in Ancient Mesopotamia. *Journal of the International College of Surgeons*, 41:544–550.

Scheil, V. 1900. *Délégation en Perse Mémoires. II Textes Elamites Sémitiques*. Paris: Leroux.

von Soden, W. 1965–1981. *Akkadisches Handwörterbuch I–III*. Wiesbaden, Germany: Harrassowitz. [I, 1965; II, 1972; III, 1981.]

von Soden, W., and A. Falkenstein. 1953. *Sumerische und Akkadische Hymnen und Gebete*. Zurich: Artemis.

Stein, J.H., M.J. Cline, W.J. Daly, J.D. Easton, J.J. Hutton, P.O. Kohler, R.A. O'Rourke, M.A. Sande, J.S. Trier, and N.J. Zvaifler, eds. 1983. *Internal Medicine*, vol. 2. Boston: Little, Brown, and Co.

Stol, M. 1979. *On Trees, Mountains and Millstones in the Ancient Near East*. Leiden, Netherlands: Ex Oriente Lux.

du Vivier, A. 1986. *Atlas of Clinical Dermatology*. Philadelphia: W.B. Saunders. Weidner, E., and W. von Soden, eds. 1971. *Reallexikon der Assyriologie und Vorderasiatischen Archäologie* 3. Berlin: de Gruyter.

SUMMARY OF AUDIENCE DISCUSSION: Because people write about what is important to them, texts may be a useful source of information regarding disease in past periods even if the purpose of the text is nonmedical. If the described condition is really leprosy, the textual emphasis on peripheral, dermatological involvement is surprising. The fact that skin changes occur early appears to be an inadequate explanation since the comment regarding virility suggests testicular involvement which occurs much later. Another limitation is the fact that this manuscript is obviously a curse, and therefore should not be expected to be a precise description of a disease. An acute, lethal disease involving predominantly skin may be more likely. A recent translation of the Egyptian Ebers Papyrus does not mention lepromatous leprosy.

The medieval diagnosis of leprosy

Johs G. Andersen

Modern clinical diagnosis

In clinical praxis the diagnosis of leprosy rests on the presence of at least two or three symptoms: hypopigmented skin lesions, reduction or loss of sensation in visible skin lesions, and enlarged peripheral nerves. Demonstration of alcohol- and acid-fast, intracellular rods in slit skin smears or biopsies confirms the diagnosis unequivocally.

Classification

In the classical texts no attempts at classification are met. The medieval literature abounds in terms that show a characteristic emphasis on individual, significant symptoms. The texts obviously presume a direct teacher-student situation.

The described symptoms fit effortlessly into a description of borderline lepromatous leprosy. As typical of this we can refer to the famous Flos Medicina from the medical school in Salerno (de Gaddesen 1492):

> De Specibus Leprae
> Tyria primo datur de flegmate qua generatur,
> Inde leonina cholera generante ferina
> Triste pilos tollens allopicia sanguine nascens,
> De melancolia tristis elephancia saevior istis,
> In facie noli tangere, in partibus herpes,
> Inferius si sit dicitur esse lupus.

Or in translation:

> Tyria in the beginning originates only from mucus
> Wild is leo, and comes to our sorrow from t'gall of the body
> Born from bad blood and extracting the hair is the sad alopecia
> Elephas, still more wild, it is born from the sorrowful black gall
> When it is seen in the face and breaks out in the skin, it is hopeless
> Lupus we call it when only the parts further down are attacked.

The first attempts at a classification that approaches our understanding appear toward the end of the 19th century. Here also for the first time we meet descriptions that can refer to tuberculoid leprosy.

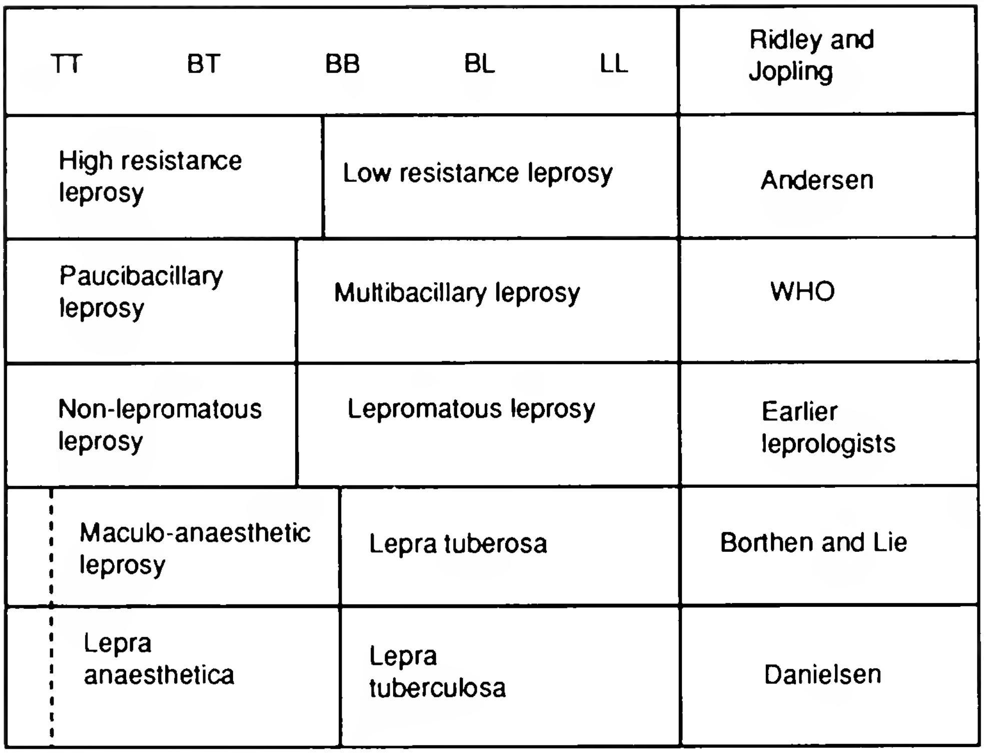
As typical examples we can refer to the classifications as described by Danielsen (1873) or Borthen and Lie (1899).

During the first half of the 20th century most practicing leprologists classified leprosy as lepromatous or non-lepromatous leprosy. Ridley and Jopling in 1966 introduced the now universally accepted five-point classification, based on the immunological response of the host. In 1969 I introduced the terms "high resistance leprosy" (HRL) and "low resistance leprosy" (LRL). They provide a reasonably accurate relation to the immunological classification. Since the introduction of multidrug therapy (WHO Study Group, 1982) there is a tendency to classify leprosy as multibacillary or paucibacillary leprosy for purposes of primary drug therapy. Unfortunately there is no generally accepted definition of these terms. The reader is referred to Figure 1 for comparison between the different classifications.

Reading of ancient texts

The free use of quotations, frequently without any acknowledgment of source, makes it difficult to assess which is an original observation, and which is a reference to existing knowledge. There is a tendency to describe symptoms, with less correlation of different symptoms to define a particular disease. It can be difficult to determine if a particular term covers a subgroup of a given disease or rather a disease in itself. We should not overlook the confusing use of the terms derived from *elephas* and *lepra*, indicating the same disease.

Direct translations can cause misunderstandings: The Greek *paus* and *cheir*, usually translated as *hand* and *foot*, are frequently used to indicate the whole extremity. The same is true about the Latin *pes* and *manus*. *Extremitas manus* can thus mean either the upper extremity or the distal part of the hand, that is, the tips of the digits.



TT BT BB BL LL	Ridley and Jopling
High resistance leprosy — Low resistance leprosy	Andersen
Paucibacillary leprosy — Multibacillary leprosy	WHO
Non-lepromatous leprosy — Lepromatous leprosy	Earlier leprologists
Maculo-anaesthetic leprosy — Lepra tuberosa	Borthen and Lie
Lepra anaesthetica — Lepra tuberculosa	Danielsen

FIGURE 1. The relationship between different classifications of leprosy.

Classical texts

Unfortunately the earliest literary reference to leprosy in the Mediterranean world has been lost. We know it only from a quotation in the works of Oribasius (A.D. 326–403). He mentions that Straton, famulus of Erasistratos from Keos (300–250 B.C.) describes elephas as a new disease. It is not clear how much of Oribasius's description is attributed to Straton, and how much represents his own observations. In any event the nodules, ecchymosis-like skin lesions, and the nonhealing ulcers definitely point toward LRL. The picture is more appropriate to leprosy in light-skinned races. This may be confusing to leprologists who are accustomed to dealing with dark-skinned races.

Albinovarus Cornelius Celsus (25 B.C.–A.D. 37) mentions leprosy as a relatively new disease in Italy. He writes a remarkably lucid, beautiful Latin, which leaves no doubt that he describes LRL: "Crebri tumores" exactly fits the appearance of partly confluent, nodular infiltration. "Summa cutis indaequaliter crassa, tenuis, dura, mollisque, quasi squamis exasperatur" reads as a description of borderline lepromatous leprosy with the characteristic, irregular, ring lesions with a flat center, pseudopodia-like spread, and a shift to the lepromatous side with widespread macules. "Digiti in manibus pedibusque sub tumore conduntur" recalls the patients with partial digital absorption and swelling of hands and feet due to reversal reaction. "Ossa quoque vitiari dicantur" could be a reference to peripheral absorption or to ulcer-induced bone destruction. In any case it fits very well into the whole picture.

Caius Publius II Plinius (A.D. 23–79) is a typical polyhistorian who writes extensively about everything. He is not a medical man. The main interest in his writings is the fact that he specifically mentions that leprosy was unknown in Italy prior to the return of Gnaeus Pompeius Magnus (106–48 B.C.) from the Pontine campaign in 62 B.C.

Aretaios Kappadox (ca. A.D. 200) is without doubt the most lucid and informative of the authors from this period. A number of interesting points can be gleaned from his writings: Elephas (i.e., leprosy) is also known as leo. This presumably refers to facial infiltration. He is aware of the insidious beginning of the disease, and mentions that foul-smelling respiration is characteristic. "Tumors of the body" easily reads as "nodular infiltration." Hoarse voice and loss of hair are characteristic symptoms. Leprous alopecia geographica is extremely rare in dark-skinned races, but fairly common in advanced borderline lepromatous or lepromatous leprosy in light-skinned races.

As a surgeon I am particularly intrigued by the first mention of plantar cracks and ulcers. The expression "loss of nose" may well refer to the contracted/collapsed nose that is so common in advanced, borderline, lepromatous leprosy and lepromatous leprosy. All in all a modern leprologist has little to add to his description of LRL.

It is remarkable that the famous physician, Claudius Galenos (A.D. 130–201), who has been held in such high regard through the ages, should fail to add to previous knowledge.

Medieval texts

When we move on to the medieval writers we must remember that their knowledge of the classical literature came from translations of the original Greek and Latin into Arabic and retranslations into Latin. From this time until the end of the 19th century, the terms lepra and elephas (and derivatives) are used alternately to describe the same disease. The confusion with biblical "leprosy" is a later phenomenon.

One is frequently struck by the simple mention of what is obviously intended as a description, but without the definition and explanation that we expect from a medical text. It probably means that the text is intended as a *vade mecum*, while the detailed teaching is presumed from a direct teacher-student relationship. The teaching hexameters in Flos Medicina (de Gaddesen 1492) provide good examples:

> De Signis Variarum Specierum Leprae
> Candescit cutis in tyria, mollescit et albet,
> Nec membris lymphae profusio facta cohaeret,
> Signa leoninae: manuum fissuraque pedum,
> Aspera rupta cutis, macies, pruritus et ardor,
> Vox est rauca, color citrinus, mobile lumen,
> Fit gingivarum corrosio, naris acuta
> contrahit et spasmat, species elephancia nervos,
> corrugat naris, oculos facit esse rotundos,
> tubera dura rigent, caro livida, sqalidus unguis

Or in translation:

> When the skin pales and is blotched, it is taken as symptom of tyria,
> Then there is no longer lymph to be found in abundance in the members,
> Leo you know from the cracks which are found in the hands and the feet,
> Also in them you find broken skin, leanness and itching and burning,
> Hoarse voice and a face with the color of lemon, and eyes which are roving,
> Gums which are eaten away, while the nostrils are pinched and obstructed.

Arnaldus de Vila Nova (A.D. 1235–1312) is a typical proponent of medieval knowledge of leprosy. From De Signis Leprosorum Libellus we glean:

> Si vox rauca est, forte signum est leprae [laryngeal infiltration]
> In superciliis oculorum leprosi non habet pilos maxime apud angulos [madarosis]
> Supercilia habent quandam rotunditatem, qua videntur quasi spherica et rotunda [lagophthalmos with retracted eyelids]
> Oculi videntur quasi exire locum eorum [lagophthalmos with pseudoexophthalmos]
> Facies—habet aspectum multum terribilem [lagophthalmos and /or facial infiltration]
> Leprosi cognascuntur ex vulnere existente in naribus [endonasal ulceration]—excoriatio in profunditate nasi [in contradistinction to e.g. scrofulosis].

He even has an excellent description of how to examine for loss of sensation:

> Item facias ipsum cooperiri ne videat et sibi dic: cave quod ego te pungam, et no pungetur, et post dic punxi te in pede. Si dicat quod sic, signum est leprae.

He also presents the first definite description of ulnar loss of sensation and paralysis. This speaks highly of his acute powers of observation:

> Item debet pungi cum acu a minimo digito manus et sibi vicio usque ad brachium, quod in instis digitis magis aliis ratio est, quod sunt debiliores et ideo citius dimittuntur a regimine naturae.

Taken as a whole, this presents a clear picture of LRL. Individual symptoms might be read as referring to HRL, but it is obvious that the descriptions as a whole cover one single condition, LRL.

Conclusions

From the earliest description of leprosy in the Mediterranean world through the Medieval period, leprosy is well described. An exact correlation with modern, immunological classification is not possible. It is possible to define classification using the terms that are used in paleopathology. The literature descriptions cover LRL. No clear reference to HRL can be found. It is not possible to reconcile the descriptions with other diseases, nor is it possible to read descriptions of other diseases as leprosy. Available paleopathological findings present the same picture (e.g., Møller-Christensen et al. 1952.

We are left with a puzzling problem: Why does HRL only appear toward the end of the 19th century? Has the immunological response of the population changed? Is it a question of interaction with other mycobacterial diseases, such as tuberculosis? Or, most improbable, has *Mycobacterium leprae* changed?

Literature cited

Andersen, J.G. 1969. *Studies in the Medieval Diagnosis of Leprosy.* Copenhagen: Costers Bogtrykkeri.

______. 1982. The Osteoarchaeological Diagnosis of Leprosy. *Proceedings of the Palaeopathology Association 4th European Meeting, Middleburg,* 221–228.

Aretaeus. 1828. *Aretaei Cappadocis Opera Omnia.* D. Carolvs Gottlob Kühn, ed. Lipsiae: prostat in officina libraria C. Cnoblochii.

Arnaldus de Villa Nova (Sive Bachudne). 1309. Breuiarium practicae a capile ad pedem (Brew. II, Cap. 46), Napoli.

Borthen L.M., and H.P. Lie. 1899. *Die Lepra des Auges: Klinische Studien von Lyder Borthen, mit Pathologisch-Anatomischen Untersuchungen.* Leipzig, GDR: Engelmann.

Celsus, A.C. 1891. *A. Cornelii Cels De medicina libri octo. Ad fidem optimorum librorum denuo recensuit, adnotatione critica indicibusque instruxit C. Daremberg.* Lipsiae: B.G. Teubneri.

Danielsen, D.C. 1873. Lungegaardshospitalets Virksomhed i Treaaret 1871–73. *Norsk Mag Lægev,* 2(4):313–396.

de Gaddesen, J. 1492. *Rosa Anglica IV Libris Distincta Papua.*

Møller-Christensen, V., S.N. Bakke., R.S.Melsom, and E. Waaler. 1952. Changes in the Anterior Nasal Spine and the Alveolar Process of the Maxillary Bone. *International Journal of Leprosy,* 20:335–340.

Oribasius. 1851–1876. *Oeuvres d'Oribase.* Translated by Drs. Bussemaker and Daremberg. Paris: Impr. nationale.

Plinius. 1829. *Ajason de Grandsagne.* Histoire Naturell de Pline. Paris.

Ridley, D.C., and W.H. Jopling. 1966. Classification of Leprosy According to Immunity. *International Journal of Leprosy,* 34: 255.

WHO Study Group. 1982. *Multidrug Therapy in Leprosy.* Geneva.

SUMMARY OF AUDIENCE DISCUSSION: In Hawaii it was possible to demonstrate a chronological succession of changes from an initial low ratio of tuberculoid (high resistance)/lepromatous (low resistance) leprosy forms to a later reversal of this ratio. If an elevated value for this ratio reflects a longer duration of leprosy within a population (and adaptation by development of resistance to it) then it would be interesting to determine this ratio among both current living populations throughout the world and in antiquity. Lack of available statistics limits study of modern groups, and absence of facial destruction makes it very difficult to differentiate the two forms in archeological skeletal populations. In modern populations local secondary mycotic infections of the extremities are common owing to loss of sensation resulting from the neuropathy. Death in antiquity may have been due primarily to amyloidosis, as it is now.

Arthritis

Rheumatoid erosive arthropathy as seen in macerated (dry) bone specimens

James C.C. Leisen, Howard Duncan, and J.M. Riddle

By definition, the process of erosion means a gradual wearing away of a substance. Medically, this term is frequently used to describe a focal loss of tissue such as occurs in the development of an ulcer. Rheumatologists use the term erosion more specifically to denote a focal loss of articular cartilage and/or bone. The affected area therefore appears radiolucent on x-ray films. Radiographic erosions can be detected in a number of rheumatic diseases involving peripheral joints, and of these, rheumatoid arthritis is the paradigm.

Rheumatoid arthritis can be defined clinically by a set of criteria (Ropes et al. 1958:175), but none of these features alone, including radiographic erosions, is specific for this type of arthritis. When summated, however, the various clinical and laboratory findings describe a cadre of patients who have a chronic, nonbacterial, noncrystalline, symmetrical polyarthritis in which 40 to 85% show bony erosions by examination of anteroposterior radiographs of the hand (Mitchel and Fries 1982: 481). Pathologically, the erosion appears to be related to chronic inflammation and proliferation of the synovial membrane, that is, the formation of rheumatoid pannus. Contact of the pannus with articular cartilage and/or bone promotes a focal loss of underlying tissue creating the erosion that is demonstrated by radiologic examination of the joint.

Although some radiopathologic correlations of the rheumatoid erosion in peripheral joints have been made (Resnick and Niwayama 1981:907), we are unaware of any published studies on the appearance of the rheumatoid erosion in macerated (dry) bone. Such a study might be useful to paleopathologists as a frame of reference for interpreting skeletal material. For this reason, we have described the morphologic features of the rheumatoid erosion as seen in macerated (dry) bone of surgically removed metacarpal heads and tibial plateaus. In our series of over 1000 patients with rheumatoid arthritis seen in the Rheumatology Clinic, approximately 50% have clinical involvement of the second and/or third metacarpophalangeal joint and knee.

Materials and methods

Twenty-six second and third metacarpal heads were obtained from the hands of 13 patients with classic rheumatoid arthritis. Ten specimens from eight patients were selected for study because they contained residual articular cartilage and adjacent pannus. Included in this series were six women and two men. Their mean age was 56 years (range 29–72 years) and the average duration of their disease was 7.9 years (range 4–20 years).

Tibial plateaus were collected from the knees of seven other rheumatoid patients. This group included six women and one man. Their mean age was 66 years (range 61–72 years). All of these samples were available because it was necessary to remove the diseased joints in order to introduce an artificial joint.

Control samples, 28 second and third metacarpal heads as well as 14 tibial plateaus were obtained from seven cadavers. The mean age of this group of three women and four men was 71 years (range 41–83 years). Their causes of death were acute myocardial infarction (4), congestive heart failure (2), and a cerebrovascular accident (1). Tibial tables were also collected from six patients who required an above-the-knee amputation because of peripheral vascular occlusive disease. This series contained equal numbers of men and women. Their mean age was 72 years (range 64–85 years).

The articular cartilage on all of the joint surfaces was examined under the dissecting microscope to detect cartilage fibrillation or the presence of pannus. Radiographs of each metacarpal head and tibial plateau were also taken to locate erosions and cysts.

Subsequently, the specimens were macerated using 5.25% sodium hypochlorite (Clorox) for approximately 24 hours to completely remove the soft tissue. This step of the process was verified by examination with a dissecting microscope. Following maceration, each specimen was defatted in acetone, air dried, mounted on aluminum stubs, and sputter coated with a thin layer of gold-palladium. This procedure not only permitted study by scanning electron microscopy as previously described (Leisen et al. 1988:17) but also allowed examination with the naked eye or a dissecting microscope.

We used the following terms to define specific anatomic sites: *Articular surface* indicated the joint surface originally covered by articular cartilage. The *chondro-osseous junction* defined the discrete line of contact between articular cartilage and bone at the joint periphery. *Para-articular bone* referred to epiphyseal bone anatomically located within the joint capsule and adjacent to the chondro-osseous junction.

Bony features described were the following: *Holes* were defined as any circumscribed break in the bony surface which exposed the marrow space or underlying trabecular bone (Duncan et al. 1987:1212). The edges of the holes were called *sclerotic* if they were rounded and slightly raised and/or thickened. An *osteophyte* was defined as any bony proliferation on or around the edge of the articular surface.

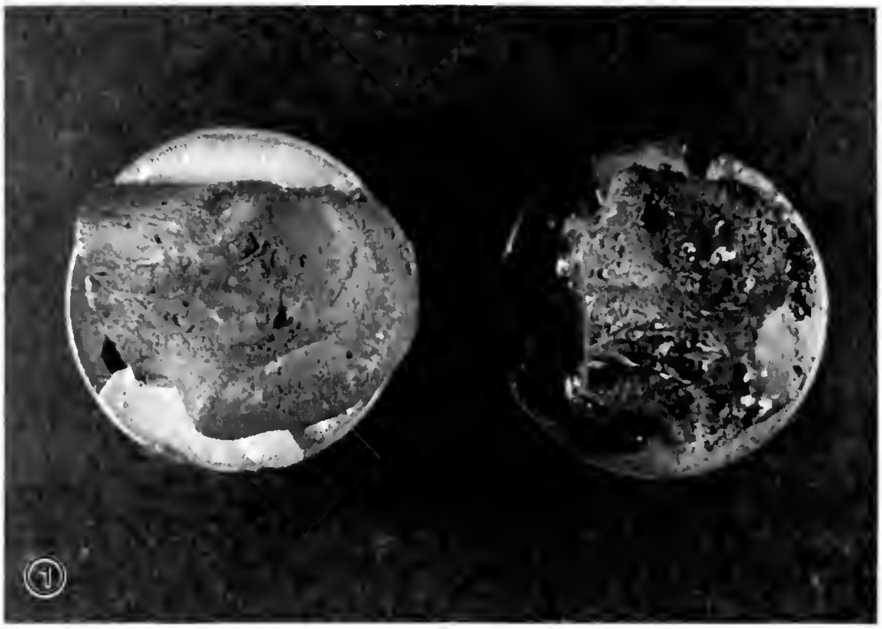


FIGURE 1. Hemisection of cadaver metacarpal head *(left)* and a rheumatoid metacarpal head *(right)* showing erosion of articular surface associated with the inflammatory process; × 2.

Results

GROSS OBSERVATIONS AND DISSECTING MICROSCOPE VIEWS

The unmacerated cadaver and amputee specimens (metacarpal heads and tibial plateaus) showed no pannus formation and only minimal articular cartilage fibrillation by examination with the naked eye. No radiographic chondrocalcinosis was seen.

The articular surface, chondro-osseous junction and para-articular structures on macerated control samples were easily identified. Prominent features of the control macerated metacarpal heads included: (1) condylar eminences which were most pronounced on the radial side, (2) a convoluted chondro-osseous junction, and (3) deep bony invaginations in the valleculae just posterior to the condylar eminences. These valleculae contained at least one dominant hole which was the site of entry of blood vessels originally supplying the bony epiphysis (Figure 1, left).

The medial and lateral plateaus of tibial tables removed from the cadavers and above-the-knee amputees were distinct and separated by a central, elevated prominence (Figure 2). The articular surfaces of the plateaus appeared smooth when viewed with the naked eye or the dissecting microscope.

Gross examination of the rheumatoid specimens by contrast showed varying degrees of architectural destruction and variable amounts of pannus. Partial to complete resorption of the articular surface was seen in the macerated specimens. Loss of the articular surface on the rheumatoid metacarpal heads was most noticeable on the condylar eminences where seven of ten specimens showed complete resorption and three samples showed partial destruction (Figure 1, right). In three specimens, the articular surface over the condyles was also perforated by many large holes. A small cap of residual surface remained intact on the radioulnar aspect of five metacarpal heads. In all specimens, there was circumferential loss of the chondro-osseous junction, and the para-articular bone was grooved as well as excavated. Many holes showed no evidence of sclerosis around their edges. Eburnation was seen on the articular surface in two of the rheumatoid specimens (Table 1).

The rheumatoid tibial plateaus often showed that the articular surfaces were eburnated toward the intercondylar areas. Both the submeniscal and central surfaces of the plateaus contained many holes without sclerotic rims through which the marrow space and/or trabeculae were visible. In some areas the chondro-osseous junctions were obliterated. Para-articular bone contained low ridges of small bony osteophytes (Table 2). The intercondylar area in all specimens had many holes with little surrounding sclerosis (Figure 3).

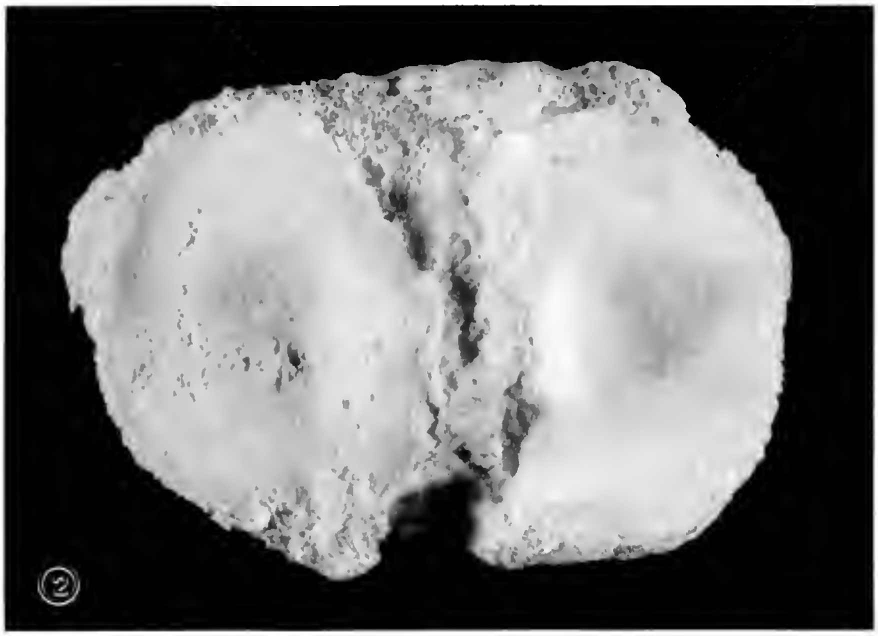


FIGURE 2. Macerated control tibial table from amputee. Note minimal change (focal collection of small holes in the lateral plateau *(left)*; × 1½

TABLE 1. Summary of morphologic changes observed on rheumatoid metacarpal heads

	Frequency (%)
DISSECTING MICROSCOPE (6-10X)	
Articular surface	
Dome	
Complete resorption	2/10 (20%)
Partial resorption	8/10 (80%)
Holes	2/10 (20%)
Condyle	
Complete resorption	7/10 (70%)
Partial resorption	3/10 (30%)
Holes	3/10 (30%)
Eburnation	2/10 (20%)
Chondro-osseous junction	
Vallecular resorption	10/10 (100%)
Para-articular bone	
Complete destruction	4/10 (40%)
Holes	6/10 (60%)
SCANNING ELECTRON MICROSCOPE (100X)	
Resorption lacunae at sites of calcified tissue destruction	10/10 (100%)

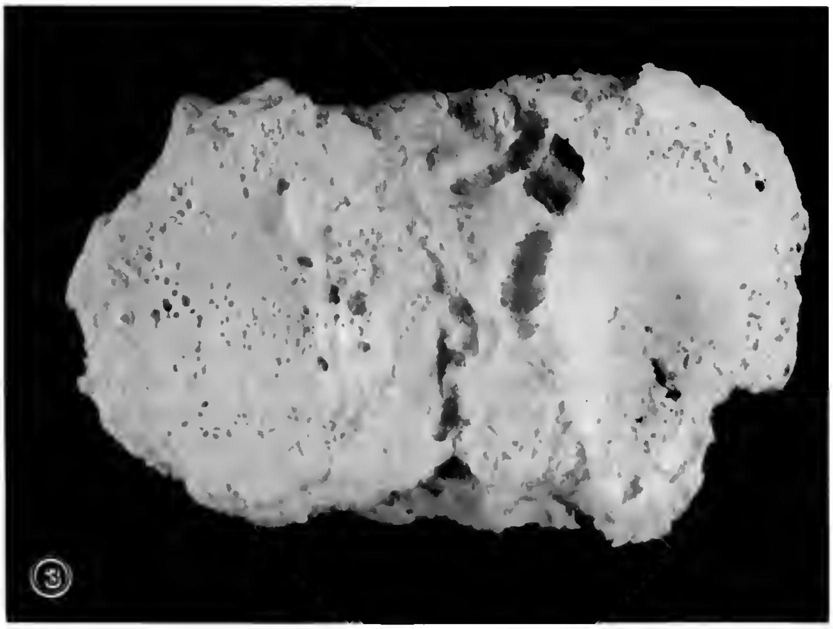


FIGURE 3. Macerated rheumatoid tibial table; × 1½.

TABLE 2. Comparison between macerated tibial plateaus of patients with rheumatoid arthritis (RA) and control subjects

	RA	*Control*
Bone thickness	thin	normal
Osteophytes	few	±
Eburnation	±	–
Holes in articular surface	+ + +	+
Holes in intercondylar area	+ + +	–
Holes in para-articular area	+ +	–

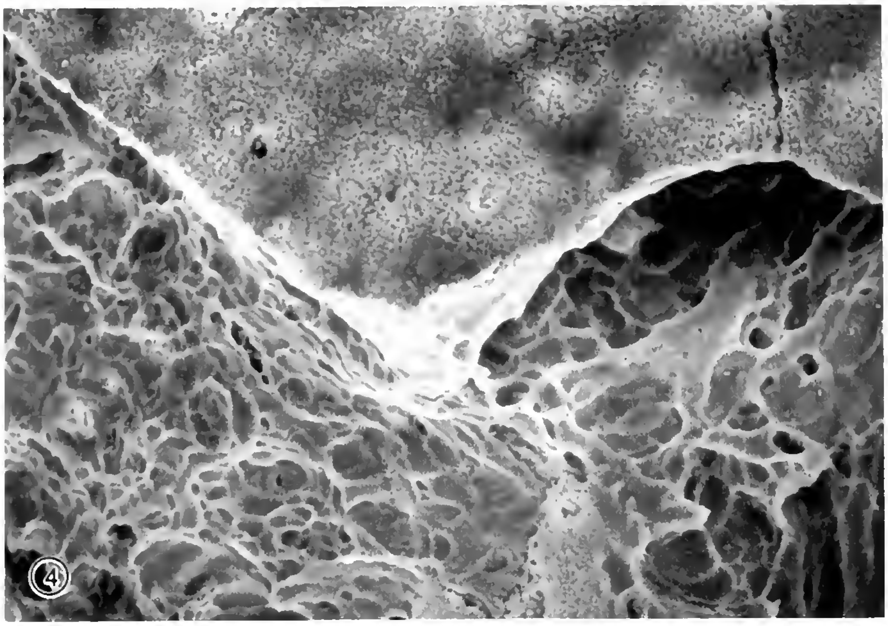


FIGURE 4. Articular surface of a rheumatoid metacarpal head showing an eroding front covered with continuous resorption bays and an adjacent mineralized surface with prominent chondrocyte lacunae (volcanoes); × 560.

SCANNING ELECTRON MICROSCOPIC OBSERVATIONS

The articular surface of the macerated specimens (both control and rheumatoid specimens) was covered by numerous volcanolike structures. These were identified as mounds of mineral enclosing lacunae which would have originally contained a single or several chondrocytes (Figure 4).

At a higher magnification, many small holes were seen perforating the subchondral plate of both the medial and lateral plateaus of the tibial tables collected from the controls. These holes were concentrated at a submeniscal location on the medial plateau and at a central location on the lateral plateau. In contrast, various-sized holes penetrated the subchondral plate covering the medial and lateral plateaus of the rheumatoid tibial tables. No distinct distribution pattern of holes was observed for the plateaus of the rheumatoid tibial table. Rather, the holes frequently were present in both the submeniscal and central locations of a plateau.

The articular surface of the rheumatoid specimens also showed focal collections of continuous resorption bays, an erosion with a morphology characteristic of osteoclastic activity (Figure 4).

Discussion

Our series included metacarpal heads and tibial plateaus removed from control subjects and patients with chronic, classic rheumatoid arthritis. The joints of the rheumatoid patients were functionally impaired and painful to the extent that the joint dysfunction necessitated surgery and replacement with prostheses. In this group, the rheumatoid erosion had characteristic features which included: (1) partial or complete resorption of the articular surface, (2) obliteration of the chondro-osseous junction, and (3) minimal para-articular osteophytosis. The remaining bony tissue, regardless of location, contained holes or perforations through the surface which often exposed epiphyseal trabecular bone and marrow space. Although histologic features were variable, the scanning electron microscope consistently revealed patches of continuous resorption bays at the eroded surfaces and lining the channels of many holes.

A review of the literature contained only four published papers (Table 3) in which an erosive arthropathy was suggested through the examination of dry bone (Klepinger 1979:119; Ortner and Utermohle 1981:23; Rogers et al. 1981:1668; Thould and Thould 1983:1909). Seven skeletons were examined and six were found to show evidence of appendicular and axial (Ortner and Utermohle 1981: 23) bony defects suggestive of erosion. These defects were studied by visual and radiographic examination. Although the authors suggest a variety of disease entities, we feel that the basic issue of "what is an erosion" needs to be addressed using radiographs and different microscopic approaches including light microscopy and scanning electron microscopy as illustrated in our study.

The peripheral joints of a number of erosive arthropathies such as seronegative rheumatoid arthritis and the spon-

TABLE 3. Summary of published studies describing an erosive arthropathy seen in dry archeological bone

Author	*Site*	*Age of specimens*	*Material*
Rogers et al. 1981	Britain	A.D. 1200	3 of 400 skeletons: 3 males with erosive disease, possible gout (1), psoriatic arthritis (1), rheumatoid arthritis
Ortner and Utermohle 1981	Alaska	>A.D. 1200	1 female skeleton age 30–35, polyarticular erosive disease
Klepinger 1979	Sicily	330–210 B.C.	1 male skeleton with distribution of abnormalities suggesting rheumatoid arthritis
Thould and Thould 1983	Britain	A.D. 0–400	2 of 416 skeletons: 1 male, 1 female with erosions involving wrist and metacarpals

dyloarthropathies have yet to be studied in this manner. In addition, morphologic surveys of patients with early and presumably less severely disabling rheumatoid arthritis might reveal erosive features that would be helpful in establishing the existence of erosive arthropathies in paleopathologic skeletal material.

Finally, although the word "erosion" implies a focal defect in bone and/or cartilage, as seen radiographically, this type of focal change was not a feature of the specimens examined in this study. Rather, the rheumatoid erosions we observed were more extensive and included the sum of the anatomical defects noted in the articular surface, chondro-osseous junction, and para-articular bone. It appears that a multidiscipline approach that includes the distribution of joint lesions, radiographic evaluation of affected joints, and a careful morphologic survey of the joints using both light as well as scanning electron microscopy may be necessary to interrelate findings on modern patients with classic rheumatoid arthritis to joint lesions present in ancient skeletal remains.

Literature cited

Duncan, H., J. Jundt, J. Riddle, W. Pitchford, and T. Christopherson. 1987. Tibial Subchondral Plate—A Scanning Electron Microscope Study. *Journal of Bone and Joint Surgery*, 69A:1212–1220.

Klepinger, L. 1979. Paleopathologic Evidence for the Evolution of Rheumatoid Arthritis. *American Journal of Physical Anthropology*, 50:119–122.

Leisen, J., H. Duncan, J. Riddle, and W. Pitchford. 1988. The Erosive Front: A Topographic Study of the Junction between Pannus and the Subchondral Plate in the Macerated Rheumatoid Metacarpal Head. *Journal of Rheumatology*, 15:17–22.

Mitchell, D.M., and J.F. Fries. 1982. An Analysis of the American Rheumatism Association Criteria for Rheumatoid Arthritis. *Arthritis and Rheumatism*, 25: 481–487.

Ortner, D.J., and C.J. Utermohle. 1981. Polyarticular Inflammatory Arthritis in a Pre-Columbian Skeleton from Kodiak Island, Alaska, USA. *American Journal of Physical Anthropology*, 56:23–31.

Resnick, D., and G. Niwayama. 1981. *Diagnosis of Bone and Joint Disorders*, vol. 2. Philadelphia: W.B. Saunders.

Rogers, J., I. Watt, and P. Dieppe. 1981. Arthritis in Saxon and Medieval Skeletons. *British Medical Journal*, 283: 1668–1670.

Ropes, M.W., G.A. Bennett, S. Cobb, R. Jacox, and R.A. Jesspar. 1958. Revision of Diagnostic Criteria for Rheumatoid Arthritis. *Bulletin of Rheumatic Diseases*, 9:175–176.

Thould, A.K., and B.T. Thould. 1983. Arthritis in Roman Britain. *British Medical Journal*, 287:1909–1911

Paleopathology of rheumatism in paintings

Jan Dequeker

The pathology and history of rheumatism and especially of rheumatoid arthritis are still poorly understood. Insight regarding the pathology may be enhanced through perspectives provided by the history of disease. Short (1974), in his review of the historical documents and reports published on paleopathological specimens, was unable to find convincing evidence for rheumatoid arthritis earlier than the 17th century A.D. prior to Sydenham's description of the disease in 1676. This observation led him and many others to speculate that rheumatoid arthritis is a recent disease, perhaps reflecting recent changes in the human environment. Thus the clarification of the history of rheumatoid conditions in human populations is of considerable anthropological interest.

Ankylosing spondylitis and some types of erosive polyarthritis were present from earliest times (Ortner and Utermohle 1981). A few examples of what could have been mild rheumatoid arthritis have been discovered, but the proliferative, erosive disorders with spinal involvement (possibly psoriasis or Reiter's syndrome) seem more common. Have the rheumatic diseases changed? is rheumatoid arthritis difficult to identify? or is it a relatively recent disease? Because the question remains open I have directed my interest in paleopathology of rheumatism to paintings, which may disclose soft tissue evidence of diseases of ancient time, which are poorly seen in skeletal remains (Dequeker 1977). The use of the visual arts, for example paintings, as a tool for paleopathology research has advantages and disadvantages which should be kept in mind clearly.

The advantage is that rheumatic diseases affect primarily soft tissue (synovia, tendons and cartilage) and only secondarily after many months and years of disease can bone lesions be detected. In paintings and sculptures the deformities due to soft tissue swelling, tendon contractions, and joint subluxations, resulting in discomfort and disabilities, can be discerned and detected by an experienced clinician.

The disadvantage of visual arts is that the artists do not necessarily make portraits of their subjects and may alter anatomical characteristics according to their "feeling" at the time of their work. While a visit to a museum may seem to yield a rich trove of medical illustrations, things are not always what they seem. Diagnostic acumen applied to paintings can be misleading if not tempered with a knowledge of artistic conventions (as mannerisms) and historical context (Ehrlich 1987).

Taking into account the above restrictions, I have the privilege to study paintings made by famous Flemish medieval artists who lived in the area where I practice my speciality of clinical rheumatology.

Paleopathological findings of rheumatism in paintings will now be reported. The adage that one only sees what one knows is certainly applicable to this field of research. A large clinical background in rheumatology is necessary to recognize early clinical features, and to put them in context to make a firm diagnosis.

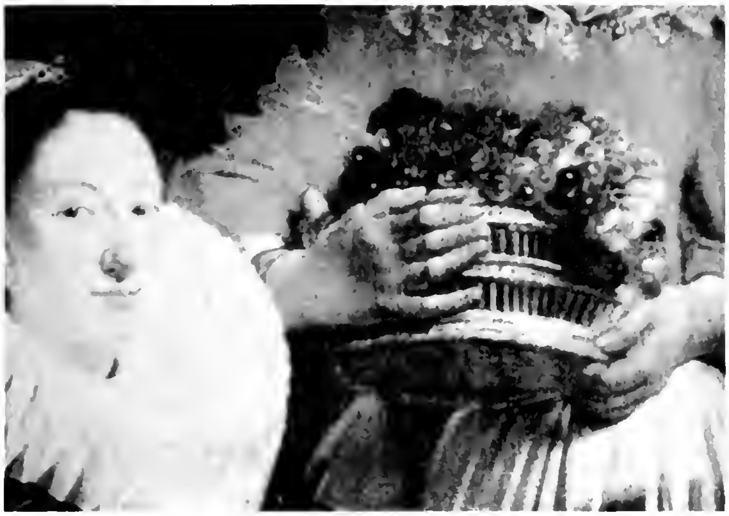
Rheumatoid arthritis

Perhaps the most convincing evidence of rheumatoid arthritis in paintings is the arthritis of the housemaid of Jacob Jordaens (1593–1678) as seen in the painting of Jordaens's own family (Figure 1).

In order to convince people inexperienced in this field, I have chosen a picture (Figure 2) of a hand of one of my rheumatoid arthritis patients of about the same age, disclosing the main features: swelling of the metacarpophalangeal joints, the proximal interphalangeal joints, and the wrist. This example of rheumatoid arthritis is sufficiently demonstrative so that it has been chosen to illustrate the latest, authoritative textbook of rheumatology in Great Britain (Scott 1986).

Another yet unpublished but very characteristic hand and wrist deformity of rheumatoid arthritis is one I found recently at the Escorial Museum near Madrid in the painting of an anonymous artist of the Dutch school of the mid 15th or early 16th century.

A number of other deformities resembling features seen in rheumatoid arthritis have been discovered in the painting of Jan Rombauts (ca. 1500) "Christ appearing to St. Peter" (Leuven, Stedelijk Museum); in the portrait by Joos (Justus) Van Gent (1430–1475) of Federigo de Montefeltre (Urbino, Ducal Palace); in the drawing by Jan Van Eyck (ca. 1411) of John IV, Duke of Brabant; and in the painting "The Donators" (Brussels) (1525–1530) by Jan Gossaert, also called Mabuse, showing flexion contraction of the second, fourth, and fifth finger of the left hand (Figure 3).



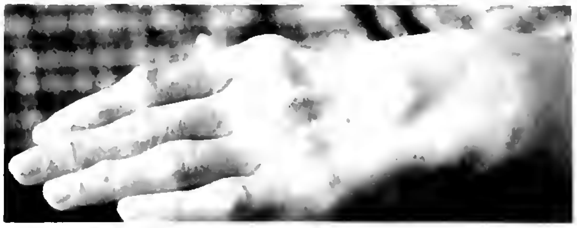


FIGURE 2. Synovial swelling of proximal interphalangeal, metacarpal, and wrist joints in a patient suffering from rheumatoid arthritis.

FIGURE 1. Jacob Jordaens: The painter's family (detail); Madrid, Prado. The maidservant's right hand shows swelling of metacarpal and proximal interphalangeal joints.

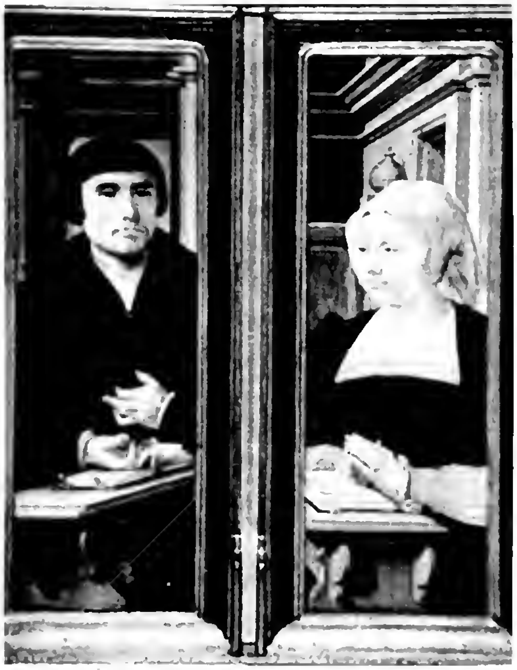


FIGURE 3. Jan Gossaert (Mabuse): The Donators; Brussels, National Museum. Flexion contractures of the man's right hand.

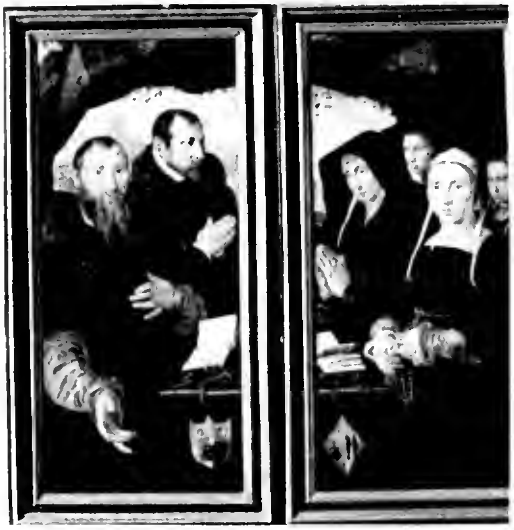


FIGURE 4. M. van Heemskerck: Altar Panels with Donors; Vienna, Kunsthistorisch Museum. Rheumatoid deformities of the fingers and atrophy of the dorsum of the hand.

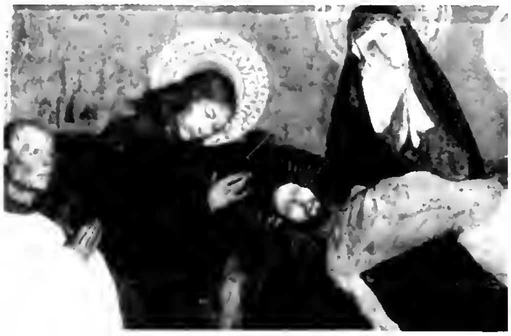


FIGURE 5. Southern French Master: Avignon Pieta; Avignon. Twisted fingers of St. John's right hand turned sideways and bent backward.

A few years ago I noticed two other remarkable examples of rheumatoid deformities of the hand in late Gothic paintings: one by M. Van Heemskerck (1498–1574) titled "Altar Panels with Donors" (Figure 4) and the Avignon Pieta (±1470) at the Louvre in Paris, by a Southern French Master entirely under the influence of the Flemish School (Figure 5). The left hand of one of the donors and the right hand of St. John show grossly deformed joints with fingers twisted and turned sideways or bent backwards.

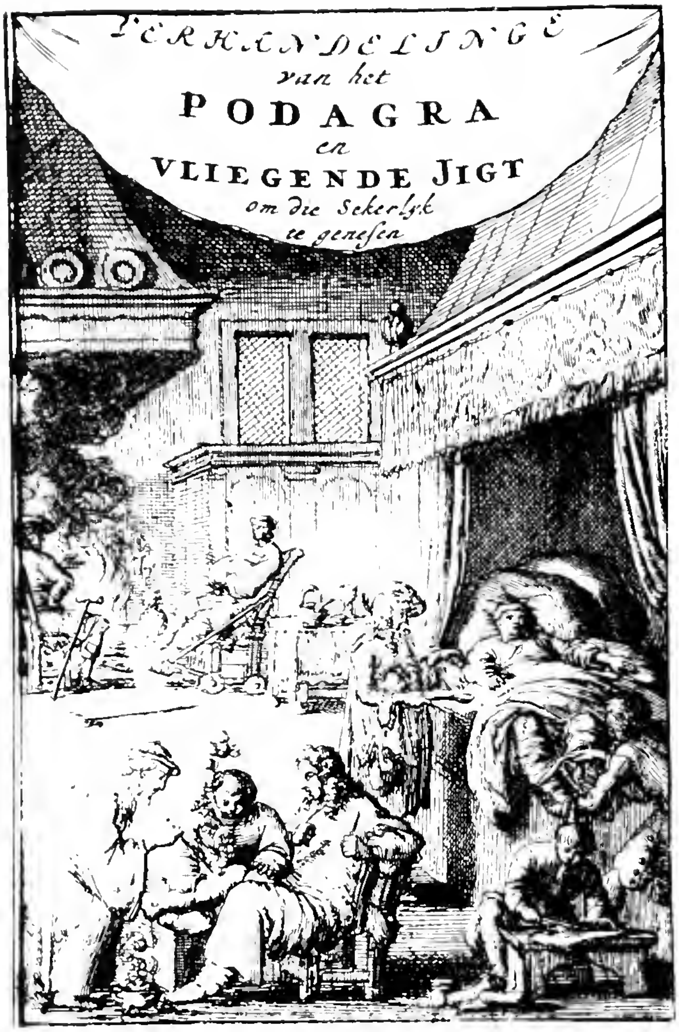


FIGURE 6. St. Blankaart: Front page of book on podagra and gout. The young man sitting in a wheelchair has an ankylosed back and knee joint.

Ankylosing spondylitis

During my search for rheumatic diseases in visual arts I have encountered only one example of possible ankylosing spondylitis. In the front page of the work of St. Blankaart (1684) "Van het podagra en vliegende jigt" on podagra and gout, a young male sits with a stiff back in a wheelchair with ankylosed knee and possibly ankylosed hip, while another male sits in a chair with a walking stick near a fire (Figure 6). Of the other two patients, one lying in bed is being bandaged around the legs and receiving medicine in a spoon, while the other in the foreground is undergoing cauterizations at his knee. Although they all seem to suffer from rheumatism, it is only the one in the wheelchair who can be identified as a probable case of ankylosing spondylitis. The others could be polyarticular gout or any other rheumatic disease entity involving one or several joints.

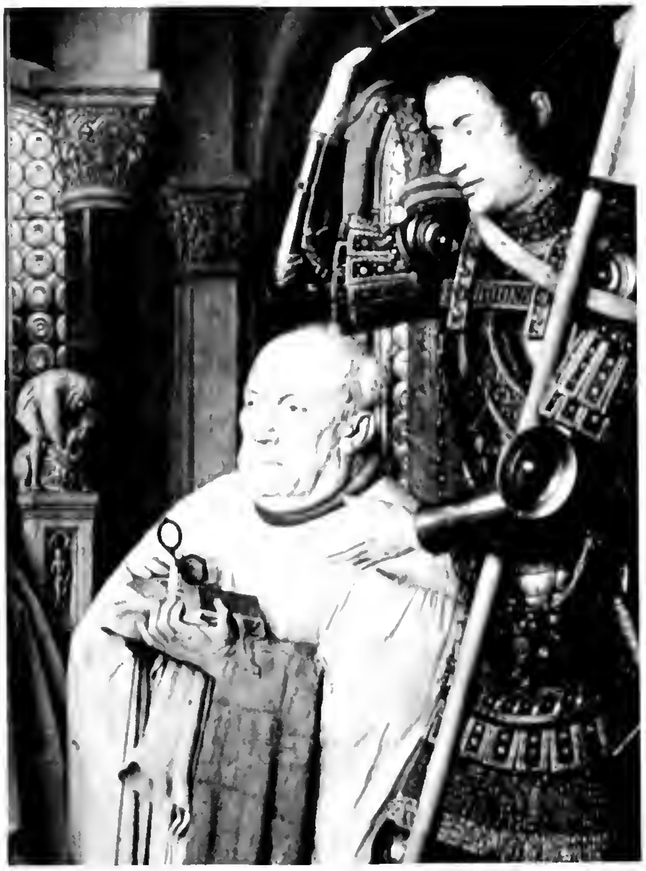


FIGURE 7. Jan Van Eyck: The Virgin with the Canon (detail); Bruges, Municipal Museum. Temporal arteritis—polymyalgia rheumatica.

Polymyalgia rheumatica

Indirect indications of polymyalgia rheumatica, a rheumatic inflammation characterized by shoulder and hip girdle muscle stiffness and often associated with temporal arteritis, are seen in the painting by Jan Van Eyck (ca. 1385–1440) of the Holy Virgin with Canon Van der Paele (Figure 7). The Canon is clearly suffering from temporal arteritis, with scar formation and loss of hair of the eyebrow and in front of the left ear. For the sake of completeness, it should be mentioned that he also has a cellular mole and a sebaceous cyst on the left ear.

Historical data on Canon Van der Paele published in the Canadian Medical Assoeiation Journal (Dequeker 1981) support my clinical diagnosis of polymyalgia rheumatica. According to the minutes of the cathedral chapter, he began having difficulty in attending the morning service in November 1431. By the time the painting was done (1434), the aging Canon was forced to stay home, first in the morning and later for the whole day, because of rheumatic pain with morning stiffness, general weakness and ill health. This illness was not fatal, however, and he survived the first symptoms for 12 years—a history compatible with the natural course of polymyalgia rheumatica.

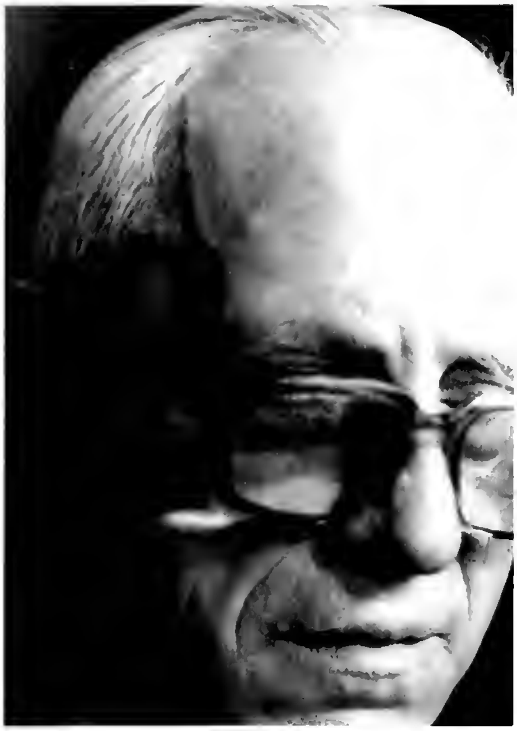


FIGURE 8. Patient suffering from biopsy-proved temporal arteritis and polymyalgia rheumatica.

In order to illustrate the great realistic capacity of Jan Van Eyck, and the likelihood of our diagnosis, the clinical picture of a patient with identical features at the temporal region and whose biopsy showed giant cell arteritis is shown (Figure 8).

Recently I discovered that a German dermatologist, Roth (1969), had made the same diagnosis in 1969. Meige (1924) described vascular abnormality in the temporal region in four paintings, but did not mention the painting of Jan Van Eyck.

The Canon was probably not the only sufferer from this disease during these centuries. Signs of temporal arteritis can be seen in Piero di Cosimo's Portrait of Francesco Gambetti (1505), now in the Rijksmuseum, Amsterdam.

Osteoarthritis

In another painting by Jan Van Eyck, "The Lamb of God" retable (Ghent), I found one of the few convincing evidences of osteoarthritis in paintings. The distal interphalangeal joint of the thumb of St. John Baptist shows clearly Heberden nodes (Figure 9), the overall landmark of generalized osteoarthritis as shown in a picture of a contemporary patient (Figure 10). A similar Heberden-like (knucklepad-like) swelling around the joints can be seen in two paintings by Bernardo Strozzi (1581–1644): "The Old Coquette" (Moscow) and "The Lute Player" (Vienna). This prominence at the bending of fingers might be a convention of mannerism as suggested by Ehrlich (1987).

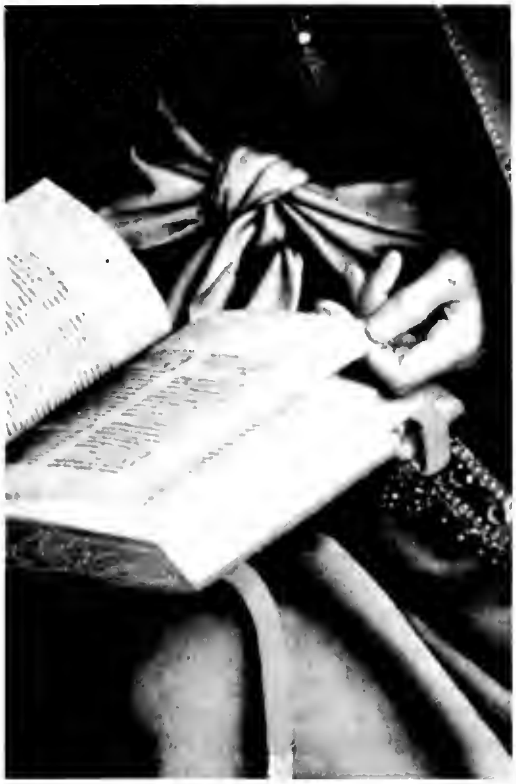


FIGURE 9. Jan Van Eyck: The Lamb of God retable (detail); Ghent, St. Bavo Cathedral. Heberden nodes on the thumb of St. John Baptist.

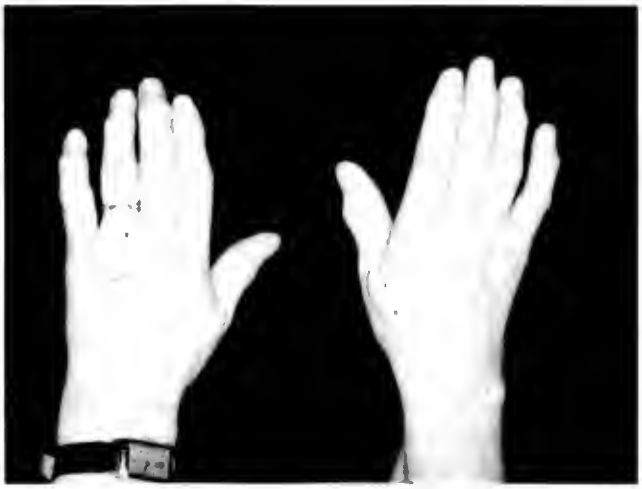


FIGURE 10. Patient with Heberden nodes at the distal interphalangeal joints.

Discussion

Since the publication of my first findings in the British Medical Journal in 1977, and more extensively in Organorama in 1979, two further papers on rheumatoid arthritis in art have been published.

Appelboom and associates (1981) described a peculiar tendency of Peter Paul Rubens (1577–1640) to paint apparent swelling of the wrists and deformities of the hands, and it has been suggested that either he or his second wife, Helena Fourment, or both, may have had rheumatoid arthritis. Indeed, biographical information on Rubens suggests that he suffered from rheumatism. In his abundant correspondence, the term "gout" is used to define the recurrent pains and swellings afflicting his hands and feet.

Alarcon-Ségovia and associates (1983) pointed out that the "Portrait of a Youth," painted in 1483 by the Florentine artist Sandro Botticelli, has features of rheumatoid arthritis in the hand of the subject, who would be young enough to be considered as having juvenile arthritis. This painting has also been discussed by Short (1974) as a possible example of a rheumatoid arthritis hand, although he admits that it also could be an artistic convention or stylistic trait, since Botticelli's hands often have this appearance. When painting an extended hand Botticelli, like many other painters of the Renaissance, placed both middle fingers together and the second and fifth apart, as if to relieve the monotony the fingers would create if all were spread. A bent fifth finger is often encountered not only in Botticelli's paintings but also in the paintings of Rogier van der Weyden and many others.

In another painting by Botticelli, "The Birth of Venus," I recently described swelling of the proximal interphalangeal joints and a sausagelike swelling of the left index finger (Dequeker 1984). Is it significant that the fingers of the right hand point in the same direction as the drifting hairlocks?

An interesting anecdote is that the model for Venus was a well-known individual, namely the 16-year-old Simonetta Vespucci, girlfriend of Giuliano di Piero, brother of Lorenzo il Magnifico. Simonetta died a few years later from tuberculosis (Alarcon-Ségovia 1985).

It is obvious that caution is called for in drawing medical conclusions from paintings and engravings. This is particularly the case when the hands are taken as the basis, for hands are often used by painters as a means of expression for feelings and sometimes as the hallmark of a particular school of painting. For example, it is striking to note that many fingers in the canvases of Rogier van der Weyden show very fine long fingers, often with a clinodactylic deformation of the little finger.

It is also well known that most of the figures in El Greco's paintings show a picture of Marfan's syndrome. The abnormalities described in this article are of a different nature. They are not the consequence of a style, except perhaps for the hands in the painting by Jan Gossaert; these could point to an expression of a mannerism typical of this artist. Similar deformations of the hands can be seen in a portrait of a woman in the Rijksmuseum in Amsterdam and in that of the man with the garland of roses in the National Gallery in London. Both of these works are ascribed to Jan Gossaert.

Probably a rheumatologist sees more in the pictures of cripples than another doctor or a layman because of the knowledge he has acquired in daily practice. What we do not know, we do not see. Although none of the deformations and swellings of the joints which have been found constitute irrefutable examples of rheumatoid arthritis, they nevertheless give grounds for a strong suspicion that polyarthritis occurred so frequently in the Middle Ages that it must have caught the attention of the masters, and this at a period in which infectious diseases such as leprosy and tuberculosis dominated pathology in every field. Arthritis was recognized in all sections of the population, among rich and poor, and men and women alike.

Ten out of 24 paintings, described in full elsewhere, in which arthritic lesions were recognized, represent donors or well-known personalities of whom a "portrait" was made (Dequeker 1987). This supports the idea that in fact these individuals might have been suffering from rheumatic diseases. Almost all of these paintings were in the time of the Flemish realistic school influence, introduced by Jan Van Eyck. Since scrupulous recording like this of wrinkles, veins, warts, stubble on the chin, and congenital malformations are typical for 15th–16th century Flemish art, the rheumatoid-like hand deformities cannot be ascribed to carelessness, incompetence, or mannerism of the painters.

It is thanks to the realism of the Flemish, Dutch and Italian schools of the late Gothic era that we can recognize these deformities. During the Renaissance and the Baroque the figures were so idealized and perfected that signs of disease are seldom recognizable in the works of these periods. Although the paintings in those earlier days were generally commissioned by patrons who also figured in them, the painters did not always spare their benefactors, as we saw in Jan Gossaert's and Van Heemskerck's paintings, and in the portraits of Federico da Montefeltro, Michelangelo, Canon Van der Paele, Jordan's Servant, Gambetti, Aegidius, Erasmus, and Vespucci.

These descriptions and pictures do not, of course, provide any scientific proof that rheumatoid arthritis and associated systemic disorders occurred frequently in the Middle Ages. But the argument that rheumatoid arthritis does not occur in old paintings equally does not provide scientific proof for the assertion that rheumatoid arthritis is a recent or modern disease. The main reason why they were not noted historically is that the mean lifespan was too short for a sufficient number of cases to develop and be recognized as examples of a specific disease. Rheumatoid arthritis was rare because age expectation was low, and potential sufferers died before contracting the disease. There is already evidence that this is a reason for the low prevalence of rheumatoid arthritis in underdeveloped countries. In contrast, ankylosing spondylitis is historically well-documented in human remains because this inflammatory rheumatic disease preferentially affects younger people. The discoveries of rheumatic disorders in the paintings and drawings of the Late Middle Ages described here nevertheless make the current view (that rheumatoid arthritis is a relatively recent complaint) a topic for discussion.

Literature cited

Alarcon-Ségovia, D. 1985. Botticelli and Arthritis. *Arthritis and Rheumatism*, 28:600.

Alarcon-Ségovia, D., A. Laffon, and J. Alcocer-Varela. 1983. Probable Depiction of Juvenile Arthritis by Sandro Botticelli. *Arthritis and Rheumatism*, 26:1266–1268.

Appelboom, T., C. de Boelpaepe, G. Ehrlich, and J.P. Famaey. 1981. Rubens and the Question of Antiquity of Rheumatoid Arthritis. *Journal of the American Medical Association*, 245:483–486.

Dequeker, J. 1977. Arthritis in Flemish Paintings (1400–1700). *British Medical Journal*, 1:1203–1205.

______. 1979. Rheumatism in the Art of the Late Middle Ages. *Organorama*, 16:9–19.

______. 1981. Polymyalgia Rheumatica with Temporal Arteritis as Painted by Jan Van Eyck in 1436. *Canadian Medical Association Journal*, 124:1597–1598.

______. 1984. Arthritis in the Paintings of Sandro Botticelli. *Arthritis and Rheumatism*, 27:1196–1197.

______. 1987. Rheumatic Diseases in Visual Arts. In T. Appelboom, ed., *Art, History and Antiquity of Rheumatic Diseases*, 31–40. Brussels: Elsevier Librico.

Ehrlich, G.E. 1987. The Cover. *Journal of the American Medical Association*, 257:892.

Meige, H. 1924. *Presse Médicale*, 26 April.

Ortner, D.J., and C.J. Utermohle. 1981. Polyarticular Inflammatory Arthritis in a Pre-Columbian Skeleton from Kodiak Island, Alaska, USA. *American Journal of Physical Anthropology*, 56:23–31.

Roth, W.G. 1969. Arteriitis temporalis dargestellt an einem Gemalde des Reichsmuseums in Amsterdam. *Der Hautarzt*, 20:330–332.

Scott, J.T. 1986. *Copeman's Textbook of the Rheumatic Diseases*, vol. 1. 6th edition. New York: Churchill Livingstone.

Short, C.L. 1974. The Antiquity of Rheumatoid Arthritis. *Arthritis and Rheumatism*, 17:193–208.

SUMMARY OF AUDIENCE DISCUSSION: Attempts to trace the history of rheumatoid arthritis to periods earlier than A.D. 1650 are frustrated by the style of painting prior to that date. Differences in pattern and symmetry assist the separation of rheumatoid arthritis in paintings from psoriasis or Reiter's syndrome to some degree but not infallibly.

Trauma

Trauma and treatment in the British Isles in the Historic Period: A design for multidisciplinary research

Charlotte Roberts

The study of disease processes in past populations has had a long history stemming from 19th century investigations. The skeletal remains of our ancestors have always attracted attention and interest from scholars and amateurs alike, often with a somewhat startling obsession. Lay attitudes to study of human remains have also varied through time up to the present day; there is now little room or finances for superficial, unplanned study yielding results insignificant with regard to the archeology of the population as a whole. In addition, current problems concern the ethical aspects of the scientific study of recent, and even more distant, ancestors of indigenous populations, notably the Aboriginal communities of Australia (Webb 1987). Prevention of study of ancestral remains and their removal from museums for reburial should be an issue of concern to all paleopathologists. The Paleopathology Association is at present heavily involved with this reburial issue (Hart and Ubelaker 1987).

Paleopathology has, however, advanced over the years in terms of its credibility as a science and its recognition by archeologists as a valuable tool in reconstructing the past. It need hardly be said that the remains of people who lived many thousands of years ago are the nearest evidence which anybody can hope to obtain to rebuild a valid picture. Nevertheless, the discipline has some way to go before it is recognized and accepted as a useful tool for reconstruction of ancient societies. At times paleopathological studies, in Britain at least, are criticized as being "unco-ordinated and desperately understaffed; therefore there is little possibility of constructive exchange of views between the paleopathologist and archeologist" (Cramp 1983:19). Obviously this is not the case for all countries where perhaps funding for research is more readily available, thereby providing an environment conducive to this interchange of ideas.

Paleopathology is but one aspect of interpreting the structure of past societies and, although contributing significantly to this aim, it cannot survive alone and in isolation from other disciplines in archeology. Alone it provides little constructive and interpretable data. Every aspect of archeological studies can contribute something to an understanding of the complexities of the lives of peoples in antiquity. As Arnold said (1986:38), "Specialists must continue with their research; none need, nor has any right, to believe that their specialization is superior to any other; each must feed their data, observed patterns and generalizations to those who seek to take a more generalized overview of past human behavior, an overview which must incorporate all types of evidence."

All experts in their own fields must collaborate to create an accurate interpretation of the society they are studying. One should try to use even fragmentary data, because, as Bisel and Angel said (1985:198): "It is by consideration of all factors together that more reliable conclusions can be drawn." Reece (1982:348) clearly is in agreement with this statement when he bemoans the lack of integration of all aspects of cemetery studies: "I have yet to find a report which integrates all these facts, and it is the separation of bones from bodies, and bodies from cemeteries and finds, that causes my dis-ease." Reece found a general failure to integrate human bone reports from Roman cemeteries in Britain with the cemetery report as a whole. He emphasizes (1977:355) "the incredible stupidity of digging a cemetery and then classing the major source of information as an optional appendix."

Attempts at using integrated multidisciplinary evidence to reproduce particular aspects of a society, such as diet and changes in economy, have been successful in America (Gilbert and Mielke 1985; Cohen and Armelagos 1984) and anthropologists have advocated a close working relationship between them and archeologists (Osborne 1969), preferably in departments employing workers in both disciplines. Interpretation and understanding should be the ultimate aim of studying all types of archeological and related data.

The following paper shows an attempt to use many different types of evidence to reconstruct how well or how badly past peoples managed injuries to the long bones and skull.

Paleopathology by its very nature relies on diagnosis based on modern clinical medical method, and interpretation of data in the ideal world has to be done with reference to clinical texts and experience. The diagnosis and interpretation of trauma is, of necessity, heavily reliant on radiographic analysis, and this particular research is based very much around this theme. Correlation with other classes of evidence is without doubt essential to the potential success of interpretation of the data.

History of trauma studies

Trauma can be defined as any bodily injury or wound and may affect the bone and/or soft tissues of the body. Fractures can be defined as the result of any traumatic event which leads to a complete or partial break in the continuity of bone. Trauma is a common affliction of modern lifestyles, as it was in the past, and people do, and did, react to it in a variety of ways. Trauma is painful, visual and debilitating, and Withers (1960:1) perhaps summarizes these thoughts on the implications of trauma: "In times of stress, pain or of sorrow, the human being will go to any length to try and find help." In human skeletal studies it is difficult to be precise in aspects such as complications of particular fractures but it is essential to be aware of these potential complexities. Applied anatomical studies in paleopathology should be one of the first considerations.

Data on fractures for paleopathologists in past years has appeared in four forms: (1) as part of a bone report (i.e., incidence of fractures), (2) as a specific study on fracture patterns in particular populations, (3) as a study of fractures of a specific bone, and (4) as part of a treatise on paleopathology.

The quality and quantity of analysis and interpretation of trauma is determined by the worker's preferences or the archeologist's requirements for a bone report. In many cases basic descriptions of skeletons present is all that is provided or required. In other cases a more detailed report is appropriate. All human skeletal reports describe any traumatic lesions observed in the populations (e.g., Wells 1982:161). Some literature has also appeared which exclusively describes trauma in a specific population (e.g., Zivanovic 1984; Lovejoy and Heiple 1981; Jurmain this volume). In Britain, occasional publications specifically on ancient skeletal trauma have also appeared (see Manchester and Elmhirst 1980; Manchester 1978; Courville 1965) but these have tended to concentrate on the wound appearance and the potential weaponry causing the injury.

Case studies of unusual pathological lesions (e.g., Roberts 1987) have been and always will be prolific in paleopathology, as the method is an efficient way of transmitting information. Studies concerning trauma have also appeared in this type of literature. Although useful, there is little possibility for synthetic study from isolated reports.

Several books deal with paleopathology generally (Bennike 1985; Ortner and Putschar 1981). Obviously, these volumes discuss trauma, but the very widespread nature of paleopathology precludes expansion of data on trauma into a more valid interpretation for human skeletal studies.

In modern clinical literature the contributions to traumatology are prolific and make comparison between modern and ancient trauma possible. Incidence rates of fractures, sex ratios and age ranges, causes, types of treatment, healing, and complications are all well documented. This provides an, as yet, unrealized potential for comparing ancient fractures with modern data.

Present study

The potential for the study of trauma in populations has not yet been fully appreciated. While trauma in osteoarcheological analysis is universally reported, there is often little expansion of the basic data. Fortunately, in Britain extensive documentary research and archeological excavation and interpretation has led to a considerable archive of information which can be utilized to supplement human skeletal observations not only in trauma but in many other paleopathological studies. Obviously, limitations have to be realized, and collaboration between the skeletal specialist and specialists conversant with all types of evidence being used is essential. As Daniels (1978:28) stated, " Archaeology has become a subject too complex for the simple polymath to handle."

Considerable thought about trauma studies, developed by the author in 1983, led to a proposal for research. The hypothesis to be tested was as follows: Populations were capable of and had the technology and intelligence to treat trauma in antiquity.To test this hypothesis the research design encompasses: (1) study of dry bone evidence for trauma in the form of fractures of long bones (humerus, radius, ulna, femur, tibia and fibula) and the skull, using macroscopic observation and radiography; (2) correlation of this data with other classes of evidence to illuminate the knowledge of treatments and technology used in each time period from the Roman to late and post-Medieval eras.

The rationale for the selective study of long bone and skull fractures is that injury to these areas of the body would be more detrimental to the individual's well being than, for example, a fracture of the rib, clavicle, or a peripheral bone. Trauma is extremely devastating not only skeletally but generally to the body system.

The main sources of evidence used were human skeletal remains, modern clinical comparative data, secondary documentary sources, ethnographical studies, art forms, and archeological data such as surgical artifacts, environmental data indicating hygiene, living conditions and diet relevant to healing of fractures.

LIMITATIONS OF THE EVIDENCE

There are a number of significant restraints on the data for these classes of evidence but the main areas become readily apparent. They are briefly reviewed here and discussed in more detail later in the paper.

FRAGMENTARY SKELETONS. Investigation of a large number of cemetery sites from different areas of the British Isles was considered essential to obtain a valid picture of trauma. However, the number of cemetery sites examined precluded estimation of an absolute incidence of particular fractures. Data was not available for counts of individual bones for most of the sites. First, it was not feasible in terms of time to undertake this work as part of the research, and second, it was felt that it was not a particularly relevant area of study for the subject matter of this project.

AGE AT FRACTURE. Age-specific incidence rates of fractures have been quoted by many workers in human skeletal reports but, in fact, these data mean little. Fractures evident on skeletons could have occurred at any time prior to death. There is currently no method of estimating at what age a fracture occurred in ancient skeletal material beyond about one year after the injury, unless the fracture occurred close to death and there is extant evidence of very new bone formation or primary callus.

SUBADULT FRACTURES. Greenstick fractures occurring in childhood, even in modern populations, may heal and remodel so well that the original fracture line may not be visible on x-ray. In ancient populations these fractures may not be recognized even macroscopically.

RECENT ANTEMORTEM FRACTURES. Fractures occurring shortly before death may be difficult to distinguish from postmortem fractures of bone due to burial and/or excavation.

STRESS FRACTURES. Stress fractures will not be identified unless all the bones of every skeleton are x-rayed. Even so, many will not be evident on an x-ray.

SKULL INJURIES AND BRAIN DAMAGE. Consideration of skull injuries and their potentially associated brain damage can be problematical. Some types of head injuries induce contrecoup damage to the brain while others produce direct damage to the subjacent brain. In addition, complications of skull injury can be complex and multifactorial.

TREPANATION. In some cases it is difficult to distinguish between postmortem holes in the skull and trepanation and, of those trepanations which have no injury associated with them, one can only speculate on the raison d'etre.

ART AND LITERATURE. Representations of past events in art or literature are inevitably controlled by the author's or artist's preferences or interpretation, and their validity as evidence of disease or therapy is questionable.

MODERN PRIMITIVE SOCIETIES. These populations are separated in both space and time from ancient British societies but they may be comparable in terms of their disease concepts and treatment.

ARCHEOLOGICAL EVIDENCE. Interpretation of archeological evidence is controlled by the remains studied. All material remains are a sample of what was actually deposited in the ground and many factors determine their survival and excavation. For example, splints used for fractures were probably constructed from biodegradable material, such as wood, and either do not survive to be excavated due to site-specific soil conditions or were not buried with the skeleton. Most fractures seen by the paleopathologist are healed and therefore do not need to remain splinted at death and burial.

Notwithstanding the limitations present in all forms of archeological research, the potential for the study of traumatic lesions in human skeletal remains is considerable.

This paper does not present a definitive analysis of results because the research program is still in progress. This report, which is therefore interim, seeks to outline the sources of evidence used and the rationale thereof, the observational methodology and the criteria of analysis of those sources.

METHOD

SKELETAL EVIDENCE

The skeletal evidence for bone fractures is extensive as shown by previous analysis of human remains. This evidence occurs in varying incidence throughout different populations and periods of time, although there is, as yet, little data available on fracture incidence in the past.

Location of skeletons in museums, archeology and anthropology departments in universities, and in archeological units was often difficult. A total of approximately 30,000 individuals were located in institutions around Britain. Approximately 6000 individuals have been examined to date. An attempt was made to look at equal numbers of skeletons from the time periods of interest. Table 1 lists the cemetery populations studied (Figure 1). The Roman, Anglo-Saxon, Medieval, and post-Medieval periods were chosen for study because of the availability of skeletal material and contemporary documentary, art, and archeological evidence for these eras. The availability and numbers of prehistoric remains in Britain would not give a representative study, and additional types of evidence of prehistoric context are not always available.

TABLE 1. Total numbers of skeletons examined

Roman		*Anglo-Saxon*		*Medieval*	
Castle Street, Chester	2	Hamwih, Southampton	104	Hickleton	60
Margidunum	15	Binchester	36	Barton Bendish	80
College of Art, Gloucester	29	Willoughby-on-the-Wolds	62	Exeter	226
Kingsholm	8	Empingham	16	Rand Church	12
Derby Racecourse	71	Stretton-on-Fosse	55	Nuns Field, Chester	18
Rudston	28	Little Chester	61	Greyfriars Court, Chester	40
Walkington	18	Leaden Hill	41	Thornhill	6
St. Bartholomew's, London	34	Rangoon, London	2	Rhuddlan	47
Cutlers Gardens, London	18	Pewsey	101	Norton Priory	149
Woodcutts	14	Collingbourne Ducis	28	Austin Friars, Leicester	25
Rotherly Down	14	Winklebury Hill	28	Elstow Abbey	20
Woodyates	19	Cheddar	2	Newark	80
Wor Barrow	22	Bedhampton	76	St. Oswalds Priory, Gloucester	50
Ilchester	45	Portchester Castle	21	St. Giles Cathedral, Edinburgh	108
Knowle Bardnip	1	Snell's Corner	45	York Minster	269
Compton Pauncefoot	3	Portsdown Hill	25	West Malling	236
Taunton	10	Berinsfield	88	Stratford Langthorne Abbey	121
Lamyatt Beacon	4	Eccles	150	Cruden Bay	36
Bradley Hill	23	Raunds	300	Linlithgow	191
Charlton Mackrell	14	Tanners Row	200	Kirk Hill	296
Northover	8	Misc.	13	Stonar, Sandwich	149
Hicknell Slait	1			Holy Trinity Priory, London	9
Chester Road, Winchester	92			Billingsgate	56
Victoria Road, Winchester	201			Aberdeen	121
Western Suburbs, Winchester	14			St. Helen on the Walls	400
N. R., Winchester	22				
Hyde Street, Winchester	26				
District, Winchester	14				
Gambier-Parry Lodge, Gloucester	93				
Beckford, Worcestershire	32				
Baldock, Hertfordshire	119				
Cirencester	435				
Misc.	20				

Numbers of fractures identified to date are in Table 2. These numbers will be supplemented with examples from a further five cemeteries by the completion of the research. Sex and age of the individuals will not be discussed since they are irrelevant at this stage.

Several problems with the skeletal material were encountered. Some institutions did not know what collections they had, and occasionally could give only the numbers of skeletons under their care and not the cemetery names. In many cases the bones were not clean and were therefore unsuitable for examination. Some collections had been divided up and stored in several places, and some pathological bones had been separated from the rest of the body. These problems prevented more efficient use of time when visiting the institutions concerned.

TABLE 2. Number of fractures identified

	Roman	*Anglo-Saxon*	*Medieval and later*
Skull	18	14	24
Humerus	2	2	6
Radius	10	6	10
Ulna	8	2	7
Femur	4	5	1
Tibia	5	5	5
Fibula	20	9	13
Multiple	14	6	7

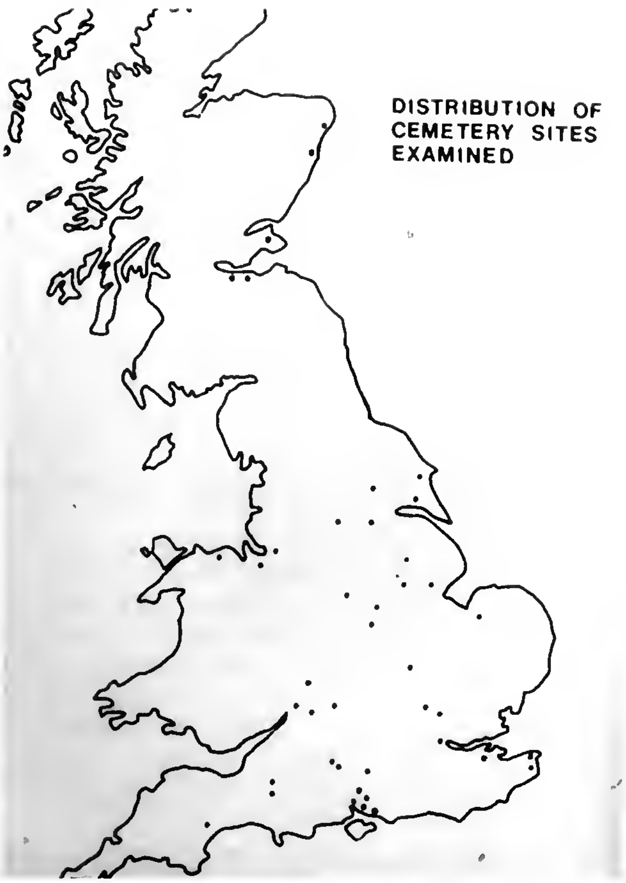


FIGURE 1. Distribution of cemetery sites examined. Note that some areas represent more than one site.

Burial conditions obviously determined how complete the collections were. On some sites the soil pH was so low that much of the skeleton was eroded and some bones were not present for examination. This, among other factors, determined the number of traumatic features observed.

CLINICAL DATA

The study of trauma in past populations is, of necessity, linked to diagnostic criteria in modern populations. Fortunately, in contrast to ancient studies of populations, modern studies of trauma are abundant in the literature. However, clinicians have far more data to study including, in many cases, a cooperative and talkative patient and clinical notes on which to base diagnosis and treatment. The clinician also has modern diagnostic tools and continuing development of new methods and drug therapy to aid treatment.

In ancient populations the researcher is limited to a study, with whatever means available, of the dry bone evidence for trauma and treatment. Use of only one source of evidence for reconstructing particular aspects of society has many inherent limitations, not least a biased picture.

The use of modern clinical data provides the opportunity to interpret the ancient material with a greater degree of accuracy than would otherwise be possible without it. Modern trauma studies appear in five forms: (1) papers on fracture patterns in populations, e.g., Fife and Barancik 1985; (2) fractures of specific bones, e.g., Einarsson 1958; (3) fractures in specific age groups, e.g., children, Sharrard 1979; (4) complete chapters in clinical textbooks e.g. Watson-Jones 1976; (5) specific features of fractures, e.g., Watson-Jones and Coltart 1982.

Studies of fracture patterns in modern populations are invaluable for workers in the field of paleopathology. This is particularly relevant for the subject of etiology. The different types of fractures observed in modern populations are usually correlated with specific forces acting on the bone. For example, an oblique fracture is usually caused by an indirect force. In some cases an occupational causation may be postulated (see Merbs 1983).

Modern studies of fractures of specific bones are usually concerned with incidence, causes (by age and sex), healing and treatment, which are all subject areas comparable to ancient skeletal material. Particularly relevant is the comparison of treatment and healing between ancient and modern populations; were ancient populations producing good results from their fracture treatments when compared to modern healing and treatment?

The modern literature abounds with information on types of fracture (cranial and postcranial), causes, healing, treatment and complications. Reference to this data is essential to the understanding of ancient trauma and treatment.

However, despite being invaluable, modern clinical data has some limitations. Forces producing fractures have remained the same but their actual mode of production has altered with changing technology. The literature is filled with data on fractures caused by high-impact traffic accidents and those caused by other modern technology. These data are not relevant to the paleopathology of trauma. Data on healing of modern fractures are influenced by modern drug therapy, particularly antibiotics, and by modern methods of fracture management such as plates, pins, screws, and bone grafts.

The age at which the fracture occurred and the sex of the patient are known in modern cases of fracture. In paleopathology these details are often not definitely determinable. The age at which the fracture occurred is problematical but would indeed have had an effect on fracture healing. Many other factors must be borne in mind when interpreting the healing of paleopathological fractures using modern data. Environment, hygiene, living conditions and diet in the developed world should be beneficial for efficient healing and should therefore influence healing times and the propensity to infection. In addition, blood vessel and nerve injuries associated with fractures in the paleopathological evidence would have significantly affected how quickly a fracture healed. In modern contexts surgical repair of vascular and neurological lesions would be available and thereby lessen healing time.

SECONDARY DOCUMENTARY SOURCES AND ART FORMS

In the process of using human skeletal remains from the Roman to late and post-Medieval periods, the availability of documentary sources and art forms becomes apparent, although many such sources were produced abroad and the texts translated into English. Sources of documentary and art evidence available for use are books by specific authors (includes herbals; see Rohde 1922); medical manuscripts (e.g., Dawson 1934); illustrations on coins, pottery vessels and frescoes and in manuscripts; and sculpture.

All these sources are secondary to the primary source. The validity of evidence is determined by the author's or artist's "clinical acumen" and by modern interpretation of documents and artforms. Although these sources of evidence are invaluable for workers in this field, they should be used with caution.

Authors may use words which have changed in meaning through time, or the words may describe something of which the people in the past did not know the meaning (Marwick 1970:4). Some words used in everyday speech may actually be confusing when used in a historical context. Translation of certain words from one language to another (e.g., Latin to English) may prove difficult if there is not an exact equivalent of the word. Historical writing is interpretative and there must therefore be a subjective element therein. All authors and artists working in each period portray history influenced by their own interests. Some historians may pay particular attention to certain areas of interest and ignore others. As primary sources are often fragmentary, there is a tendency for conflicting conclusions (Marwick 1970:23). The very nature of the raw material, however, dictates the historian's function to convert it into the finished product by whatever means.

Illustration and sculpture of events in the past also reflect the prevailing and stylistic conventions at the time of production. Before the advent of television, photography, film and printing the only way to convey information was to make a picture of it. The artist or sculptor may produce what he/she wishes to portray to observers and not the true factual record of the event in question.Nevertheless, this source of evidence is invaluable for the study of the treatment of trauma in antiquity and, as Herrlinger (1970:7) said, "A good illustration is often better than a thousand words."

Documentary and art evidence for treatment in the past is abundant in the sources for each period under consideration. Its abundance, however, does vary; illustrations and documentary evidence are, of course, more prominent in the Medieval and later periods rather than in earlier times. However, much of the evidence in the later periods has clear connections with earlier Greco-Roman medicine and surgery as displayed in early texts. A large quantity of relevant documentary material was transferred to Britain from the east by travelers and invaders of the island. However, before texts came to Britain they were mixed with ancient Roman religion, Mediterranean folk elements, and magico-religious ideas from the Far East (Grattan and Singer 1952). Texts were translated into English from Latin, mainly by monastic scribes, and made more accessible to a wider cross-section of society.

In the Roman period much of the medical and surgical knowledge was gained from earlier Greek practice and tradition. By the sixth century B.C. the Greeks had contact with Egypt and had gained much valuable information. Medical dogma, especially in the Anglo-Saxon period, was modified by Celtic elements, southern Italian influences, and Anglo-Saxon tradition. In the Medieval period a new influence came from the Arabic medical world, works which were then translated into Latin.

The synthesis of different sources of information seems to have been a mixture of ideas from Britain and abroad. Much of this medical and surgical evidence can be traced to its original source and therefore differences between primary and secondary sources can be noted. It is necessary to take an overall view of the evidence to assess any inconsistencies which may arise. Nevertheless, there is much value in using these sources of evidence for reconstructing past treatment of trauma.

The use of documentary and art evidence in the context of this research is undoubtedly hazardous. No one person has comprehensive knowledge of all the subject areas which need to be covered. A multidisciplinary approach to trauma and treatment is therefore the basis of the current research. In that respect, there is a need to rely on experts in other fields of study such as art and documentary research, to supply evidence to supplement and integrate with the rest of the data. While the limitations are clear and realized, the evidence will be used.

ETHNOGRAPHICAL DATA

The use of data from modern primitive societies on medical and surgical treatment of trauma is a further method of reconstructing this complex feature of past societies. Direct evidence of medicine and surgery in early man is meager, although secondary sources are abundant. It is reasonable to assume that modern primitive societies have retained the characteristics of their prehistoric predecessors in the field of medicine (Ackerknecht 1982:10).

Although modern primitive societies are removed from ancient populations under study in terms of time and space, they are probably the most comparable equivalent. However, use of this type of data has received some criticism in the past. Scholars believed that information from modern primitive peoples was too different to use to explain archeological data. However, by the late 1960s, in Britain at least, more interest was shown in the use of ethnoarcheological evidence. The problem with studying archeology is the loss of human character in all the remains observed (Schwarz 1978:vii). It is by studying societies of similar characteristics in existence today that a more humanistic and relevant interpretation of past behavior can emerge.

The problems encountered in the use of ethnographical evidence are apparent. There is a bias of study of modern primitive societies toward hunter-gatherers (Kramer 1979:3) but this is perhaps a necessary and useful act before these societies are encompassed into a more settled way of life with modern ideas and technology. The periods of time being studied in this particular research deal with people whose economy is not hunter-gathering, so care in the comparison and interpretation therefrom of these diverse societies is realized.

Ethnographical studies to date have also concentrated on certain areas of the world, such as Africa, South America, and Alaska (e.g., Carroll 1972). Although these are areas where many societies remain uninfluenced by western ideas, such concentration leads to a bias in data availability. Problems also arise when workers try to use only one society to explain their archeological data. A wide variety of ethnographical evidence should be utilized at all times.

Past societies in Britain, as we have seen, are very distant from the surviving modern primitive societies. For example, British researchers could not justifiably use analogies between our contemporary industrialized society and the prehistoric and early historic past. But, analogs for past societies in Britain are provided for by historical documents which reduce the time and space elements. Even withstanding these limitations, use of this type of data should be considered beneficial rather than being rejected.

ARCHEOLOGICAL DATA

Three types of archeological data are relevant to the research: artifactual, structural, and environmental (Table 3). The information which can be derived from these data is extensive but can particularly reflect on relevant aspects of past societies in relation to the natural process of fracture healing, and the therapeutic management thereof. The environment in which individuals were living (both macro and microscopic), climate, hygiene, diet (including food available and food preparation), medicinal plants available, and clothing are all relevant to the healing process.

The availability of this data in Britain is variable. Archeological studies, until recently, have tended to concentrate on the artifactual and structural aspects of research activities, primarily because these classes of data were the most abundant. It is commonly accepted that pottery is "one of the most commonly analyzed and useful kinds of artifacts available to archeologists" (Sharer and Ashmore 1979:306). As methods of extraction of material remains, particularly environmental evidence, have improved over the past two decades, more relevant questions have been asked of the data. Diet, living conditions and hygiene are accepted as subjects worthy of study. Sir Mortimer Wheeler's words (1954) were quite applicable, "Too often we dig up mere things unrepentantly forgetful that our proper aim is to dig up people."

TABLE 3. Potential data sets for three types of archeological data

	Examples
ARTIFACTUAL	
pottery	vessels: diet, food and medicinal preparation
metal	surgical instruments: surgery
wood	vessels, utensils: diet, food preparation
stone	quernstones: food preparation, diet
leather	clothing: living conditions, climate
bone/ivory	combs: hygiene
wall plaster	illustrations of life
STRUCTURAL	
houses	environment, hygiene, living conditions
hospital	care of the sick
hearths, wells, cesspits	climate, hygiene, environment
latrines, sewers	hygiene, living conditions
bath-houses	hygiene
ENVIRONMENTAL	
pollen	all depicting environment, diet, climate living conditions, diet, hygiene
seeds	
molluscs	
animal bones	
soil	
parasites	
insects	
coprolites	

Work in environmental archeology has advanced in many areas of Britain but notably in York at the Environmental Archaeology Unit (O'Connor 1986). Meticulous extraction, identification and interpretation of environmental evidence such as seeds, insects and animal bones has helped to extend archeological interpretation beyond the pottery stage. Researchers no longer end their studies at "what kind of pottery did they have" but can deduce "how people lived." Environmental archeology "enables archeologists to move away from examining the sterile remnants of ancient lives and envisage the communities as they actually lived" (Shackley 1985:13). Studying one piece of evidence is no longer accepted as the sole basis for archeological research.

The study of archeological data in association with the paleopathological evidence is essential for an understanding of the treatment and healing of fractures. Environment, in all its facets, and hygiene will affect how well and quickly a fracture heals.

In Britain, in general terms, archeological data is in abundance but there is often a bias in favor of particular periods of time (often merely because of the abundance of evidence available and of archeologists to deal with it) or regions of Britain. There is especially a tendency in environmental studies to produce detailed syntheses on the environmental conditions of one structure (see Kenward et al. 1986; Grieg 1981). These studies are inevitable but useful. Disregarding financial constraints, however, these analyses are very labor-consuming. It will be some years before more regional studies of environmental archeology will be available for use in such archeological research as paleopathology. In the case of environmental archeology at York, the process of analyzing organic remains is ongoing in order to extend knowledge of the archaic environment of York.

The study of trauma and treatment in antiquity has by necessity generated many avenues of research to follow. One must consult many areas of evidence to gain an accurate picture of how well cranial and postcranial fractures were treated and how well they healed. This research encompasses the skeletal evidence of fractures and the therapeutic measures of reduction, splinting and trepanation. But it also covers many other subject areas: concepts of disease and treatment, anesthesia, diagnostic procedures, anatomical knowledge, dressings, surgical instruments and herbal remedies, blood letting and hemostasis, complications of fractures, hospitals and personnel. The Roman to late and post-Medieval periods were interesting eras and the wealth of evidence spanning 1600 years will provide abundant data for this research.

Observational methods

RECORDING OF LONG BONE AND SKULL FRACTURES: MACROSCOPIC

Consultation of modern clinical data on fractures was necessary to compile a recording form adequate to describe the nature of the fracture with reference to modern accounts of fractures. Recording forms were developed for both long bone and skull fractures.

Cemetery site, period of time (Roman, Anglo-Saxon and Medieval), location of bone, age and sex were recorded as basic data. The bone or anatomical part affected and side of body were noted so that quick reference could be made. Fracture position on long bones was recorded in terms of proximal, mid or distal third of the bone shaft. Fractures occurring proximally or distally to these three levels were described with reference to anatomical points on the bone (Warwick and Williams 1973). The level of fracture on the bone has important implications for particular neural and vascular complications; for example, healing of a fracture to the distal third of a tibia may be delayed due to a disruption in the blood supply to the distal fragment and a fracture to the midshaft of the humerus may lead to radial nerve palsy. The radial nerve is close to the bone at this point and is therefore very vulnerable (Klenerman 1966). The effect of continuous radial nerve palsy would be paralysis of the extensor muscles of the wrist, thumb and fingers causing wrist drop.

The type of fracture an individual sustains will give an indication of the type of force acting on the bone to produce the break. This feature can have implications for

(1) interpreting occupation (e.g., Merbs 1983), warfare (e.g., Manchester and Elmhirst 1980), or domestic accidents;

(2) determining how quickly the fracture healed. For example, oblique or spiral fractures are more stable than transverse fractures. In addition, some types of fractures are, in modern populations, correlated with particular types of accidents—a Colles fracture of the distal end of the radius, for example, which occurs when a person, particularly an elderly woman with osteoporosis, falls on an outstretched hand;

(3) identifying potential complications of injuries to the skull; for example, a blade injury and depressed fracture produce different types of brain injury. Different areas of the skull produce contrasting complications.

To record healing of the fracture, a general assessment was made of how well the bone had healed, taking into account many different features identifiable at the fracture site:

SHORTENING. By comparison with the opposite leg or arm, the degree of loss of length was assessed (Figure 2). This gave an indication of how well or how badly the fracture was reduced and/or splinted in the right position.

INFECTION. Evidence of an infective process was defined by new bone growth and/or pitting of the bone surface around the fractured site with or without an associated osteomyelitic lesion displayed as a sinus on the bone surface (Figure 3). Presence or absence of infection gives an indication of the

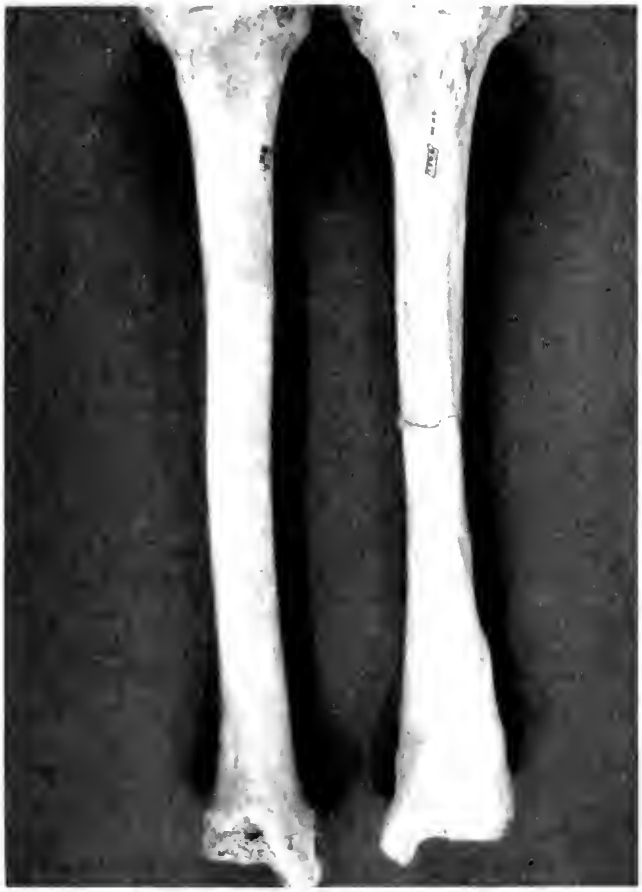


FIGURE 2. Oblique fracture to distal left tibia of an Anglo-Saxon individual (8th century A.D.) from Raunds, Northamptonshire. Healing has led to shortened affected limb.

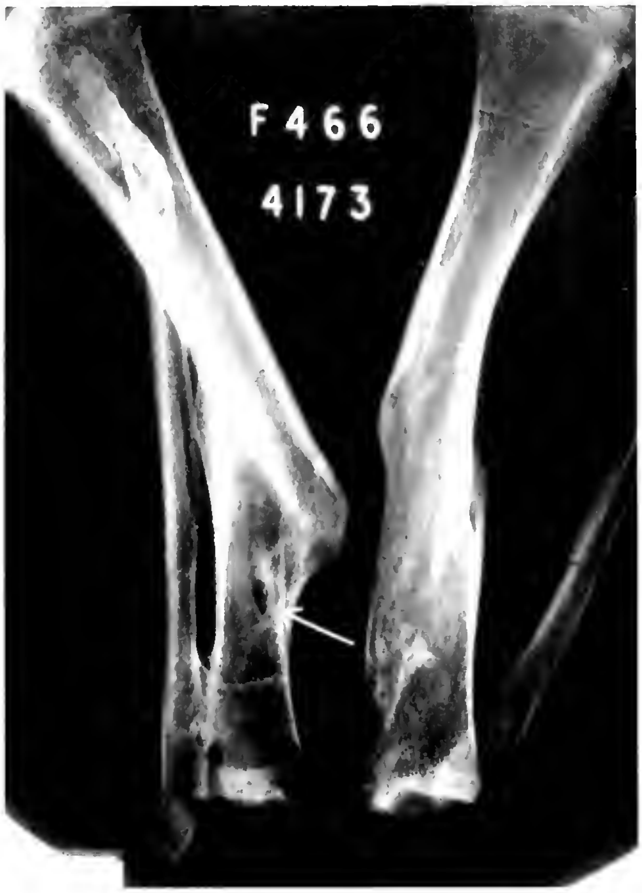


FIGURE 3. Radiograph of a Roman (4th century A.D.) individual from Baldock, Hertfordshire, showing bilateral tibial fractures and sinus in right tibia.

environment in which the person was living and the type of fracture (simple or compound).

DEFORMITY. Rotational or linear deformity was recorded as present or absent by comparing with a normal bone. More detailed analyses of deformity could be measured on the radiograph (see below). The presence of a deformity in the fracture once healed may suggest that the fracture was not treated by reduction and splinting. However, the additional complication of fractures of different parts of the body being harder to treat needs to be taken into account here.

OSTEOARTHROSIS. Degenerative change on joint surfaces of long bones sustaining fractures usually occurs in reaction to stresses placed on the joint caused by deformity of the bone on healing. Again, the presence or absence of this feature was recorded. In addition, the factor of age was also borne in mind.

ALIGNMENT. The alignment of the fractured bone, once healed, was recorded to indicate how efficiently the fracture was reduced and splinted. Additional factors are as outlined under deformity.

Fractures to the skull were recorded on a form modified from that used for long bone fractures. The bone affected and fracture position were noted both on the record sheet and in a diagram. Two types of head injury are commonly identified in human remains: a blunt head injury causing a depressed fracture with or without comminution, or a sharp injury caused by a blade or other sharp object (Figures 4,5).

Evidence of healing was noted if the wound appeared to have rounded and remodeled edges and the presence or absence of endocranial involvement was recorded. This latter feature would have had severe implications for brain integrity. Infection, in the form of periostitic pitting of the bone surface around the fracture site, was documented. The presence of an infection of a compound or open skull fracture

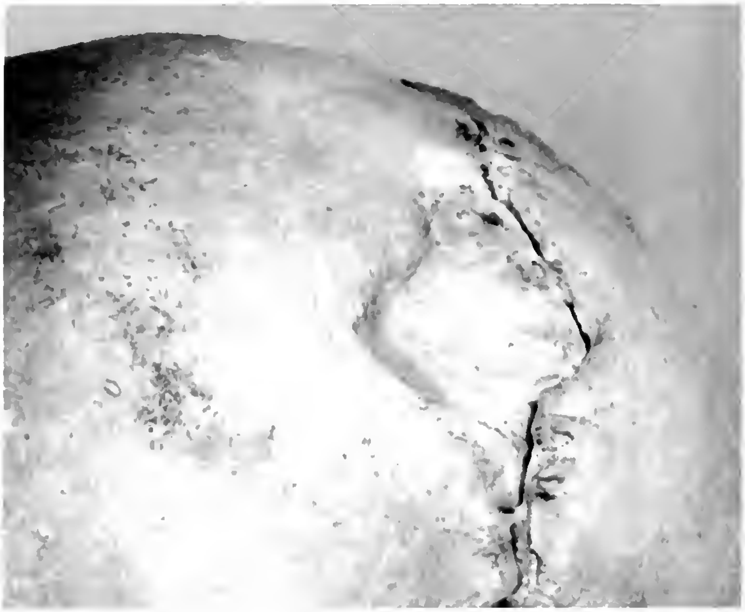


FIGURE 4. Depressed skull fracture to right parietal bone of a Roman individual from Hyde Street cemetery site, Winchester, Hampshire.

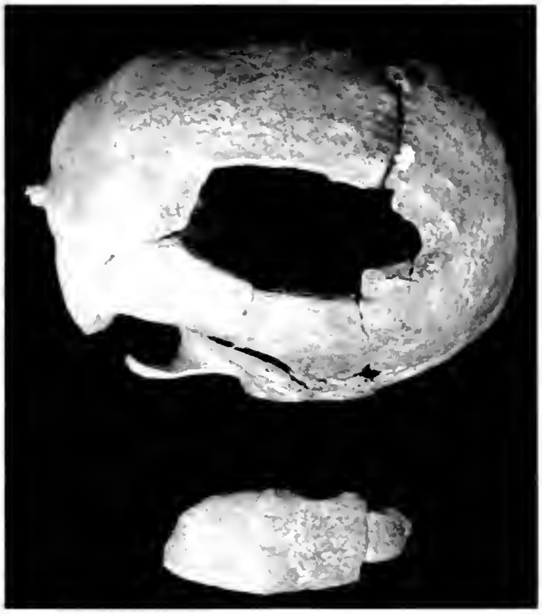


FIGURE 5. Unhealed blade injury to left parietal bone of an Anglo-Saxon individual from Pewsey, Wiltshire.

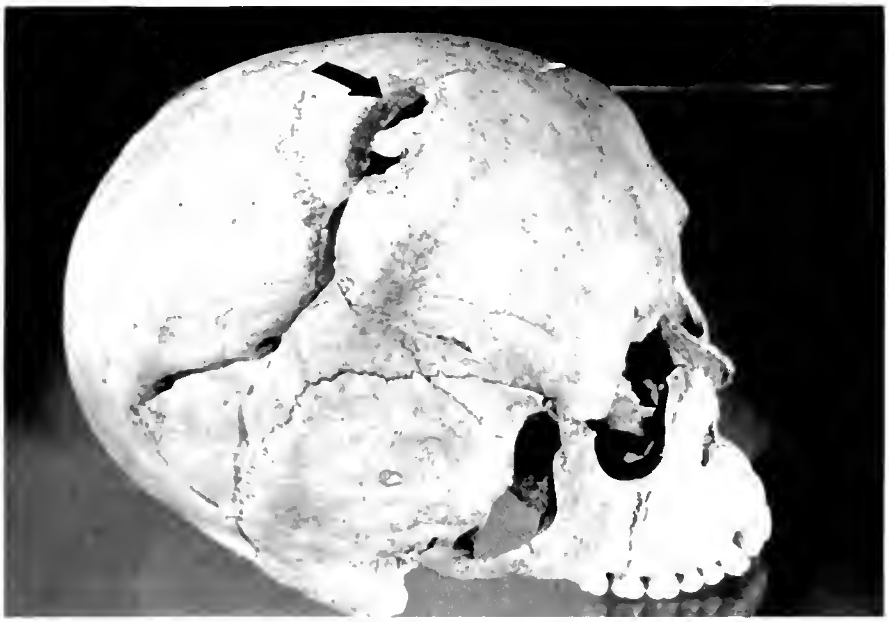


FIGURE 6. Healed trepanation associated with a blade injury to right side of skull of a Roman individual from Cirencester.

would present an opportunity for bacteria to enter the cranial cavity and cause endocranial infection, either abscess or meningitis.

Both long bone and skull fracture forms had a section for commenting on possible treatment of the fracture. Many recorded features of long bone fractures are related to treatment, as has been seen, but only by assessing the totality of these features could comments be made regarding treatment. Head injuries in the past were sometimes treated by trepanation or surgical removal of a piece of bone from the skull. In some cases there is evidence of a wound to the skull associated with a trepanation (Figure 6). Features of the trepanation were also recorded—operative site, type, shape and size (length, breadth, depth, internally and externally) of trepanation, presence of healing and any indication of infection present.

In all individuals, the presence or absence of cribra orbitalia, porotic hyperostosis, and dental enamel hypoplasia was recorded to indicate the health status of the individual (Goodman et al. 1984). These features were important in terms of healing of the fracture. Fractures might heal slowly or not at all in an individual who has a poor diet, living in conditions unfavorable for healing to take place. These stress indicators were also noted in the individuals in the cemetery as a whole to compare the general health status of the population with that of the affected individual.

RECORDING OF LONG BONE FRACTURES: RADIOGRAPHIC

By necessity, the evaluation of trauma in ancient populations is heavily reliant on the use of radiography; macroscopic evidence alone is insufficient to give the information paleopathologists should seek. Radiography is perhaps one of the few nondestructive informative tools the paleopathologist has. In Britain facilities for radiography are abundant but many workers in the field of human remains either do not have the resources to produce many x-rays or do not have access to a machine or friendly radiographer. In order to adequately record the fractures observed in this research it was necessary to ensure the availability of an x-ray machine and film.

Treatment of trauma in past populations was not aided by radiography, which we use today to, first, diagnose whether a fracture was present and, second, to assess the relationship of the fracture fragments within the limb affected pre-, during, and post-treatment. Today the availability of such modern technology allows the paleopathological researcher to x-ray fractures at the stage where the break has, in most circumstances, already healed.

Following examination and recording of the long bone fractures in the data set, each one was x-rayed in standard clinical views, anteroposteriorly and mediolaterally. These are the minimum views which should be taken of any pathological bone because one projection may be insufficient to assess the abnormality.

It was essential that these basic views were adhered to so that comparison with modern x-rays would be feasible; this was a later stage of the project. The radiographic work was done on a Siemens "Orbix" machine at Bradford Royal Infirmary using 3M XUD film contained in a cassette fitted with T2 screens.

Processing of the films was carried out using a 3M X P507 90 second processing machine. The radiographic film was donated by an x-ray film company and the work done out of normal clinical working hours. The film used for this research produces considerable image detail and was therefore ideal for the purposes of this study.

Several x-rays of the smaller long bones (forearm and fragments of other long bones) were produced on a "Faxitron" machine, a portable industrial unit within the Archaeological Sciences Department at the University of Bradford. Industrex C film and manual processing made this system unsuitable for the research in terms of time and machine capacity for larger long bones.

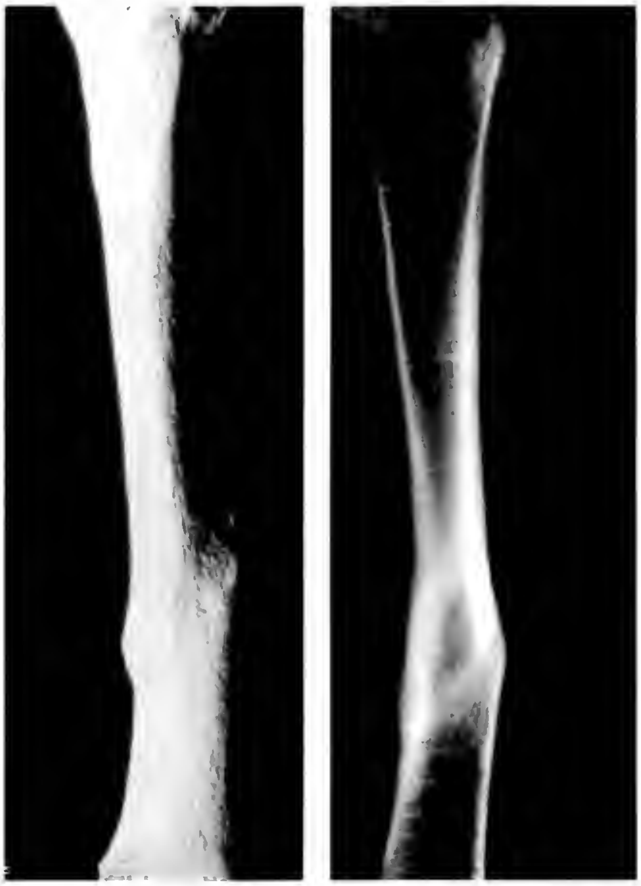


FIGURE 7. Distal shaft of tibia of a Roman individual from Baldock, Hertfordshire (4th century A.D.): an apparently oblique fracture *(left)* is, in fact, shown to be spiral in x-ray *(right)*.

The skull fractures were not x-rayed as it was considered that little further information could be gained by observing a film of the injury.

X-RAY ANALYSIS

Each radiograph was assessed and recorded on a standard record sheet designed after considerable consultation of current radiographic analytical literature, especially Rogers (1982). Features specific to the x-ray film were recorded and included the following:

TYPE OF FRACTURE. The type of fracture was not always clear from macroscopic observation, so an x-ray was the only sure way to distinguish the type of break and potential causation (Figure 7).

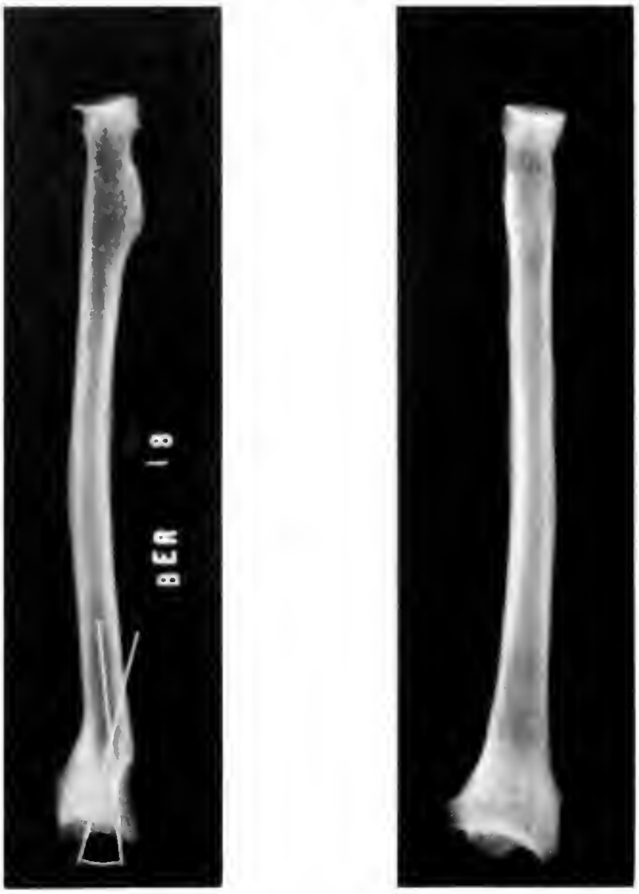


FIGURE 8. Illustration of the method of measuring angular deformity in a healed, fractured radius from a Medieval individual.

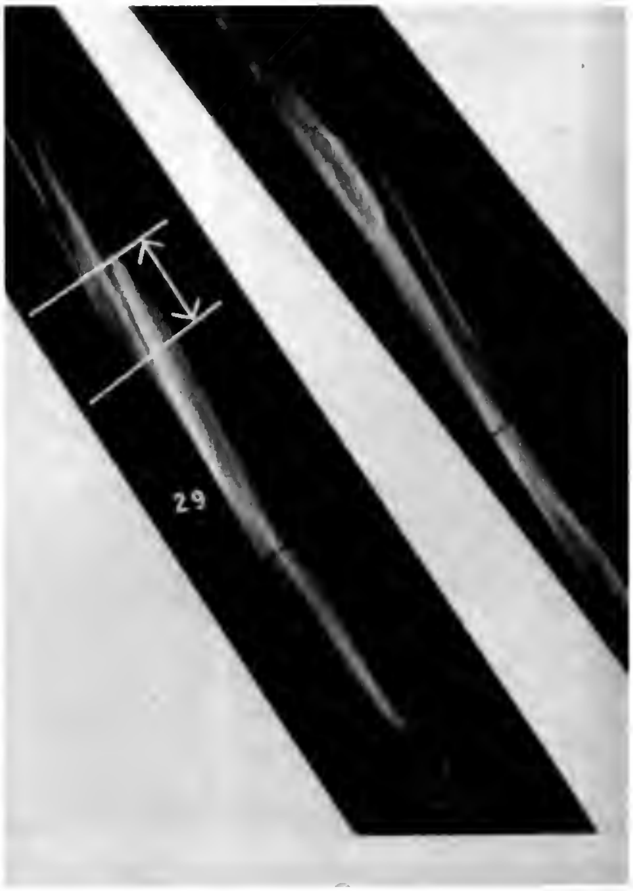


FIGURE 9. Measurement of degree of apposition of fractured fragments. A healed, fractured tibia in an individual from a Roman cemetery in Winchester, Hampshire.

FRACTURE LINE. The presence of a fracture line on the x-ray gave an indication of the stage of healing which the fracture, in relative terms, had reached. Early stages of healing will show a clear fracture line while a long-standing fracture would display a partially or fully obliterated line.

CALLUS. The development of callus or immature bone initially around the fracture site is evident, in the early stages of healing, on the x-ray as an area of bone with a "fluffy" outline with flecks of radio-dense material in it. This is evident within the first few weeks following the injury. As the callus becomes older and more mature, it becomes more uniformly radio-dense and opaque than the rest of the bone on the x-ray. As the fracture ages and the callus is calcified (from calcium salts from the bloodstream), the callus approaches the density of normal bone. The fracture line becomes obliterated and the trabecular pattern across the line is restored.

A large amount of callus can indicate many things, but particularly the absence of inadequate immobilization after the fracture allowing the fragments to move and precipitate new bone formation.

The rate of healing of a fracture is dependent on many factors, some of which have already been mentioned. The anatomical part of the body affected and the age of the individual are perhaps two of the most obvious. In most cases of fracture, however, the better the fracture has been treated the less the body has to work to repair and remodel the fractured area.

DISPLACEMENT. Linear displacement of the fracture fragments was recorded by measuring the angle of displacement with a ruler and protractor on the x-ray film (Figure 8). Attempting to measure this feature on the bone itself leads to errors. Rotational displacement of a fracture could only be

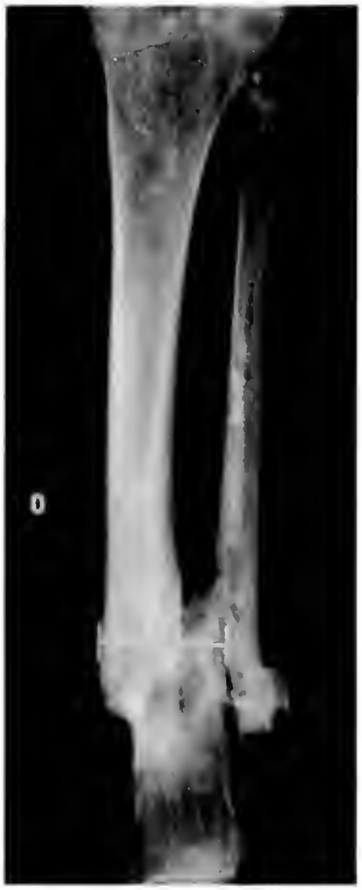


FIGURE 10 *(left)*. Measurement of amount of overlap of fracture fragments in a fibula fracture of a Medieval individual from Billingsgate cemetery, London.

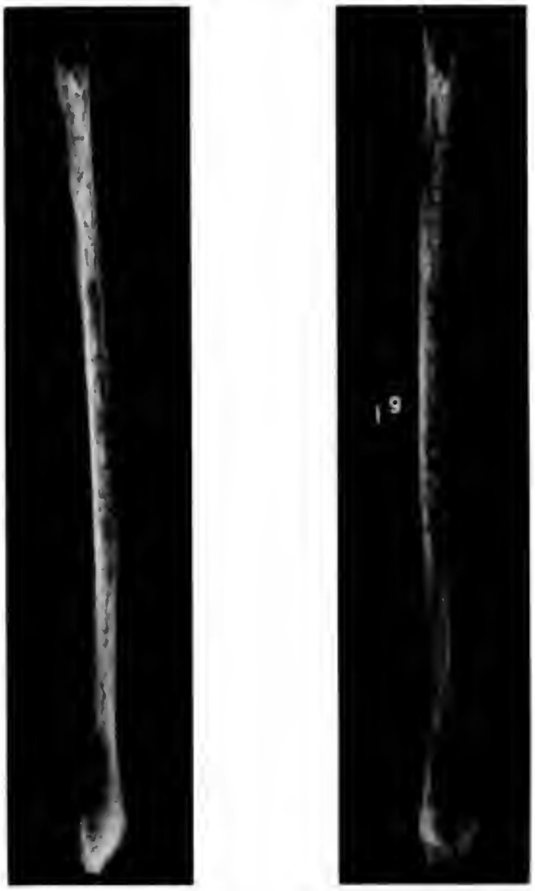


FIGURE 11 *(right)*. Soil in the medullary cavity of a fibula obscuring a fracture in proximal shaft.

measured roughly. The relationship of the ends of the fracture fragments to each other was also recorded (Figure 9). Muscular contraction at the time of injury can make reduction of fractures problematical, and this would have been particularly so in ancient populations.

OVERLAP. The amount of overlap of the fracture fragments and degree of apposition could also be measured on the x-ray by direct measurement (Figure 9). Overlap of fragments may mean (for example in a tibial fracture) a shortened leg for the individual and difficulties with mobility. One-third to one-half apposition of the broken fragments in a fracture is believed to give a good functional result in modern populations (Figure 10).

X-RAY ANALYSIS: PROBLEMS

Several problems were inherent in examining x-ray films, mostly generated by the fact that archeological populations have been buried in the ground, unlike their modern counterparts. Soil in the medullary cavity of long bones made detection of the original fracture line difficult (Figure 11) even though, macroscopically, there was evidence of a fracture. A fracture could also be misdiagnosed by the appearance of vascular channels in the bone shaft. Fractures of very long standing were often so well healed that the original type of fracture was not identified.

On the x-ray in the callus there were often flecks of radiodense soil particles mimicking calcified healed areas to the untrained eye. Alternatively, radiolucent areas around the fracture site could be mistaken for infective lesions, but the main difficulty was to determine whether the radiolucent area was ante or post mortem.

Pathological fractures were often difficult to identify, especially in the case of osteoporosis underlying the fracture. Osteoporosis is a very common condition today, especially in older women where the quality of the bone remains constant but the quantity decreases. This is particularly seen in ribs, vertebrae and the pelvis. The osteoblast and osteoclast balance is lost and the bone is liable to break under minimal force. However, diagenetic factors working on skeletons in the ground in cemetery sites may make the bone appear to be osteoporotic. The main problems are whether the individual had osteoporosis before the fracture, after the fracture, or whether it developed because of bed rest (disuse osteoporosis) or as a postdepositional syndrome.

The final problem with x-ray analysis is the age of the fracture. The complete healing process, in normal circumstances, takes about one year; after that time there is little change histologically or radiographically in the fracture appearance. It is, therefore, difficult to ascertain at what age the fracture occurred in the individual unless the incident occurred just prior to death. At present, in paleopathology, ages of individuals with fractures are meaningless in terms of the timing of the fracture.

We hope that work to be undertaken in the near future will give more accurate indicators of the age of fractures in ancient populations. Modern, documented, clinical x-rays from the Institute of Orthopaedics in London will be studied. The x-rays will be comparable in terms of the following features: nonoperatively treated fractures, i.e., those treated with basic reduction and splinting; simple, not compound, fractures; all adult individuals; fractures resulting from accidents not related to modern technology.

These x-rays will be compared with the archeological films, and clinical records will be consulted to ascertain the age of the fracture. The same recording form will be used to record the modern x-rays. The availability of clinical records now provides an additional potential to assess the causes of particular fractures in the ancient data, to know exactly how the fractures were treated and whether infection was present, and to observe the fracture on x-ray pre-, during and post-treatment. Modern clinical x-rays give an added time dimension.

Summary discussion and concluding remarks

This paper has outlined the author's methodological approach to trauma and treatment in the British Historic Period. It is fortunate for paleopathology that there has recently been a strong move toward this type of multidisciplinary approach in the interpretation of archeological sites.

Skeletal remains from cemeteries do not represent individuals who lived cocooned in isolation from their environment. These people were constantly interacting with their environment. As Calvin Wells said in 1964 (17):

> The pattern of disease or injury that affects any group of people is never a matter of chance. It is invariably the expression of stresses and strains to which they are exposed, a response to everything in their environment and behavior. It reflects their genetic inheritance (which is their internal environment) the climate in which they lived, the soil that gave them sustenance and the animals or plants that shared their homelands. It is influenced by their daily occupations, their habits of diet, their choice of dwelling and clothes, their social structure, even their folklore and mythology.

These words hold true over 20 years later. Archeological data, in whatever form, is fragmentary and reflects a sample of the original deposit. The nature of this deposit and its later interpretation, whether of human skeletal remains or pottery, is influenced first by the individuals who ensured its burial or created an environment in which it was discarded, second by burial conditions in the ground, third by the people who excavated and processed the material, and last by the person who examined the remains. The complete picture of particular aspects of societies in the past is gradually lost through all these processes. As time proceeds from true life to a distant past, interpretation becomes more difficult, especially when only one type of evidence is being assessed. All types of archeological data are fragmentary but the maximum amount of information must be generated. Perhaps this is why researchers in human skeletal remains are beginning to realize the vast potential of using other sets of data to help them interpret data generated from human skeletal remains. This approach, however, is still in its infancy.

There will always be a special place in the literature for unusual and isolated pathological conditions, but in the future there will be an increasing demand for more wider ranging analyses of human skeletal data. It will no longer be deemed acceptable to consider skeletons as a single entity, unresponsive to their surroundings. The use of modern clinical data in paleopathology will further help to broaden our horizons and help interpretation go further than mere diagnoses of cases. More epidemiological considerations in the future will enable paleopathologists to look upon their data more critically. There is much to be learned by all parties whether they be clinicians, paleopathologists, art historians, or ethnographers. There is also a move toward multidisciplinary conferences (e.g., Dieppe and Rogers 1986) where exchange of ideas is encouraged.

In this particular type of study it has already been noted that the use of multiple sets of evidence creates problems which can, with care, be solved. Reliance on individuals who are experts in their own fields to produce data relevant to the questions being posed is essential. However, these data can then be assessed by the paleopathologist and considered relevant or rejected. The limitations of each type of evidence being used have already been outlined and due consideration will be taken of these limitations in the final interpretation.

The end results of this study will be (1) to assess the healing of each fracture observed, taking into consideration all the factors relevant to healing, and extensively consulting modern clinical data and x-rays; and (2) to consider the evidence for diagnosis and treatment in all periods examined, including all subject areas already mentioned relevant to diagnosis and treatment, and to work the two sets of data together to interpret how well fractures in the British Historic Period were being treated, what techniques, equipment and knowledge were available, in what kind of environments did fractures have to heal, and whether the type of diet individuals were eating was good enough for fracture healing to occur.

There will be some areas of evidence grossly lacking and some will be plentiful, but it is hoped that a representative picture of past trauma and treatment will be produced in the not too distant future.

Acknowledgements

Due thanks go to Keith Manchester who made helpful comments throughout the paper, the many museums and archeologial units who have allowed me access to human skeletal material, Tony Margel, Senior Radiographer at the Bradford Royal Infirmary, for producing most of the x-rays (also to a generous x-ray film company) and to Jean Brown, formerly of the Photography Department of the University of Bradford, for producing the photographs and diagrams. There are also many, many more people who have been helpful throughout this research and thanks will be given to them in the final thesis.

Literature cited

Ackernecht, E.H. 1982. *Short History of Medicine*. Baltimore: Johns Hopkins University Press.

Arnold, C.J. 1986. Archeology and History: The Shades of Confrontation. In J. Bintliff and C. Gaffney, eds., Archeology at the Interface: Studies in Archeology's Relationships with History, Geography, Biology and Physical Science. *British Archaeological Reports, International Series*, 300:32–39.

Bennike, P. 1985. *Paleopathology of Danish Skeletons*. Copenhagen: Akademisk Forlag.

Bisel, S.C., and J.L. Angel. 1985. Health and Nutrition in Myceneacan Greece. A Study in Human Skeletal Remains. In N.C. Wilkie and W.D.E. Coulson, eds., Contributions to Aegean Archeology. *Publications in Ancient Studies*, 1:197–209. Minneapolis: Center for Ancient Studies, University of Minnesota.

Carroll, G.A. 1972. Traditional Medical Cures along the Yukon. *Alaska Medicine*, 14:50–53.

Cohen, M.N., and G.J. Armelagos. 1984. *Paleopathology at the Origins of Agriculture*. New York: Academic Press.

Courville, C.B. 1965. War Wounds of the Cranium in the Middle Ages. *Bulletin of the Los Angeles Neurological Society*, 30:27–33.

Cramp, R. 1983. The Archeologist's View—General. In G.D. Hart, ed., *Disease in Ancient Man*. Toronto: Clarke Irwin.

Daniels, S.G.H. 1978. Research Design and the Structure of Archeology. *World Archeology*, 10:28.

Dawson, W.R. 1934. *A Leechbook or Collection of Medical Recipes of the 15th Century*. London: Macmillan and Company.

Dieppe, P., and J. Rogers. 1986. Art, History and the Antiquity of the Rheumatic Diseases. *Clinical Rheumatology*, 5:179–180.

Einarsson, F. 1958. Fracture of the Upper End of the Humerus. Discussion Based on the Follow Up of 302 Cases. *Acta Orthopaedica Scandinavica*, supplement, 32.

Fife, D., and J.I. Barancik. 1985. Northeastern Ohio Trauma Study III. Incidence of Fractures. *Annals of Emergency Medicine*, 14:244–248.

Gilbert, R.I., and J.H. Mielke, eds. 1985. *The Analysis of Prehistoric Diets*. London: Academic Press.

Goodman, A., D. Martin, and G.J. Armelagos. 1984. Indicators of Stress from Bone and Teeth. In M.N. Cohen and G.J. Armelagos, eds., *Paleopathology at the Origins of Agriculture*, 13–49. New York: Academic Press.

Grattan, J.H., and C. Singer. 1952. *Anglo-Saxon Magic and Medicine*. Oxford: Oxford University Press.

Grieg, J. 1981. Investigation of a Medieval Barrel Latrine from Worcester. *Journal of Archeological Sciences*, 8:265–282.

Hart, G., and D. Ubelaker. 1987. Preservation Committee Report. *Paleopathology Association Newsletter*, 58:3–4.

Herrlinger, R. 1970. *History of Medical Illustration from Antiquity to 1600 A.D.* London: Pitman Medical and Scientific Publishing Company.

Kenward, H., A.R. Hall, and A.K.G. Jones. 1986. Environmental Evidence from a Roman Well and Anglian Pits in the Legionary Fortress. *Archeology of York. The Past Environment of York*, 14(5). London: Council for British Archeology.

Klenerman, L. 1966. Fractures of the Shaft of the Humerus. *Journal of Bone and Joint Surgery*, 48B:105–111.

Kramer, C., ed. 1979. *Ethnoarcheology. Implications of Ethnography for Archeology*. New York: Columbia University Press.

Lovejoy, C.O., and K.G. Heiple. 1981. Analysis of Fractures in Skeletal Populations with an Example from the Libben Site, Ottawa County, Ohio. *American Journal of Physical Anthropology*, 55:529–541.

Manchester, K. 1978. Paleopathology of a Royalist Garrison. *Ossa*, 5:25–33.

Manchester, K., and O.E.C. Elmhirst. 1980. Forensic Aspects of an AngloSaxon Injury. *Ossa*, 7:179–188.

Marwick, A. 1970. *Common Pitfalls in Historical Writing*. New York: Open University Press.

Merbs, C. 1983. *Patterns of Activity Induced Pathology in an Inuit Population*. Ottawa: National Museums of Canada.

O'Connor, T. 1986. What the Vikings Left Behind. *New Scientist*, 1533:42–47.

Ortner, D.J., and W.G.J. Putschar. 1981. Identification of Pathological Conditions in Human Skeletal Remains. *Smithsonian Contributions to Anthropology*, 28. Washington, D.C.: Smithsonian Institution Press.

Osborne, D. 1969. The Archeological Determination of Diet and Natural Resources and the Case for Cooperation. *American Journal of Physical Anthropology*, 30:439–442.

Reece, R. 1982. Bones, Bodies and Disease. *Oxford Journal of Archaeology*, 1:347–358.

Reece, R., ed. 1977. Burial in the Roman World. *Council for British Archaeology Research Report*, 22. London: Council for British Archaeology.

Roberts, C.A. 1987. Case Reports in Paleopathology Number 9. Possible Case of Scurvy. *Paleopathology Newsletter*, 57:14–15.

Rogers, L.R. 1982. *Radiology of Skeletal Trauma*. 2 volumes. London: Churchill Livingstone.

Rohde, E.S. 1922. *Old English Herbals*. London: Longmans, Green and Company.

Schwartz, D. 1978. Preface. In R.A. Gould, ed., *Explorations in Ethnoarcheology*, iii–viii. Albuquerque: University of New Mexico Press.

Shackley, M. 1985. *Using Environmental Archeology.* London: Batsford.

Sharer, R.J., and W. Ashmore. 1979. *Fundamentals of Archeology.* London: Benjamin/Cummings Publishing Company.

Sharrard, W.J.W. 1979. *Pediatric Orthopedics and Fractures.* 2 volumes. London: Blackwell Scientific Publications.

Warwick, R., and P.L. Williams 1973. *Gray's Anatomy.* 35th edition. London: Longman.

Watson-Jones, R. 1976. *Fractures and Joint Injuries.* 2 volumes. London: Churchill Livingstone.

Watson-Jones, R., and W.D. Coltart. 1982. The Classic: Slow Union of Fractures with a Study of 804 Fractures. *Clinical Orthopaedics,* 168:2–16.

Webb, S. 1987. Reburying Australian Skeletons. *Antiquity,* 61: 292–296.

Wells, C. 1964. *Bones, Bodies and Disease.* London: Thames and Hudson.

______. 1982. The Human Burials. In A. McWhirr, L. Viner, and C. Wells, *Romano-British Cemeteries at Cirencester,* 135–202. Cirencester, U.K.: Cirencester Excavations Committee.

Wheeler, M. 1954. *Archaeology from the Earth.* London: Pelican Books.

Withers, R.J.W. 1960. On Bone Setting. *Ulster Medical Journal,* 29:1–13.

Zivanovic, S. 1984. The Changing Pattern of Injuries and Fractures of Bones in Medieval Serbian Population. In *Proceedings of the 5th European Paleopathology Association Meeting, Siena, Italy.* Siena, Italy: University of Siena.

SUMMARY OF AUDIENCE DISCUSSION: Dr. Pahl reported that his dissections of Egyptian bodies suggested that the presence of a stick apparently splinting a fractured bone may in fact have been a splint on a long bone fractured post mortem, perhaps during the mummification process. He bases this on the common absence of blood or reactive bone changes as well as bandages. Consequently he warned that the mere presence of a splint adjacent to a fractured long bone in an Egyptian body is not invariably evidence of antemortem treatment efforts. The presence of bony reaction in some splinted bodies, however, makes it clear that Egyptians did indeed make some therapeutic efforts in cases of long bone fracture.

Paleoepidemiology of trauma in a prehistoric central California population

Robert D. Jurmain

The challenges faced by and the personal catastrophes that beset prehistoric peoples sometimes fortuitously leave traces in their skeletons. One of the better perspectives from which to obtain data concerning particularly challenging events in the archeological past is the analysis of traumatic lesions.

Several researchers have noted previously that evidence of fractures, projectile wounds, and dislocations can provide information concerning the incidence of accidents or interpersonal violence in prehistoric populations (Elliot-Smith and Wood-Jones 1910; Angel 1974; Edynak 1976; Lovejoy and Heiple 1981).

A large and very well preserved skeletal collection from the prehistoric central Californian site of Ala-329 offers further illumination concerning traumatic episodes in the past. Differential diagnosis and analysis of traumatic lesions in this skeletal population provide examples of severe accidents as well as many cases of interpersonal violence. In addition, comparison of reactive changes about the hip joint seen in this group help further clarify the distinction between traumatic and congenital hip dislocation.

Materials and methods

Ala-329 is a large shell mound site located on the eastern shore of San Francisco Bay, approximately 20 miles north of San Jose. While now located approximately 2 ½ miles inland, the site probably once stood adjacent to the bay shore surrounded by saltwater marshes. Newly determined radiocarbon dates chronologically place the site between at least A.D. 500 and European contact (approx. A.D. 1700).

Ala-329 is a large site with dimensions extending 133 × 90 × 4 m high, and no doubt contains hundreds of burials. Excavations carried out by Stanford University and San Jose State University mostly during the 1960s exposed 20–25% of the mound and still removed 440 burials. The sample available for analysis in this study includes the 420 grave lots (representing a minimum of 440 individuals) excavated between 1959 and 1968 by San Jose State University and Stanford University field classes. Of these, the most relevant group for the present study is the 248 aged and sexed adults—138 males and 110 females. Overall, the condition of the burials is good to excellent and, as such, this collection represents one of the larger and better preserved osteological samples in the western United States.

Moreover, most graves were undisturbed, and careful excavation retrieved many elements intact. Indeed, more than one-third of all burials could be described as "complete" (i.e., containing most major elements intact). Soil conditions at this site, apparently physically and chemically buffered by the large quantity of shell, afforded excellent conditions for preservation (see Figure 1).

Pathological lesions were diagnosed by gross macroscopic examination and supported by radiographic analysis including standard x-ray as well as computed tomography (CT) scans. In most cases the diagnoses from gross specimens and radiographs were corroborated through examination by an orthopedic surgeon. Most fractures were ascertained by the presence of angular deformity often accompanied by shortening of the affected element. In other cases, in the absence of gross morphological change, a diagnosis of healed fracture could not be supported unequivocally. Indeed, detailed examination of more than 100 radiographs of long bones by the author as well as an orthopedic surgeon could not find one additional healed fracture. Long-standing, very well healed fractures with no deformity or shortening are thought by many researchers to be virtually impossible to detect. Some reports (notably, Lovejoy and Heiple 1981) have relied upon subtle radiographic criteria to support diagnoses of most traumas. While such evidence may, in fact, be applicable to some specific cases, the ultimate effect will be to raise the apparent fracture rate in those samples to which such criteria have been applied, as compared to those groups (e.g., Ala–329) where such diagnoses are not supported.

Results and discussion

After dental disease and degenerative involvement (Jurmain 1983), trauma is the most common type of pathological lesion seen in this population. In many circumstances it is not possible to differentiate trauma from degenerative joint dis-

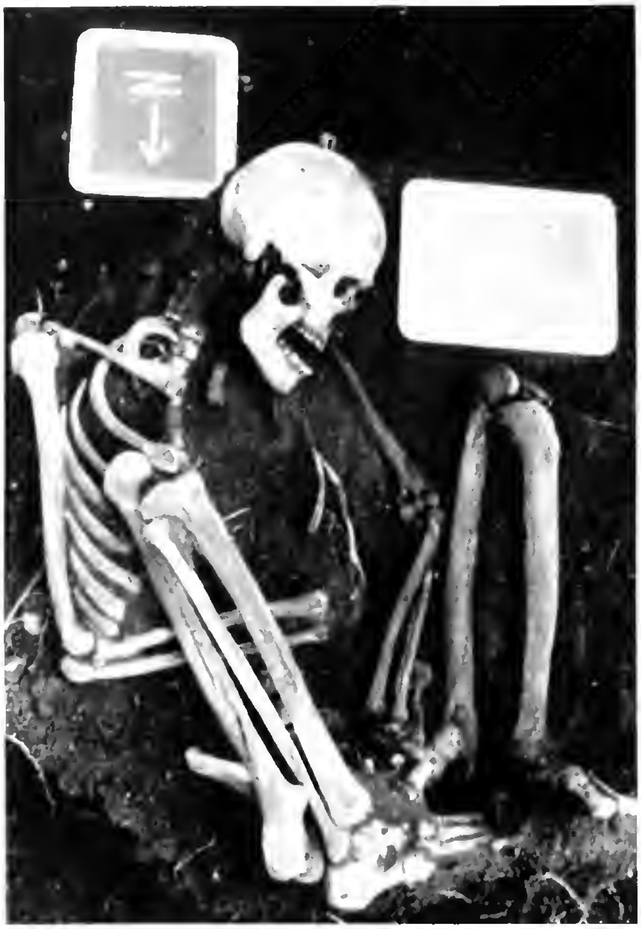


FIGURE 1. Intact human burial at Ala-329.

TABLE 1. Number of intact long bones by side

Clavicle	Left	143	
	Right	148	
			291
Humerus	Left	154	
	Right	146	
			300
Radius	Left	152	
	Right	149	
			301
Ulna	Left	147	
	Right	143	
			290
Femur	Left	163	
	Right	150	
			313
Tibia	Left	162	
	Right	153	
			315
Fibula	Left	121	
	Right	116	
			237
Grand Total:			2047

ease, especially in the hands and feet. And, as the hands and feet show the highest incidence of DJD, it can be reasoned that many of these degenerative changes result from recurrent trauma. However, since precise differential diagnosis of remodeled lesions of hand and foot bones is not feasible, this study will focus upon those unambiguous changes found in long bones.

For all the long bones (including the clavicle, humerus, radius, ulna, femur, tibia, and fibula) a total of 36 fractures are seen in 2047 intact long bones (Table 1), yielding a combined frequency of 1.8%. By far the most frequently fractured elements are in the forearm with 13 fractures of radii and 15 of ulnae. Indeed, many of these fractures are probably "parry" fractures and a predilection for left side involvement is manifested (62% of radial and 60% of ulnar fractures are of the left side). Further, five of these fractures did not fully unite. Although they are seen clinically with some frequency, ununited fractures are not that commonly observed in archeological materials. Stewart (1974) reports less than 10 cases from a total sample of several thousand individuals. From the large Libben collection (> 1300 individuals) only one further case of nonunion has been observed.

Thus, five additional involved elements in a single population of 440 individuals deserves our consideration. The first case, a male 30–35 years old, has ununited fractures of both left forearm bones—although at different locations. The ulna is fractured in the distal shaft, with the marrow cavity sealed, but the most distal piece is missing. Comparison with the right ulna indicates the injury occurred approximately 50 mm above the distal end. The left radius is fractured farther up, 123 mm above the distal end, and is also ununited. Again, the marrow cavity is completely sealed and a large reactive area is seen where fibrous union[1] would have occurred. The proximal piece, unfortunately is missing. Interestingly, in Stewart's review, only one other case of such double nonunion is mentioned.

---

[1] Some authors (e.g., Ortner and Putschar 1981) prefer the term "pseudarthrosis" for cases of nonunion where no new joint space is formed. However, other researchers (Steinbock, pers. comm.) believe "fibrous union" is a more accurate term; here, the latter terminology will be used.

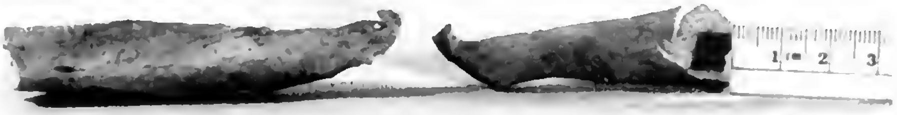


FIGURE 2. Ununited fracture of right radius. Male, 35–50 years.

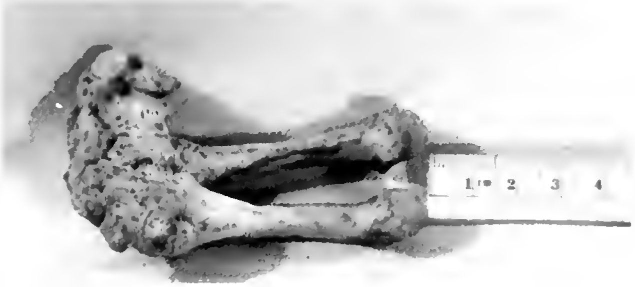


FIGURE 3. Fused right hand elements; all carpals and 2d and 3d metacarpals. Female, 39–44 years.

The second case, also a male (35–50 years of age), displays an ununited fracture of the distal right radius. Most of the radius is missing, but the distal piece that is present has a completely sealed marrow cavity. In addition, another small piece, representing the middle third of the diaphysis, also has a sealed marrow cavity (Figure 2). The right ulna is also fractured at approximately the same location, but shows good healing with a slight, angular deformity.

The third case of nonunion is a probable female, aged 21–30, with an ununited fracture of the left ulna 111 mm below the proximal end. The marrow cavity is again sealed and there is evidence of fibrous union with extreme hypertrophic reaction particularly in the area of attachment of the pronator quadratus. The distal piece, however, is missing. The radius is uninvolved.

The final case of nonunion is seen in the right ulna of a male, aged at more than 40 years at death. The lesion occurs 73.9 mm below the proximal end, and both ends are sealed with fibrous union. However, the bone still appears highly reactive (vascularized), and there is secondary degenerative disease of the wrist.

As seen, all cases of nonunion are of the forearm, as is consistently the pattern in other reports of this type of lesion. All the elements described by Stewart are also in the forearm, although he does mention cases involving the clavicle and femoral neck. Likewise, the only involved element at Libben is also a forearm bone (an ulna). However, it must also be noted that, as a result of poor recognition and diagnosis, this type of lesion may be considerably underrepresented in the paleopathological literature. Indeed, in discussion at the symposium and subsequent communication, several other cases of ununited fractures were noted: four from the Windover site in Florida (Dickel, pers. comm.) and two cases from archaic populations of the Great Lakes region of North America (Pfeiffer 1985, pers. comm.).

Other than the forearm, fractures in general are very rare in this population. Only eight other definite fractures of long bones are seen (two clavicles, one humerus, and five tibias). It must be emphasized that, as noted above, even with radiographic analysis, diagnosis of well-healed fractures is often impossible.

Fracture rates are difficult to compare between populations, as often the frequencies are not computed by individual elements, and even where they are, the degree of completeness of the sample is not taken into account. Among the better paleoepidemiological approaches to the study of fracture incidence is Lovejoy and Heiple's work with the Libben population. In order to control for effect of preservation, only intact long bones were included and carefully tabulated. A similar methodology is used here. The overall fracture incidence at Libben was 72/2383 (3.0%) compared to 36/2047 (1.8%), that is, only about half as high in this study. In addition to the clear quantitative difference in frequency, the pattern of involvement also varies dramatically. At Libben, the most frequently fractured element was the clavicle, involved in 15/260 cases (5.8%). At Ala-329, however, only two definite clavicular fractures are found in 291 intact elements (less than 1%). Certainly, in modern groups, most clavicular fractures result from severe falls (or auto accidents). The frequency of bad falls at Ala-329 thus appears to have been lower than at Libben. Indeed, Lovejoy and Heiple (1981) state that from modern U.S. data the clavicle is the most fractured long bone.

A particularly dramatic case at Ala-329 that did result from a severe fall is a 39–44-year-old female who had an apparent fracture to the distal right ulna (or Allen's fracture) that also severely traumatized the hand. Indeed, all the carpals plus the 2d and 3d metacarpals are fused into a solid block (Figure 3). In addition, the left arm is also broken (distal left radius), all perhaps as a result of one very serious fall.

Therefore, it would seem a different fracture pattern is indicated at Ala-329 compared to that at Libben. At Ala-329, severe falls were not the single primary cause of fracture. The relative infrequency of Colles' and Allen's fractures, seen in only 11 cases (5 Colles', 6 Allen's), further suggests this. Another possible explanation for the lower frequency of fractures seen in the Californian population could be demographic. Lovejoy and Heiple (1981) make the explicit point that fracture risk is directly linked to longevity. And, indeed, older individuals in both samples have more healed fractures.

TABLE 2. Fracture incidence by element

	Ala-329 N (%)	*Libben* N (%)	*Danish* N (%)	*Great Lakes* N (%)
Clavicle	2/291 (0.7)	15/260 (5.7)	5/386 (1.3)	2/66 (3.0)
Humerus	1/300 (0.3)	3/450 (0.7)	1/703 (0.1)	3/140 (2.1)
Radius	13/301 (4.3)	20/369 (5.4)	9/608 (1.5)	4/103 (3.9)
Ulna	15/290 (5.2)	11/351 (3.1)	13/607 (2.1)	3/124 (2.4)
Femur	0/313 (0.0)	9/347 (2.6)	0/998 (0.0)	1/112 (0.9)
Tibia	5/315 (1.6)	5/349 (1.4)	6/852 (0.7)	2/82 (2.4)
Fibula	0/237 (0.0)	9/257 (3.5)	2/364 (0.6)	1/? -

SOURCES: Libben, Lovejoy and Heiple 1981; Danish, Bennike 1985; Great Lakes, Pfeiffer 1985 (pers. comm.).

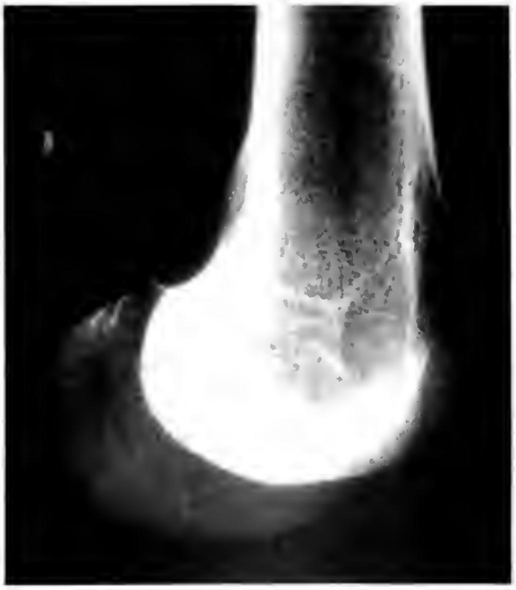


FIGURE 4. Radiograph, distal left femur showing healed lesion in center, superior to medial condyle. Male, 20–30 years.

The average age for individuals with ulnar fractures is 41 years at Libben and 39 years at Ala-329. For radial involvement the average age is 42 years at Libben and 41 at Ala-329.

It could be asked, then, if the Libben sample represents an older population. Here also, no clear differences appear, as the demographic profiles are generally quite similar. At Libben 26% of adults lived past age 40, while at Ala-329 28% of adults survived into the fifth decade.

It can be concluded, then, that age differences in these two populations do not adequately account for the differences in fracture incidence. Further systematic and thus comparative data on fracture frequency are available from a few other studies. In particular, Bennike (1985) has carefully computed incidence for a Danish skeletal series (Neolithic-Medieval) by element, and Pfeiffer (pers. comm.) has recently done likewise on archaic North American skeletal samples from the Great Lakes region of the U.S. and Canada. The results of these two studies as well as from the Libben series are compared with fracture incidence at Ala-329 in Table 2.

The frequency of forearm midshaft injuries suggests interpersonal violence (i.e., parry fractures), which is further indicated by the predilection mentioned earlier for left side involvement. Arguing against intergroup fighting is the similar incidence of female involvement in the forearm as that seen among males (13 male, 14 female). This observation does not of course preclude violence directed at women within the group.

However, there is more direct evidence of interpersonal violence in this group: nine individuals have embedded projectile points, and a tenth has evidence of a healed wound also probably from a projectile.

The first case is the healed wound, seen in the distal left femur of an adult male, 20–30 years of age. The lesion is an ovoid defect superior to the medial femoral condyle and showing hypertrophic formation both ventrally (entrance) and dorsally (exit), indicating the wound completely pierced the bone. On x-ray, the lesion appears quite "nonphysiological" in origin, given the nonregular shape of the canal (Figure 4). Obviously, considerable healing occurred following the injury. Interestingly, at the time of excavation, spent projectiles were found in the mouth of this individual.

The second case is a young adult female aged 19–21 years with a large obsidian projectile point embedded in the ventral body of the 5th lumbar vertebra (Figure 5). X-ray and CT scan analysis revealed no remodeling (healing) around the wound (Figure 6). Indeed, it would not be expected that this victim survived the injury, as soft tissue trauma due to hemorrhaging and intra-abdominal infection would have been unavoidable.

The next two cases are also vertebral wounds, but both of these are from the back. A teenager (14–18 years old) of indeterminate sex also has an obsidian point in the 5th lumbar vertebra embedded in the right dorsoinferior portion of its body. Under magnification a "nick" is also apparent along the lateral edge of the right pars interarticularis. Potential involvement of the right first sacral nerve root is thus suggested. No evidence of healing is apparent. The other individual "shot in the back" is a 25–35-year-old male with a small obsidian fragment embedded in the 1st lumbar vertebra dorsally on the left side of the neural arch lateral to the left

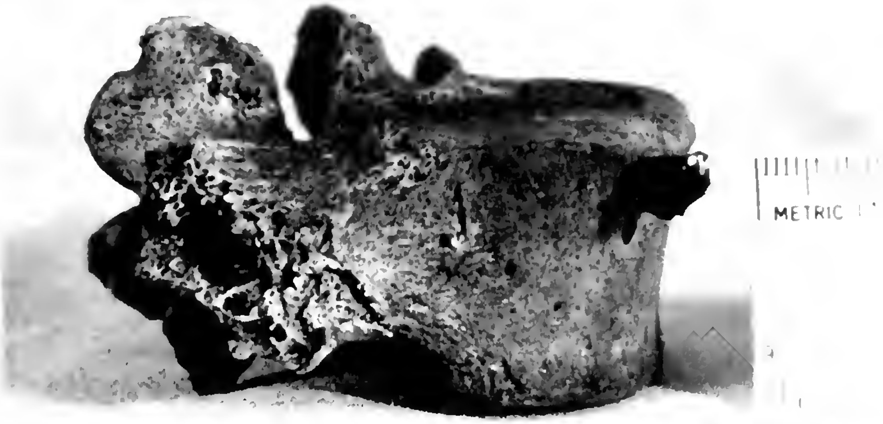


FIGURE 5. Fifth lumbar vertebra with projectile point embedded in anterior centrum. Female, 19–21 years.

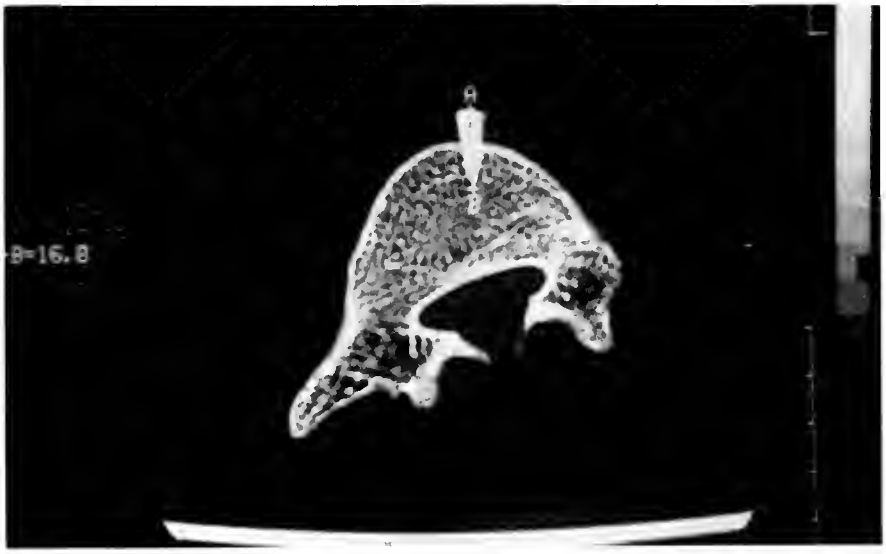


FIGURE 6. CT scan of same element as Figure 5. Distance A–B (16.8mm) is length of obsidian point.

superior articular facet. The remainder of the projectile point was also found with the burial. This individual apparently survived his wound as indicated by the resorption of bone about the embedded tip.

Three other cases also involve the vertebral column, one in a probable male, 15–17 years of age, with a small point in the dorsum of the 6th thoracic vertebra (T6) just below the left transverse process. No sign of healing is evident. In another case, a male 35–44 years of age at death, a large obsidian point was found in the right centrum of T12 just superior to the rib facet; the angle of trajectory indicates the projectile entered through the front, no doubt causing massive injury. Indeed, the projectile is deeply embedded, and there is no sign of healing. Moreover, there is another projectile wound in the distal end of the left radius, also showing no sign of healing (and thus probably resulting from the same obviously fatal incident). The last case of a vertebral projectile lesion is a young male, aged 20–22 at death. The broken end of an obsidian point is lodged in the right transverse process of his second lumbar vertebra. Since the point is broken, the angle of trajectory is difficult to ascertain; however, it appears he was shot from the front or to the side from the front. Additionally, the angle of trajectory appears to have come from below, unless of course the victim was shot while on the ground. There is no sign of healing about the wound.

Another interesting case of a projectile wound presents a somewhat different pattern, as this individual, a probable female aged 17–21, was shot from the front and fairly high up with the obsidian point partially penetrating the manubrium. The lesion is well bounded (i.e., healed) and thus appears at least moderately long standing. Given the relatively young age of this individual at death, it suggests that quite young individuals, even females, found their way into the firing line of projectiles.

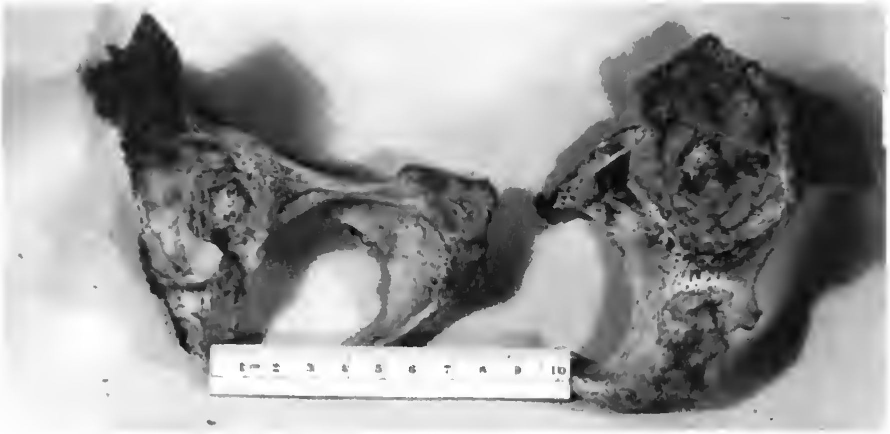


FIGURE 7. Left and right innominate bones, showing hypertrophy of left acetabulum. Female, adult.

The final two cases of projectile wounds both involve the innominate. A young adult male (18–25 years) exhibits a through-and-through lesion of the left ilium 30 mm below the iliac crest on the ventral surface, 40 mm lateral to the sacroiliac articulation.

Evaluation of the bone immediately adjacent to the point was inhibited by the presence of adhesive that had previously been applied to hold the point in place. Nevertheless, evidence of reparative processes is not evident. In fact, it would have been unlikely this individual would have long survived such a wound. The angle of trajectory (from the front) suggests the projectile penetrated the descending colon, small intestine, and intrapelvic vessels, most probably resulting in rapid death.

The last individual with clear evidence of a projectile wound is an adult of indeterminate sex—a very fragmentary burial that was mostly cremated. Here, an obsidian projectile fragment was found in the right ilium approximately 20 mm below the crest and 100 mm anterolateral to the auricular facet. The probable trajectory was anterolateral in relation to the pelvis and may have penetrated the ascending colon causing intra-abdominal infection. In any case, no evidence of healing is seen on gross examination, on magnification, or on x-ray.

The final class of trauma of note that is diagnostic in this population is dislocation. Such lesions do not usually leave their traces on bone frequently enough to approach them epidemiologically. Still, they are of interest, especially the differential diagnosis of traumatic dislocation from congenital problems. In this population a good example of each is seen in the hip. The first example, a probable dislocation, is in an adult female and displays considerable remodeling about the left acetabulum, as though the joint capsule had been ruptured (Figure 7). The femur head is preserved as only a fragment, but still shows the typical hypertrophic appearance of the "mushroom-head." In addition, the lesser trochanter is remodeled, possibly indicating a pulled tendon.

The second case, a female 21–30 years old, has a deformed right acetabulum. The rim is not completely developed, and there is a small nearthrosis inferolaterally. The femoral head is flattened inferiorly and extended dorsolaterally (fitting the nearthrosis on the innominate) (Figure 8). Moreover, the entire right femur appears deformed, is narrower (maximum diaphyseal diameter immediately below lesser trochanter: R = 27.5 mm, L = 33.2 mm) than the left and is twisted approximately 90° along the entire proximal two-thirds of its shaft. In all respects this appears as a very good example of a congenital malformation in both the acetabulum and femur resulting in chronic dislocation of the hip.

A third case of hip dislocation is also of interest. In this case (a male more than 30 years of age) the femur head is flattened and partly mushroomed. Likewise, the acetabulum is greatly hypertrophied and expanded. While no permanent disruption of the joint or nearthrosis is evident, a probable etiology is suggested by other bony changes. On the femur a slight myositis ossificans is seen (medially 47 mm below the lesser trochanter), suggesting a muscle injury of the superior portion of *vastus internus*. In addition, the anterior, inferior iliac spine is moderately hypertrophic (suggesting a further traumatic injury of *rectus femoris*). Thus, while this case may not present the classic picture of a major dislocation, a severe traumatic incident followed by secondary degenerative joint disease is suggested.

Conclusions

While the incidence of trauma resulting from accidents is low in this population, the evidence of interpersonal violence is unusually common. Most healed fractures that were found are in the forearm, and a high proportion of these may have resulted from parrying blows. In addition, the unambiguous evidence of ten embedded projectiles (in nine different individuals plus a probable healed wound from a projectile in another individual) is of remarkably high incidence in this population.

In other North American populations a comparable, high incidence of projectile wounds has not been reported from any single site. For example, at Libben, which includes more than 1300 individuals, no projectile wounds were detected.

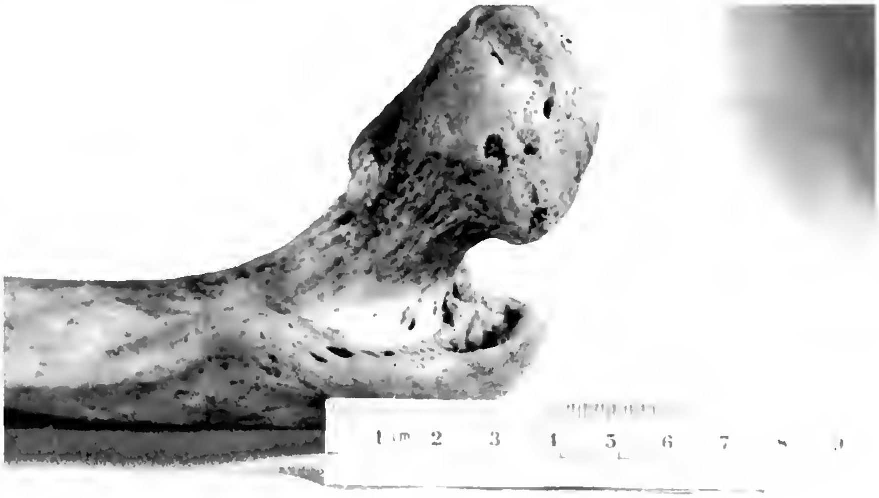


FIGURE 8. Right femur head, deformed inferiorly and dorsolaterally. Female, 21–30 years.

Even in the Old World such high frequencies of unambiguous projectile wounds is but rarely seen. Bennike (1985) in a comprehensive review of basically the entire, prehistoric Danish skeletal collection (including more than 1000 crania as well as thousands of postcranial elements) describes five individuals with six projectile wounds.

Indeed, the only comparable incidence comes from other prehistoric, central Californian contexts. Tenney (1986) reports 18 projectile wounds in 13 individuals from a detailed survey of more than 2000 skeletons housed at Berkeley's Lowie Museum. However, even here the incidence per site is less, as the material represents skeletal samples from several locations.

Thus, the Ala-329 population holds a unique position as perhaps the single most afflicted group with this type of deliberately induced lesion. As noted above in Tenney's review, the high frequency of such wounds in central California, particularly among San Francisco Bay groups, is not a complete surprise. Indeed, a case of a projectile wound in a cranium was received in the 19th century by the Smithsonian Institution from a doctor in Alameda County, the same area as Ala-329 (Wilson 1901).

The evidence, clearly, is more than suggestive. Interpersonal violence at Ala-329 prior to European contact was frequent, deliberate, and often fatal. In fact, the evidence from osseous remains almost certainly underestimates the rate of projectile wounds (and other violence-induced trauma), as no doubt a high proportion of wounds affected only soft tissue (see Wilson 1901 for a discussion of projectile wounds in prehistoric and historic contexts).

An interesting pattern emerges in looking at the skeletal distribution of projectile wounds. All embedded projectile points but one are found low in the body in the vicinity of lower thorax and abdomen (assuming the affected radius was held down at the side when the victim was wounded). While possible, it seems unlikely that such a concentrated cluster would have resulted by chance or even by deliberate aim at distant, moving targets. Therefore, many of the wounds at Ala-329 may have occurred when victims were restrained at close range. Indeed, some of these victims may have been "executed."

Summary

1. Healed fractures in this population are relatively rare, seen in only 36/2047 intact long bones.
2. Of those elements exhibiting healed fractures, the forearm is most often involved (13 radial and 15 ulnar fractures).
3. Of these forearm fractures, an unusually high incidence (for prehistoric samples) of ununited fractures is seen (five elements in four different individuals).
4. Frequent interpersonal violence is suggested by many of these forearm ("parry") fractures and even more clearly by nine individuals who have ten embedded obsidian projectile points.
5. Two cases of traumatically induced hip dislocations are also found; moreover, their differential diagnosis from another case that was congenital in origin is made clear.

Acknowledgments

For their direct contribution to this research and helpful comments on this paper, I am greatly indebted to Lynn Kilgore, Tony Musladen, and Alan Leventhal. Encouragement and assistance with the radiography were provided by Margaret Binns, San Jose State Student Health Service. The analysis of skeletal material could not have been accomplished without the dedication and expertise of students Lorna Pierce, Rhonda Gillett, Charlene Gross, Patricia Rafter, and Sandra Weldon. Grateful appreciation also goes to Bert Gerow for generously allowing access to the collection and to Donald J. Ortner for his friendship and support. Financial support was provided by San Jose State University, School of Social Sciences Research Grant.

Literature cited

Angel, J.L. 1974. Patterns of Fracture from Neolithic to Modern Times. *Anthropologiai Kozlemenyek*, 18:9–18.

Bennike, P. 1985. *Paleopathology of Danish Skeletons.* Copenhagen: Akademisk Forlag.

Edynak, G.J. 1976. Life-Styles from Skeletal Material. In E. Giles and J.S. Friedlaender, eds., *The Measures of Man*, 408–432. Cambridge, Mass.: Peabody Museum Press.

Elliot-Smith, G., and F. Wood-Jones. 1910. *The Archaeological Survey of Nubia.* Report for 1907–1908, vol. 2, Report on the Human Remains. Cairo: National Printing Department.

Jurmain, R.D. 1983. Paleopathology of a Native Californian Skeletal Population. *American Journal of Physical Anthropology*, 60:211–212.

Lovejoy, C.O., and K.G. Heiple. 1981. The Analysis of Fractures in Skeletal Populations with an Example from the Libben Site, Ottawa County, Ohio. *American Journal of Physical Anthropology*, 55:529–541.

Ortner, D.J., and W.G.J. Putschar. 1981. Identification of Pathological Conditions in Human Skeletal Remains. *Smithsonian Contributions to Anthropology*, 28. Washington, D.C.: Smithsonian Institution Press.

Pfeiffer, S. 1985. Paleopathology of Archaic Peoples of the Great Lakes. *Canadian Review of Physical Anthropology*, 4:1–7.

Stewart, T.D. 1974. Nonunion of Fractures in Antiquity, with Description of Five Cases from the New World Involving the Forearm. *Bulletin of the New York Academy of Medicine*, 50:875–891.

Tenney, J.A. 1986. Trauma among Early Californian Populations. *American Journal of Physical Anthropology*, 69:271.

Wilson, T. 1901. Arrow Wounds. *American Anthropologist*, 3:513–53

SUMMARY OF AUDIENCE DISCUSSION: Causes of fracture union failure include recurrent violence before completion of healing and premature use of the arm. Even in the modern period the ulna is the bone most frequently associated with nonunion. Furthermore, frequency estimates of ununited fractures may be lower than reality because of nonrecognition.

Tumors

Tumors in antiquity in East and Middle Europe

Judyta Gladykowska-Rzeczycka

Research on the paleopathology of tumors is problematic. There is an initial problem in deciding which osseous alterations can be called tumors. For example, should we include abnormal bone growths that are the result of trauma such as myositis ossificans? Are bone changes provoked by soft tissue tumors which press on the bone and produce some form of a depression to be considered tumors? Is a bone mass contained entirely within the bone a tumor? The second problem is classifying neoplasms as benign or malignant. Some initially benign tumors may become malignant. As in other areas of paleopathology, tumor nomenclature is a problem in our investigation. An additional factor is that the ancient skeletal material is often in a poor state of preservation. Osteolytic tumors destroy either the affected part or the whole bone but evidence of this may be eliminated by postmortem diagenesis. Similarly, proliferative tumors, such as osteosarcoma, are difficult to evaluate because the original margins of these tumors may have been eradicated either during interment or later during excavation. Diagnosis of even well-preserved cases can be uncertain because bone changes caused by different tumors are very similar, such as the destructive lesions of multiple myeloma and those of metastatic carcinoma of the breast.

Certain modern, precise instrumental methods, including histology and microradiology as well as scanning and transmission electron microscopy, permit recognition of conditions not diagnosable previously. However, all laboratories do not yet possess such equipment so that this type of analysis may not be available in the study of many paleopathological specimens. Papers dealing with tumors are principally descriptive and have a casuistic focus. As a result we have information about many individual cases but we lack statistical data. It is true that all our descriptions and statistical data will never be satisfactory, but, if well done, they can, at least, make the picture of the history of diseases more realistic. If we wish to get such a picture the basic data, in every case we describe, must be more detailed. The basic data include number of all excavated skeletons, state of their preservation, number of all pathological cases, and the sex, age, and archeological provenience of each case.

This summary review of archeological evidence for tumors in Middle and East Europe is limited by the fact that the basic information mentioned above is not always available. On the basis of literature at my disposal I will try to present the "state of tumors" in ancient peoples on the Baltic Coast, Czechoslovakia, ancient Russia, and Poland.

Ancient Baltics

The main source of information about diseases of the ancient Baltics is the book written by Derums (1970). He presents tumors from the Mesolithic, Neolithic, Bronze, and Middle ages (8000 B.C. to 18th century A.D.). About 35 cases of exostoses were observed in 505 skeletons dating from the Mesolithic to the ninth century A.D., but Derums suggests that these are posttraumatic exostoses (p.58). Osteomas were visible in skeletons dated from the fifth to the ninth century A.D., but Derums described only one case—on both clavicles of an adult male from Kriksztonic cemetery (6th–12th century A.D.). This exhibit is in the Museum of History and Ethnography in Vilnius, Lithuania. Two more cases were observed among 710 skeletons dated to the 16th–17th century A.D. One of them (No. 450) comes from Lejasviteni, in which the osteoma is located on the humerus. The second case is on the ulna. Both cases are currently in the Museum of History of Medicine in Riga, Latvia. There are also two cases of osteochondroma (exostosis solitaria). One of them is on the right tibia of a young male (15th–16th century A.D.); the second is not described. The author noted that metastases and other malignant tumors were not observed.

Czechoslovakia

Much information about tumors comes from Czechoslovakia (Table 1). The skeletons examined come from 27 cemeteries dating mainly from the Middle Ages (18 cemeteries), with two from the Period of Wandering Nations, one from Hallstatt (early Iron Age), one dated to the Eneolithic (Chalcolithic), and five from the Bronze Age. Table 1 also

TABLE 1. Reports of neoplasms from Czechoslovakia

Tumor	*Location*	*Inv. no. skeletons excavated*	*Cemetery*	*Period (c=century A.D.)*	*Source*[a]
Multiple myeloma	whole	106	Libice	VII-XIII c	Vyhananek 1969
Metastases	cranium	2	Bratcice	Bronze	Jelinek 1959
Metastases	cranium	1	Knezeves	Eneolith	Chochol, Blajerova 1964
Metastases	cranium	F	Caslavsky Hradek	VII-XIII c	Matiegka 1891
Metastases	cranium	451	Mikulcice	VII-XIII c	Stloukal, Vyhnanek 1976
Metastases	cranium	2	Virt	VII-XIII c	Hanakova et al. 1976
Metastases	vertebra	2	Virt	VII-XIII c	Hanakova et al. 1976
Metastases	vertebra	?	Pribice	Bronze	Stloukal, Vyhnanek 1975
Angioma	cranium	124	Mikulcice I	VII-XIII c	Stloukal 1963, Stloukal, Vyhnanek 1976
Meningioma ?	cranium	30	Nove Zamky	VII-XIII c	Jakab 1977
Osteoid osteoma	tibia	XLVI	Abraham	VII-XIII c	Stloukal, Hanakova 1971, Vyhnanek 1971
Osteoma	cranium	18	Bajc	Bronze	Hanakova et al. 1973
Osteoma	cranium	13/63, 61/63, 26/66, 35/66	Bilina	VII-XIII c	Vyhnanek 1971
Osteoma	cranium	62	Cezavy	Bronze	Jelinek 1959
Osteoma	cranium	DD, H, FF, G, K, II, E	Caslavsky Hradek	VII-XIII c	Matiegka 1891
Osteoma	cranium	5	Dolany	XIV-XIX c	Vurian, Stloukal 1967
Osteoma	cranium	?	Dolny Jator	VII-XIII c	Frankenberger 1935
Osteoma	cranium	133	Josefov	VII-XIII c	Hanakova, Stloukal 1966
Osteoma	cranium	64/57	Lahovice	VII-XIII c	Chochol 1973
Osteoma	cranium	28	Levy Hradec	VII-XIII c	Matiegka 1891
Osteoma	cranium	13, 43, 80a, 86, 115, 199, 262, 4337 a/b	Libice	VII-XIII c	Vyhnanek 1969
Osteoma	cranium	124	Mikulcice I	VII-XIII c	Stloukal 1963, Stloukal, Vyhnanek 1976
Osteoma	cranium	604	Mikulcice II	VII-XIII c	Stloukal 1963, Stloukal, Vyhnanek 1976
Osteoma	cranium	86/IV	Mikulcice III	VII-XIII c	Stloukal 1969
Osteoma	cranium	59, 220	Nove Zamky	VII-XIII c	Stloukal, Hanakova 1966
Osteoma	cranium	92	Nove Zamky	VII-XIII c	Vladarova-Mojzisova 1968
Osteoma	cranium	100, 113	Oskobrh	XIV-XIX c	Blajerova 1974
Osteoma	cranium	?	Praha-Troja	Hallstatt	Hanakova 1971
Osteoma	cranium	63/68, 220, 305, 357	Radomysl	VII-XIII c	Blajerova 1974
Osteoma	cranium	16	Sakvice	W.o.N.[b]	Stloukal 1977
Osteoma	cranium	4	Vazany	VII-XIII c	Matiegka 1909
Osteoma	cranium	3	Veterov	Bronze	Jelinek 1964
Osteoma	cranium	23	Vyskov	W.o.N.	Stloukal 1973
Osteoma	cranium	619	Zelovce	VII-XIII c	Stloukal, Hanakova 1974
Osteoma	cranium	?	? Karel IV	XIV-XIX c	Matiegka 1932
Osteoma	sacrum	26/60	Lahovice	VII-XIII c	Chochol 1973
Osteoma	femur	53/60	Bilina	VII-XIII c	Vyhnanek 1971
Cyst	humerus	30	Nove Zamky	VII-XIII c	Stloukal, Hanakova 1966
Cyst	metatarsal I	31	Mikulcice I	VII-XIII c	Stloukal, Vyhnanek 1968, 1969

a. All sources referenced in summary articles by Hanakova and Vyhananek (1981) and Hanakova (1983).
b. W.o.N. = Wandering of Nations.

TABLE 2. Tumors from ancient Russian skeletons

Tumor	*Cemetery*	*Period*	*Material*
	EUROPEAN USSR		
Osteochondroma	Sarkiel	X-XI c AD	1F ad
			1M m
Enchondroma	Sarkiel	X-XI c AD	1?
	Stara Wjatka	?	1?
Osteoma	Sarkeil	X-XI c AD	2?
			1? s
Solitary exostosis	Sarkeil	X-XI c AD	1 M j
Metastases	Sarkiel	X-XI c AD	1 F, 1 M ad/m
	ASIAN USSR		
Osteochondroma	Hakassk (Krasnojarsk)	X-VIII c BC	1 ? ad
Multiple exostoses	Resp. Tuwinska	XIII c BC	1 ? i/I
	Saragasz (Krasnojarsk)	IV-III c BC	1 M j
Hemangioma	Resp. Krasnojarsk	IV-III c BC	1 ?
Metastases	Saragasz (Krasnojarsk)	VIII c BC	1 F m
		VIII c AD	1M m
		?	2?
	Usoienskoje (Tuwinska)	III c BC	1 F ad
	Bijsk	I c BC	1 M m
	Dandybaj (Kazachstan)	1500 BC	1 F 40–45
Multiple myeloma	Kyzyl-Dzar (Altaj)	IV-III c BC	1 ?
	Kamien 2 (Altaj)	III-II c BC	1 M ?

ABBREVIATIONS: c, century; F, female; M, male; ad, adult; m, mature adult; s, senile adult; j, juvenile; i, infant; I, indeterminate

SOURCES: Rochlin (1963); data for Kyzyl-Dzar and Kamien from Zacharow et al. (1983)

lists the kinds of tumors, their frequency, their location, and time period. The only case of multiple myeloma comes from a Medieval skeleton (7th–13th century) at Libice where there were also eight cases of osteoma. In the Medieval Caslavsky Hradek cemetery one case of metastasis and seven of osteoma were observed, in the Mikulcice II burial ground one case of metastasis and one of osteoma, and in the Virt cemetery two cases of metastases. Other neoplasms were found in Bartcice and Pribice cemeteries, both dating from the Bronze Age. Most of the tumors were benign—52 (86.6%). They include 45 osteomas (86.5% of all benign tumors), two cysts, one angioma, one meningioma, and one osteoid osteoma. Eight cases (13.4%) are malignant: one multiple myeloma and seven metastases.

Ancient Russia

Tumors from ancient Russia are listed in Table 2. It is very difficult to present these data because some diagnoses are equivocal. In the Sarkiel cemetery (10th–11th century A.D.) among 294 skeletons there are nine burials (3.1%) with tumors, seven benign and two malignant. More malignant tumors are known from the Asian part of Russia. This part of Russia also presents the only case of tumor known to be transmitted in a hereditary fashion: exostoses multiplices (manifesting as multiple exostoses).

Poland

Basic data on tumors from Polish cemeteries are presented in Table 3 (and see Gladykowska-Rzeczycka 1978, 1982, 1985; Gladykowska-Rzeczycka and Mysliwski 1986). Skeletons are found in two Neolithic and eight Medieval cemeteries, but no tumors were observed in two of the Medieval cemeteries and they are not included in the table. The state of preservation of these bones is not good, but as we have learned, such specimens can still reveal identifiable tumors.

Benign tumors include 23 (37.7%) "ivory" osteomas, 19 (31.1%) exostoses solitaria, 2 (3.3%) osteoid osteomas, 1 (1.6%) ameloblastoma, 1 (1.6%) chondroma, 4 (6.6%) angiomas, 1 (1.6%) probable meningioma, and 2 (3.3%) cysts. Malignant tumors were observed in three individuals (4.9%). Five skeletons (8.2%) had changes probably produced by tumors.

TABLE 3. Tumors from Polish cemeteries (Neolithic and Middle Ages)

	Mierza-nowice		*Zlota*		*Czersk*		*Czarna Wielka*		*Suraz*		*Pyrzyce*		*Szczecin Loco*		*Ostrow Lednicki*	
	Inv. no.	Sex/ age	Inv. no.	Sex/ age	Inv. no.	Sex/ age	Inv. no.	Sex/ age	Inv. no.	Sex/ age	Inv. no.	Sex/ age	Inv. no.	Sex/ age	Inv. no.	Sex/ age
Osteoma, "ivory"																
cranium	-	-	-	-	-	-	p9.s.	F m/s	-	-	-	-	-	-	-	-
	"a"	F m/s	74	M ?	338	M m	10a	M m	-	-	20	M m	-	-	1085	F ad
	-	-	-	-	-	-	4/II	F ad/m	-	-	-	-	-	-	376	F ad
	-	-	246	M ad	47?	M m	17/II	M m/s	-	-	-	-	-	-	1126	F ad
	-	-	-	-	-	-	18/II	M m/s	-	-	-	-	-	-	671	M m/s
	-	-	?	M m	-	-	36/67	M m/s	-	-	-	-	-	-	476	M ?
	-	-	-	-	-	-	28/I	F ad	-	-	-	-	-	-	9a	M s
	-	-	-	-	-	-	173	? m/s	-	-	-	-	-	-	-	-
ilium	-	-	-	-	-	-	-	-	-	-	-	-	-	-	478	M ad/m
vertebra	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1094	F m
TOTAL 23 (37.7%)																
Solitary exostosis																
costa	-	-	93	M m	-	-	-	-	-	-	-	-	-	-	56	? m
	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1120	? m
humerus	-	-	-	-	-	-	28	M ad	21	M ad/m	-	-	-	-	225	M ad
coxa	-	-	-	-	120	F ad	-	-	-	-	-	-	-	-	-	-
femur	-	-	-	-	122	M m	190	M m/s	-	-	-	-	-	-	1473	? ?
	-	-	-	-	-	-	?	? m	-	-	-	-	-	-	683	M m
tibia	-	-	18/II	M m/s	32	i II	1b/I	M m	-	-	-	-	-	-	-	-
	-	-	-	-	-	-	62/67	M m/s	-	-	-	-	-	-	-	-
fibula	-	-	7/IX	F ad	-	-	-	-	-	-	-	-	-	-	370	F ad
phalanx	-	-	-	-	-	-	1227	? m	-	-	-	-	-	-	-	-
TOTAL 19 (31.1%)																
Osteoid osteoma																
tibia	-	-	-	-	-	-	-	-	-	-	29	? m	1/74	M ad?	-	-
TOTAL 2 (3.3%)																
Ameloblastoma																
mandibula	-	-	-	-	286	M m	-	-	-	-	-	-	-	-	-	-
TOTAL 1 (1.6%)																

Chondroma																
1 metacarpal	-	-	2	F? ?	-	-	-	-	-	-	-	-	-	-	-	-
TOTAL 1 (1.6%)																
Angioma																
cranium	-	-	-	-	-	-	-	-	3	i I	-	-	-	-	791	? m
vertebra	-	-	-	-	-	-	-	-	-	-	-	-	-	-	792	F m
	-	-	-	-	-	-	-	-	-	-	-	-	-	-	259	F ad
TOTAL 4 (6.6%)																
Meningioma																
cranium	-	-	-	-	-	-	169	M s	-	-	-	-	-	-	-	-
TOTAL 1 (1.6%)																
Cyst																
cranium	-	-	-	-	-	-	-	-	23	M m	-	-	-	-	-	-
ulna	-	-	-	-	-	-	-	-	-	-	-	-	-	-	101	F ad/m
TOTAL 2 (3.3%)																
Malignant																
multiple myeloma	-	-	-	-	-	-	-	-	-	-	-	-	-	-	1202	F ad/m
metastases	-	-	-	-	92	F ad	-	-	-	-	-	-	-	-	1184	F m
TOTAL 3 (4.9%)																
Unidentified																
ilium	-	-	-	-	575	i II	-	-	-	-	-	-	-	-	-	-
femur	-	-	-	-	207	M ad	-	-	-	-	-	-	-	-	-	-
tibia	-	-	-	-	-	-	17/1	M m	-	-	-	-	-	-	-	-
cranium	-	-	-	-	-	-	214	? ad/m	-	-	-	-	-	-	313	M m
TOTAL 5 (8.2%)																
TOTALS[a]	1/67/143		7/88/218		9/315/624		17/194/250		3/45/55		2/9/31		1/2/4		21/503/1126	
%[b]	0.7/1.5/±30		3.2/8.0/±40		1.4/2.9/±50		6.8/8.8/±60		5.5/6.7/±70		6.5/22.2/±30		25.0/50.0/±20		1.9/4.1/±80	

NOTE: Abbreviations as in Table 2.

CEMETERY DATA: *Mierzanowice*: Tarnobrzeg district (Sandomierz district before 1975); Neolithic. *Zlota*: Tarnobrzeg district; Neolithic. *Czersk*: Warsaw district (Piaseczno district before 1975); 12-13th century. *Czarna Wielka*: Biylystok district (Grodzisk district before 1975); 12-13th century. *Suraz*: Biylystok district; 11-14th century. *Pyrzyce*: Szczecin district; 14-15th century. *Szczecin Loco*: 14-15th century. *Ostrow Lednicki*: Poznan district; 10-12th century; material diagnosed by M. Cwirko-Godycki (Cwirko-Godycki and Swedborg 1978).

a. Totals given are numbers of tumors/pathology specimens/excavated skeletons.

b. Percentages given are for tumors/pathology cases/well-preserved bones.

TABLE 4. Summarized findings from all areas

Location	*Total skeletons*	*No. of tumors* *Benign*	*Malignant*	*Total tumors*	*% skeletons with tumors*
Czechoslovakia	2584	52 (86.7%)	8 (13.3%)	60	2.3 (2.0)
Poland	2653	52 (95.1%)	3 (4.9%)	61	2.3
Baltic Coast	2763	5 or 45 ?	0	45 ?	0.2? (1.6)
Russia (East Europe)	?	8 ?	2 ?	10 ?	?
Sarkiel X-XI c	294	7 (77.4%)	2 (22.6%)	9	3.1

Of the Neolithic sites, the Mierzanowice cemetery has only one case of osteoma in 67 skeletons with pathologic changes. In the Zlota cemetery, tumors were found in seven individuals out of 88 skeletons with lesions. Among the Medieval cemeteries, the populations most commonly affected by tumor come from cemeteries at Czarna Wielka and Ostrow Lednicki. The preservation state of the skeletons from these cemeteries is relatively good (60–80%). The most dramatic cases—malignant tumors—come from the Czersk and Ostrow Lednicki cemeteries.

A total of 61 tumors were observed in all parts of the skeleton. Most are located in the skull (28) and the lower extremities (19), especially in males (16.4%). The frequency of neoplasms in males is higher (50.8%) than in females (31.1%) but the malignant tumors (3) were observed only in females. Of 31 males, 5 were adults, 2 adult-mature, 15 mature, 6 mature-senile, 1 senile, and 2 of unknown age; of 19 females, 11 were adults, 2 adult-mature, 2 mature-senile, and 1 of unknown age. There were three cases of neoplasms in children and eight cases in adults of unidentified sex (2 adults, 1 adult-mature, 3 mature, and 2 of unknown age).

There are also cases of tumors known from other cemeteries but they are presented by their authors (Komitowski 1975; Halka 1935; Spettowa and Koczanowski 1973) in a very general way; all (± 10) are reported as benign. They are not included in this paper.

Summary

Summarized results are shown in Table 4. In the material from Czechoslovakia, tumors were found in about 2% of all 2584 excavated skeletons; 86.7% were benign and 13.3% malignant. From 10 Polish cemeteries (tumors found in only 8 of them), 61 (2.3%) neoplasms were found in 2653 excavated individuals; 95.1% were benign, 4.9% malignant. Tumors from the Baltic Coast are sporadic. Only 5 or 45 cases (0.2 or 1.6%) were observed among 2763 skeletons, dated from Mesolithic up to the 18th century A.D. None were malignant. Information from the eastern part of Russia is problematic. Only one cemetery from Sarkiel is presented in detail. There were 9 tumors (3.1%) in 294 excavated skeletons of which 77.4% were benign and 22.6% malignant.

Literature cited

Cwirko-Godycki, M., and M.J. Swedborg. 1978. Anthropometric Characteristics of Bones from a Medieval Cemetery at Ostrow Lednicki and Analysis of Pathological Symptoms Occurring on Them. *Przeglad Antropologiczny*, 44:221–239.

Derums, W.J. 1970. Diseases and Treatment in the Ancient Baltics. *Zinatne*. Riga, USSR.

Gladykowska-Rzeczycka, J. 1978. Mandibular Tumor in a Male Skeleton from a Medieval Burial Ground in Czersk. *Folia Morphologica*, 37:191–196. Warsaw.

———. 1982. Neoplasms from Ancient Cemeteries in Poland. *Anthropos*, 21:353–364. Brno, Czechoslovakia.

———. 1985. Development and Program of Studies on Paleopathology in Poland. *Teoria i Empiria w Polskiej Szkole Antropologicznej, S. Antrop.*, Nr. 11. Poznan, Poland.

Gladykowska-Rzeczycka, J., and A. Mysliwski. 1986. Osteoid Osteoma from a Middle Ages Cemetery in Poland. *Ossa*, 12:33–39.

Halka, S. 1935. Czaszki z XV-XVIII Wieku Pochodzace z Dawnego Cmentarzyska Kolo Kosciola SW. Marcina w Poznaniu. *Przeglad Antropologiczny*, 9:47–54.

Hanakova, H. 1983. The Frequency of Various Types of Paleopathological Finds on Czechoslovak Territory. *Casopis Narodniho Muzea v Praze*, 152:123–129.

Hanakova, H., and L. Vyhnanek. 1981. Palaeopathologische Befunde aus dem Gebit der Tchechoslovakei. *Sbornik Narodniho Muzea v Praze*, 37B(1).

Komitowski, D. 1975. Paleopathological Examinations of Bone Remains from an Early Medieval Cemetery at Zlota, Pinczow District. *Wiadomosci Archeologiczne*, 40:113–118.

Rochlin, D.G. 1966. *Diseases of Ancient Man*. Moscow and Leningrad: Nauka.

Spettowa, S., and K. Kaczanowski. 1973. Therapeutic Skull Trepanation Done for Intracranial Space-Occupying Lesion in Middle Ages Found in Archaeological Investigations in Krakow-Zakrzowek. *Neurochirurgia Polska*, 7(23)3:333–338.

Zacharow, B.J., H.P. Zacharowa, and J.A. Bielawskij. 1983. Manifestations of Multiple Myeloma in the Bones of Ancient People. *Woprosy Onkologji*, 29:86–88.

SUMMARY OF AUDIENCE DISCUSSION: Unexpectedly high frequencies of certain benign tumors (especially osteomas) in some of these collections may be due to difficulty in differentiating reactive (such as myositis ossificans) from neoplastic processes, a problem created by the descriptive vagaries in the reviewed reports.

Human soft tissue tumors in paleopathology

Enrique Gerszten and Marvin J. Allison

Since the beginning of this century, the main cause of death in the United States has changed from tuberculosis to diseases of the heart. Although cancer is not the main cause of death, the percentage of cancer-related deaths has steadily increased, today reaching more than 22.1% of the total number of deaths in the United States (American Cancer Society 1988). It is of interest that in the last half-century the primary sites of malignant tumors have also changed. In some cases we know the factor or factors involved in these changes, while in others we do not.

In the United States, the age-adjusted cancer death rates for selected sites in males show that carcinoma of the stomach has fallen from about 38 deaths per 100,000 in 1930 to below 10 in 1985. Carcinoma of the lung, on the other hand, rose markedly during the same period from around 5 deaths per 100,000 to more than 70. During this same time period, carcinoma of the esophagus has remained rather constant at 5/100,000, while prostatic carcinoma has risen from 15 to 25. The rates for primary carcinomas in other organs have not changed significantly over the same time period.

This same table of statistics reveals that, among females, invasive cancer of the uterus has fallen from 31/100,000 to fewer than 10 in 1985, while carcinoma of the lung has risen from around 3 in 1930 to almost 30, surpassing carcinoma of the breast in 1986 as the leading cause of cancer deaths in females. In females, carcinoma of the stomach has diminished considerably as it has in males, while carcinoma of the breast has persisted at a frequency of about 27/100,000.

These figures show that in a short period of time different types of tumors have changed in their frequency. In some types, such as carcinoma of the uterus, cervix, colon, and stomach, these changes can be attributed to preventive measures, changes in diet or association with certain substances. Other tumors, such as carcinoma of the breast, ovary, or pancreas have maintained the same frequency patterns, unexplainable by present cancer research.

A complete review of the literature of tumors in antiquity shows that in different parts of the world only very few types of tumors have been recorded. It is understandable that most of the findings are described in bones, mainly in the skull, because these parts of a buried human body are more resistant to deterioration over time. The literature on this subject has dealt mostly with primary or metastatic tumors of bone. Today it is known that certain soft tissue tumors such as breast and prostate have a high frequency of metastases compared to bones. Only a few tumors of soft tissue have been documented in antiquity. The study of this subject can lead to a better understanding of the history of cancer.

Materials and methods

Table 1 is arranged by geographic areas and contains the total list of literature references to primary tumors of soft tissues reported from antiquity. The last two soft tissue tumors listed in the table were found in our studies of pre-Columbian South American mummies.

The first tumor, a benign lipoma, was an incidental finding in a male adolescent age 14, of the San Miguel Culture of Northern Chile from A.D. 1100 to 1200. This mummy was in poor condition as only its heart and lungs were found at autopsy; the rest of the internal organs had been destroyed.

TABLE 1. Literature sources of reported soft tissue tumors from antiquity

Geographic area	*Type of tumor*	*Reference*
Egypt	Histiocytoma	Zimmerman 1981
	Leiomyoma	Strouhal 1976
	Cystadenoma	Rowling 1961
	Basal cell nevus syndrome	Satinoff and Wells 1969
	Squamous papilloma	Sandison 1967
Europe	Leiomyoma	Kramar et al. 1983
South America	Lipoma	Present report
	Rhabdomyosarcoma	Present report

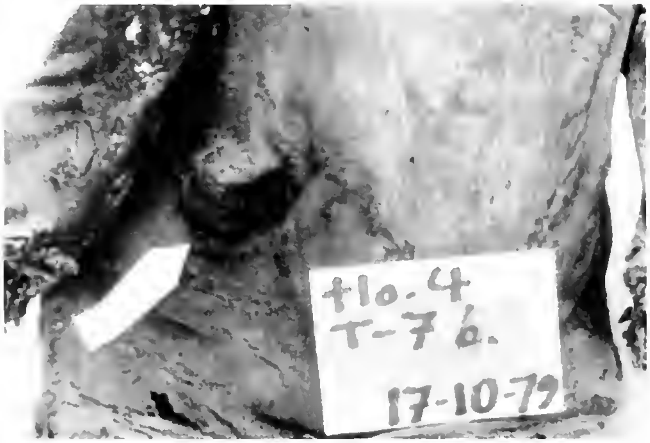


FIGURE 1. Subcutaneous lipoma in right side of chest.

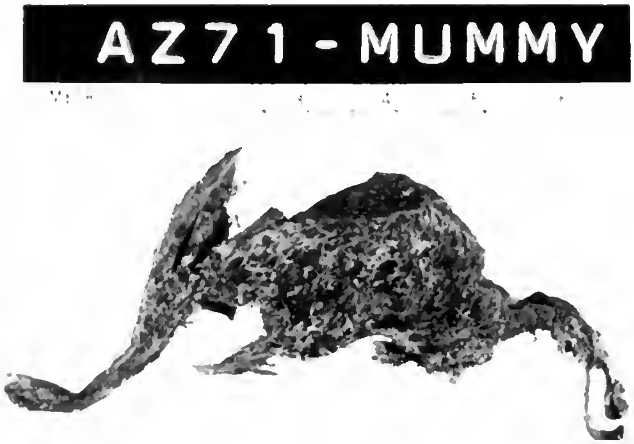


FIGURE 3. Cross section of tumor on right cheek below eye.

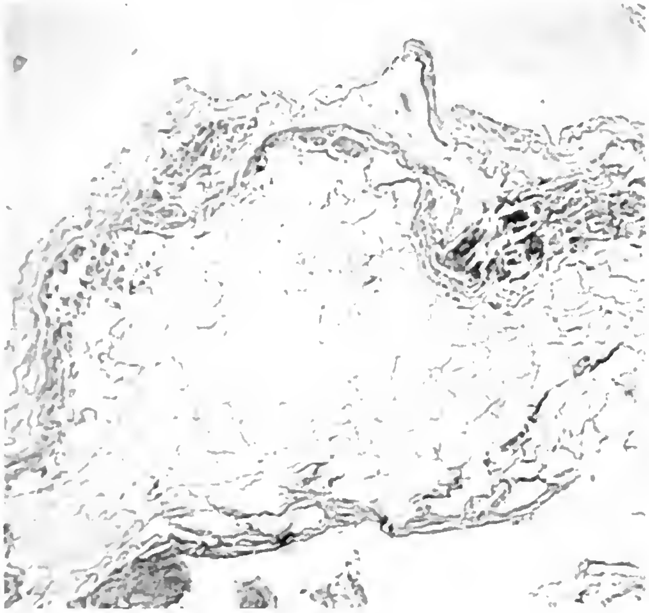


FIGURE 2. Microscopic features of a lipoma.

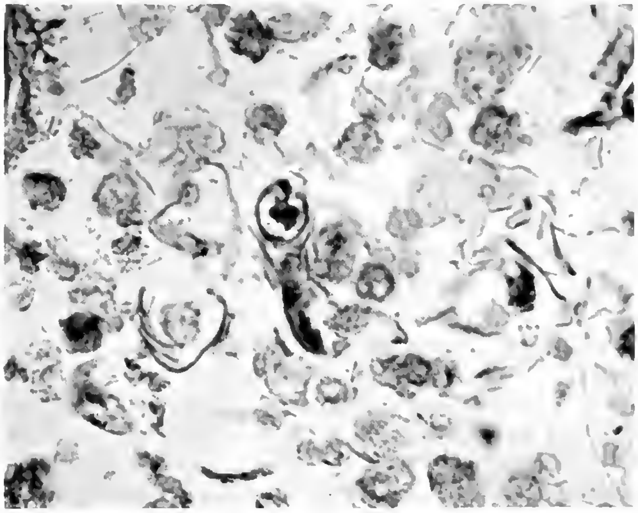


FIGURE 4. Microscopic features of tumor composed of malignant cells.

On gross examination there was a 4 × 4 × 2-cm subcutaneous mass on the right side of the chest approximately 6 cm below the axilla (Figure 1). Histologic sections of the chest wall mass show a conglomeration of fat cells intermingling with fibrous septa (Figure 2). As occurs normally in most paleopathological studies, the nuclei of the neoplastic cells were not seen. The findings are consistent with previous descriptions of lipomas (Robbins et al. 1984:270–271).

The second soft tissue tumor is consistent with a rhabdomyosarcoma. It was found in a male child, approximately 12 to 18 months old, of the Cabuza culture (A.D. 300–600) of northern Chile. The lesion was that of a hard swelling on the mummy's right cheek below the eye, forcing that eye closed, and measured 5.5 × 5 × 2 cm (Figure 3). The bones of the right orbit were normal, as were the lungs, heart, and liver. The cause of death could not be determined.

The histology of this kind of tumor usually shows cells forming islands or broad cords, separated by fibrovascular stroma (Robbins et al. 1984:1316). The histopathology of the present case shows pleomorphic, disintegrated cells surrounded by a delicate, fibrous stroma (Figure 4). Most of the cells show a shrunken cytoplasm, and in a few instances nuclear material can be observed. This marked pleomorphism is characteristic of the tumor. Alveolar rhabdomysarcoma occurs chiefly in young persons under the age of 20 (Enzinger and Shiraki 1969) and, in children, more than 55% of the cases have primary tumor locations in the head and neck (Weichert et al. 1976). This tumor can be confused easily with an undifferentiated carcinoma or a lymphoma.

Discussion

It is impossible to estimate how many total mummies, complete skeletons, and partial bones have been examined for tumors since the initial studies of paleopathology by Ruffer in the early part of this century. It may be stated that only a few dozen neoplasms, the majority in bones, have been recorded from the thousands of ancient bodies. Why is this scarcity of tumors still an enigma?

It has been suggested that in antiquity people did not live long enough to develop tumors, that age was the most important factor in determining who would develop neoplastic lesions. However, in many of the 23 pre-Columbian cultures we have studied, at least 40% of the population lived past the age of 40 years, including them in a geriatric population. Many of these cultures had a geriatric survival rate of greater than 25%.

In the last century, numerous substances have been introduced into the daily lives of humans that have been associated with carcinogenesis, such as asbestos, azo dyes, etc. Humans were not in contact with the large majority of these substances in earlier eras; in fact, most of these carcinogenic substances are products developed after the industrial revolution. Among the known carcinogenic factors associated with ancient cultures were radiation energy from the sun and tobacco. There are no published reports of malignant tumors of the skin in ancient populations that lived in tropical areas most affected by the solar rays. It is also known that many of the primitive societies used tobacco, and often individuals inhaled large quantities of smoke, both from tobacco and from cooking/heating fires. Not a single carcinoma of the lung has yet been found.

It is now known that diet has major implications in the incidence of cancer in today's world, most notably affecting carcinomas of the gastrointestinal tract. It is of interest to note that among the pre-Columbian Indian studies, the low incidence of cancer did not change with the different diets of the area. The diets of those Indians varied greatly over relatively short distances, with some foodstuffs limited to very small geographic locations. The diet of the coastal economy in Chile, for example, was dependent mostly on seafood, while a short distance inland the diet of the mainland cultures consisted of foodstuffs from both agriculture and hunting.

The study of neoplasms in antiquity is a difficult task. Though most tumors are of soft tissue origin, most of the material available is bone. The possibility of missing tumors in paleopathological studies has been suggested, but Zimmerman, in experimental studies, has shown that modern tumors can be mummified and rehydrated later and that sections taken from this material can be easily interpreted (Zimmerman 1977). This evidence suggests that tumors are not "missed," but that they were indeed less frequent in antiquity.

The factor likely to play the most decisive role in the incidence of cancer is the genetic structure of the individual. In the case of primitive societies, Klepinger has suggested that these people may have had immune systems that later became depressed secondary to as yet undiscovered reasons, thereby allowing benign factors associated with these ancient men to become oncogenic (Klepinger 1980). In support of this theory she points out that certain papova viruses that have been observed to be benign can in turn become oncogenic in laboratory animals with depressed immune systems.

Conclusions

The total number of documented soft tissue tumors from ancient civilizations is fewer than 10, including the present findings of a lipoma and a rhabdomyosarcoma, with more than a thousand complete autopsies performed in our studies in Peru and Chile of pre-Columbian mummies alone. The most important factors for this low incidence of neoplastic lesions in mummified materials include the facts that almost all of the known carcinogenic agents prevalent in today's world have only recently been brought into contact with humans, and that the immune system of ancient populations may have been different. Additional research in the pathological investigation of mummified soft tissues may disclose further neoplastic lesions and, working with a team of immunologists, anthropologists, geneticists, and epidemiologists, we may one day arrive at the factors involved in the pathogenesis of neoplasia.

Literature cited

American Cancer Society. 1988. Cancer Statistics. *Ca—A Cancer Journal for Clinicians*, 38:1–22.

Enzinger, F.M., and M. Shiraki. 1969. Alveolar Rhabdomyosarcoma: An Analysis of 110 Cases. *Cancer*, 24:18–31.

Klepinger, L.L. 1980. The Evolution of Human Disease: New Findings and Problems. *Journal of Biosocial Sciences*, 12:481.

Kramar, C., C.A. Baud, and R. Lagier. 1983. Presumed Calcified Leiomyoma of Uterus: Morphologic and Clinical Studies of a Calcified Mass Dating from the Neolithic Period. *Archives of Pathology and Laboratory Medicine*, 107:91–103.

Robbins, S.L., R.S. Cotran, and V. Kumar. 1984. *Pathologic Basis of Disease*. Philadelphia: W.B. Saunders.

Rowling, J.T. 1961. *Disease in Ancient Egypt: Evidence from Pathological Lesions Found in Mummies*. M.D. thesis. Cambridge, U.K.: University of Cambridge Press.

Sandison, A.T. 1967. Diseases of Skin. In D. Brothwell and A.T. Sandison, eds., *Diseases in Antiquity*, 449–456. Springfield, Ill.: Charles C Thomas.

Satinoff, M.J., and C. Wells. 1969. Multiple Basal Cell Nevus Syndrome in Ancient Egypt. *Medical History*, 13:294–297.

Strouhal, E. 1976. Tumors in the Remains of Ancient Egyptians. *American Journal of Physical Anthropology*, 45:613–620.

Weichert, K.A., K.C. Bove, B.S. Aron, and B. Lampkin. 1976. Rhabdomyosarcoma in Children: A Clinicopathologic Study of 35 Patients. *American Journal of Clinical Pathology*, 66:692–701.

Zimmerman, M.R. 1977. An Experimental Study of Mummification Pertinent to the Antiquity of Cancer. *Cancer*, 40:1358–1362.

———. 1981. A Possible Histiocytoma in an Egyptian Mummy. *Archives of Dermatology*, 117:364–368.

SUMMARY OF AUDIENCE DISCUSSION: We do not know when the high incidence of cancer began. We know the frequency was low in antiquity—at least in South America's Acacama desert where climatic conditions provide such good soft tissue preservation that it is difficult to overlook cancers that now occur commonly in such areas as the breast and where study material up to 8000 years old and as recent as 500 years ago is available. Unfortunately good records of cancer during the historic period have only been kept during the past century. The high frequency of lung cancer in Hungary might be influenced by air pollution there.

Identification and study of carcinoma in paleopathological material: Present status and future directions

James M. Tenney

Soft tissue has not received the extensive study that bone has in paleopathology owing largely to the limited availability of material and the more destructive nature of the techniques involved. The discussion centers about past effort in soft tissue paleopathology with respect to carcinoma, and its relation to present and future studies, problems, limitations, and potential value. Carcinomas of prostate, colon, and breast are taken as prototypes representing common present-day tumors.

Carcinoma is defined as a malignant neoplasm arising from an epithelial surface, be it skin, the lining of an organ (colon, bronchus of lung), the epithelium-lined ducts of an organ (breast, liver), or small epithelial glandular structures present in the organ itself (prostate, pancreas). Though the term "soft tissue tumor" is used somewhat differently in paleopathology, its general usage in pathology refers to any neoplasm arising from mesenchymal tissue other than bone, bone marrow, cartilage, or lymph nodes, and excluding carcinoma (Stout and Lattes 1967:15).

In the older medical literature and that of ancient times, the word "tumor" referred to any swelling, whether it was infectious (boil or abscess), traumatic ("goose-egg"), or neoplastic (benign or malignant). The large abdomen in a gestational woman was even referred to as the "ovoid tumor of pregnancy." Current usage restricts the meaning to neoplasia, benign or malignant. This would exclude many lesions classified as tumors in the paleopathology literature (auditory osteomas, "collar-button" osteomas, congenital and traumatic epidermoid cysts, tori, myositis ossificans, osteochondromas, etc.). An acceptable alternative would be to refer to these as "tumor-like conditions" (Aegerter and Kirkpatrick 1968: 546).

Past

In the past there has been a fair amount of descriptive literature regarding cancer in ancients, mostly in bone. Some was carefully documented and described, with cautious conclusions as to general diagnosis of cancer and sometimes even a specific diagnosis (for example, osteogenic sarcoma). A well thought-out differential diagnosis was included. In other cases, however, a hodgepodge of fanciful guess work with little scientific basis was submitted with no mention of other diagnostic possibilities. Both kinds become equals in the literature and get incorporated into tables and statistics of later papers, becoming translated as fact. There have been few attempts to restrain excessively speculative diagnoses.

Only a handful of nonosseous tumors have been described, and even fewer with microscopic findings (Zimmerman 1981:364). A recent survey summarizes bone and soft tissue tumors in Egypt and Nubia (Pahl 1986).

Uniformity of diagnosis presents a recurring problem. This lack is not surprising, as there are more problems than answers in paleopathology. It is always tempting to classify things, and some of the problems might be placed in the following manner:

1. Problems inherent in the archeological site
   a. provenience
   b. is the burial representative of the group at that site?
   c. is the group at that site representative of the population as a whole at that time and at that place?
2. Problems with the specimen itself
   a. incomplete material (bones and/or soft tissue missing or partially missing)
   b. poor condition or preservation of the specimen

3. Problems with the investigative process
   a. oversight (not seeing the lesion)
   b. inexperience (not recognizing the lesion if seen)
   c. not seeing the lesion because it is too small to be detected visually on the surface
   d. clerical misidentification
4. Problems related to diagnosis
   a. pseudopathology (Wells 1967)
   b. determination of normal and range of normal (Dastugue 1986)
   c. insufficient criteria present to support the diagnosis given
   d. the lesion seen represents a disease no longer present, or one rarely seen
   e. modern-day incidence not applicable owing to changing longevity, better treatment, etc.
5. Problems related to conclusions
   a. even if the diagnosis is correct, there may be insufficient numbers to have statistical significance.

It is with one of these areas, problems related to diagnosis, that some progress could be made. First, it should be recognized that negative findings do not exclude a disease process, as the disease may have had a fulminant course or the individual may have died of intercurrent disease of some other sort before his primary disease had had time to manifest its full expression. The problem of host resistance is one of the important issues that faces paleopathologists, and is also one of the potentially more rewarding products of our efforts. The matter of provenience is obviously essential. It is difficult to appreciate the paleopathologic, paleoepidemiologic or any other usefulness of a diagnosis such as "possible carcinoma in a mummy of unknown provenience." Quite aside from this, matters of provenience become important in diagnosing a given disease—did it exist during the same era and in the same geographical area as the individual? We know that diseases have their own evolution, with some disappearing (smallpox, poliomyelitis) and others appearing apparently *de novo* (AIDS). Some diseases change their geographic distribution depending on socioeconomic and other factors (measles, cancrum oris). Vaccinations, antibiotics, and public health measures determine these changes in geographic distribution to quite an extent, although not entirely. The form as well as the manifestation and severity of a given disease are variable.

Diagnostic criteria for disease need to be established. An example of criteria derived for a very difficult group of diseases is given for the treponematoses (Hackett 1978). Where insufficient criteria are present for a specific diagnosis, one should not be made.

Current practice

In modern clinical medicine, diagnostic criteria are established and well known for carcinoma. They all ultimately depend upon an unequivocal, microscopic appearance of a representative tissue biopsy for definitive diagnosis. This is the state of the art, and has been for decades, whether the carcinoma be of breast, colon, prostate or some other site. Under normal circumstances there is a definite, diagnostic sequence of events:

HISTORY. The patient's complaint that brings him to the physician, along with related matters such as duration, family history, environment, and so forth.

PHYSICAL EXAMINATION. The presence of a lump in the breast, abdominal mass, enlarged liver, or stony hard prostate gland may be noted.

These two items, the history and physical examination, suggest a differential diagnosis. A plan is then made to narrow the list by ancillary methods.

RADIOLOGICAL STUDIES. X-ray, computed tomagraphy (CT) scan, magnetic resonance imaging (MRI), and so forth, may show a distribution or pattern of bony and soft tissue changes to suggest a diagnosis of carcinoma and occasionally even a likely primary site.

LABORATORY STUDIES. Certain blood tests such as serum levels of carcinoembryonic antigen (CEA; elevated in carcinoma of the colon), serum prostatic acid phosphatase (elevated in carcinoma of the prostate), and serum alkaline phosphatase (reflecting bony destruction/regeneration from any cause) are sometimes useful. Serum protein electrophoresis and immunoelectrophoresis are virtually diagnostic of multiple myeloma when positive.

As the differential diagnosis is narrowed, an operative procedure is planned: whether to remove the entire tumor (excisional biopsy) and possibly attempt a cure, or to remove only a portion of the tumor (incisional biopsy). Both result in obtaining sufficient tissue for microscopic confirmation of the clinical impression.

It is now, and only now, that the "100% certain diagnosis" can be made, and up to this point, there is still a differential diagnosis. The question arises as to whether there is a place for something less than the "100% certain diagnosis." In some instances a patient is terminally ill or his condition is too poor to consider surgery. In other cases, prognosis is too poor to attempt any extensive diagnostic workup, and the only procedures considered are to be palliative. Even then, some sort of assurance other than a history and physical examination is required that the disease is, in fact, carcinoma and that the extent and severity is as imagined. An example of a "75% certain diagnosis" for carcinoma of the breast would be a terminally ill lady with a palpable breast mass and a bony metastatic pattern on x-ray consistent with a primary in the breast. An equivalent level of diagnostic certainty (or uncertainty) in a patient with carcinoma of the colon would be the finding of a constrictive lesion on barium enema and liver metastases on CT scan. If a serum CEA level were markedly elevated, the level of diagnostic confidence would be increased. Findings in a patient of a stony hard prostate on palpation, urinary retention, and x-ray findings of multiple, osteoblastic lesions of the pelvis and spine would be highly suggestive of carcinoma of the prostate. An increased serum level of prostatic acid phosphatase would extend the level of confidence even more. In none of these instances, however, would a "100% certain diagnosis" be possible without a biopsy.

There are analogies to modern clinical medical diagnosis in soft tissue paleopathology:

HISTORY. The site and provenience may be of definite help, such as when the remains are taken from a known burial site for lepers, or where there is a mass burial suggesting that many deaths occurred at the same time, as in a battle, famine, or rapidly progressing disease. Provenience may suggest certain diseases common at the time and place.

PHYSICAL EXAMINATION. This is essentially the gross autopsy of the mummy and the major source of our diagnostic possibilities in paleopathology. Lesions in the soft tissue may suggest carcinoma (versus tuberculosis, fungus, etc.) if the primary site can be located, as carcinoma arises by definition at an epithelial surface anatomically. The tumor, if primary, is usually but not necessarily concentrated there (breast, colon, prostate). Many carcinomas have a rather characteristic, natural course and way of spreading. Thus, carcinoma of the prostate invades locally and metastasizes most often to bone (80% of metastases), usually of the pelvis and vertebral column. Carcinoma of the colon proceeds to regional lymph nodes and 75% of other metastases are in the liver, though not commonly in bone (11.7 % of metastases). Carcinoma of the breast proceeds to the regional axillary nodes and when metastatic elsewhere, 70% of metastases are in bone (ribs, long bones, skull vertebrae), and lung (66% of metastases) (Del-Regato et al. 1985:687,542,866). In each instance, a new dimension over the x-ray appearance of the bony lesions alone is added by soft tissue examination. Prior x-ray or CT procedures may give an indication as to where attention should be directed during the gross autopsy. Primary malignant bone tumors are rare. When they occur, they often have a favored site. Metastases to bone, however, account for the great majority of cancers in bone and also have favored distributions (Abrams et al. 1950:77). The size of a bony lesion is not necessarily related to the primary site: a large defect with several smaller ones does not mean that the large one is, or is near to, the primary. A further caveat is that present-day statistical data for sites of bony metastases are not comparable.

RADIOLOGIC STUDIES. Since metastatic carcinoma to bone is by far the most common malignant tumor of bone, and since x-ray can detect lesions not visible from the surface, these studies form a very important role in the study of carcinoma. Bone is involved by metastases in up to 70% of malignancies in some series (Jaffe 1958:589–618), though it should not be expected that any value near this figure is attainable in ancient material for a variety of reasons, including incomplete skeletons. Further, ancient (untreated) cancer victims died earlier in the course (from intercurrent disease, then as now) and probably did not often manifest the fuller expression seen now (Ortner and Putschar 1981:365,366). In addition, series from medical literature include metastases to bone marrow and cancellous bone that have not yet involved the cortex. Breast, lung, and kidney currently make up a large percentage of primary lesions involving bone, but their relative frequency in ancient populations is absolutely unknown and cannot even be estimated.

The general advantage of radiologic procedures is their nondestructiveness. Whatever anatomic relations still intact after careful removal from the burial site, transportation, and so forth, can be recorded before the autopsy starts if practicable. This permits reevaluation of bone and soft tissue relations after completion of the autopsy. It also may suggest whether an autopsy would even be fruitful. If x-ray shows all of the organs to have undergone extensive degeneration and amalgamation, the yield in soft tissue studies is low. Cost and time need to be offset by potential gain.

X-RAY. This simple procedure is often available even in remote areas. When a lesion is osteoblastic (as most prostatic carcinomas are) x-ray is very helpful. Unfortunately osteoblastic lesions are less common than osteolytic ones, though they generally become visible earlier than osteolytic lesions of comparable size. The pattern of metastasis may suggest a primary site. Osteoblastic lesions involving the pelvis and lower vertebrae in an older male are highly suggestive of prostatic carcinoma. Breast tumor metastases to bone may be either osteoblastic or osteolytic, while those of colon carcinomas are usually osteolytic. An otherwise osteolytic lesion may appear osteoblastic if there is sufficient bony destruction in addition to adequate time for repair.

CT SCAN. This modality has markedly increased the accuracy of osteolytic bone lesion diagnosis and is considerably more sensitive. All else being equal CT scan can detect a smaller lesion than simple x-ray can. It is also of great help in soft tissue paleopathology (Pahl 1980:189; Wong 1981:101; Notman et al. 1986:95) since it both shows involvement of soft tissue adjacent to bone better than x-ray alone, and potentially also the relation of the tumor to body organs *in situ* in the mummy bundle before these relations have been destroyed by autopsy.

MAGNETIC RESONANCE IMAGING. MRI is of little value at present in paleopathology owing to the lack of moisture in mummified tissue and bone (Notman et al. 1986:95).

XERORADIOGRAPHY. This produces a positive picture (as opposed to the x-ray negative) and shows lesions of less density (such as osteolytic lesions) to better advantage. This proved helpful in finding a soft tissue pleural mass in a Peruvian mummy (Heinemann 1974).

Future

What can we hope for in the future to improve diagnostic accuracy in paleopathology?

LINES OF RESEARCH

Serologic procedures as they currently exist are not too promising. For example, CEA is nonspecific and subject to interfering substances under the best of circumstances. The basic problem lies in quantitation, as neither weight nor volume applies unless on a "per gram of tissue" basis. Even this basis is difficult to compare among individual specimens. Since most carcinomas mimic the function of the primary tissue in which they have arisen and carry on many of the same chemical processes, serologic diagnoses are dependent more on quantitative results than on qualitative ones. Most current tumor serologic procedures require careful handling and sometimes even rapid freezing of the specimen to prevent loss of antigen/antibody potency.

Histology still remains the best hope, and efforts to reconstitute dry tissue and make respectable slides should continue. Practically though, when a lung is concentrated and reduced to the thickness of a piece of paper it is hard to imagine that a meaningful slide could ever be made. Some experimental work has shown that this is nevertheless possible (Zimmerman 1977). Another potential avenue is that of histochemistry (special stains). Of these, immunoperoxidase is promising. Here a specific, peroxidase-labelled antibody is incubated with the tissue containing the suspected tumor antigen. The tumor portion then stains preferentially and more or less specifically. These labeled antibodies are commercially available and in use currently for breast, colon, and prostate carcinomas, as well as many others.

Endoscopy should be mentioned as a means of obtaining small amounts of tissue for histologic study (Notman et al. 1986:94).

SUGGESTIONS FOR FUTURE NOMENCLATURE

Provocative titles which cannot be well substantiated should not be used. For example "possible concomitant syphilis and leprosy in a population of cave dwellers," aside from piquing interest as to how anyone could make such a diagnosis, causes a serious problem. The article may become classified as syphilis, leprosy, or both, with the "possible" omitted. The key words will appear in the literature as such, become incorporated in the future statistics, and may even be used as a basis for the existence of a given disease at a given place and time.

Diagnoses should be uniform. For example the word "tumor" should be limited to neoplasia; "tumor-like process" should be used in other appropriate instances.

A diagnosis should be related somehow to the probability that it is correct, and the criteria used clearly stated. A differential diagnosis should be included, along with reasons, if any, why one particular diagnosis is favored over the others. Unless the diagnosis is almost certain, it should be omitted from the title and key reference words. If the diagnosis is based on statistical probability, it should be stated how the statistics were derived, that is, which populations are being compared.

VALUE OF MUSEUMS AND LARGE COLLECTIONS

While it is desirable to have assemblages containing large numbers of known examples of a given disease, the purpose is more that of teaching pathology than in comparing individuals. A large series of individual skeletons showing known metastatic breast carcinoma will look very much like a large series showing known metastatic lung carcinoma. An unknown individual case therefore cannot be fitted into either category with any degree of certainty. The severity of a disease itself is not a scalar quantity, since it reflects the presence of many factors. Its description can only be in relative terms: "A worse than B worse than C" (Medawar 1974:180).

Conclusions

Paleopathology is a young science and has developed few tools to date. An important application of paleopathology is that of assisting other scientists, such as paleoepidemiologists and paleodemographers. In addition, historians need data in assessing the presence and extent of disease, along with what effects it might have had upon its victims individually or as a population. A knowledge of the past distribution and cause of disease helps with the modern understanding of that disease. Matters of population resistance and susceptibility are reflected by the general health of the population.

On a social level, an understanding of diseases and their prevalence at a given time might help in a general understanding of one's past. Paleopathology offers a good opportunity for population studies as populations were relatively stable geographically over fairly long periods of time. Disease determination can also provide good markers for population studies, as has been the case with sickle cell hemoglobin. All of these benefits depend on accurate diagnosis, and it is in that direction that we should turn our attention.

Apart from the technical innovations and improvements, there is the matter of improving data. Most discoveries in science are based on empirical findings, at least initially. The potential for future studies, such as a change in immunity over the millennia, changing environmental factors and their effects, or changing customs with respect to perception of disease, depends on an accurate data base. An accurate data base depends in turn on large numbers of examples. We need larger numbers of examples, documented as accurately as possible as to disease and provenience, to make real progress.

Literature cited

Abrams, H.L., R. Spiro, and N. Goldstein. 1950. Metastases in Carcinoma. *Cancer*, 3:74–85.

Aegerter, E., and J.A. Kirkpatrick. 1968. *Orthopedic Diseases*. Philadelphia: W.D. Saunders.

Dastugue, J. 1986. The Borderlines between Normal and Pathologic in Paleopathology. Paper presented at the 6th European Meeting of the Paleopathology Association, Madrid, September 10th.

DelRegato, J.A., H.J. Spjut, and J.D. Cox. 1985. *Cancer: Diagnosis and Treatment*. St. Louis: C.V. Mosby.

Hackett, C.J. 1978. Treponematosis (Yaws and Treponarid) in Exhumed Australian Aboriginal Bones. *Records of the South Australian Museum*, 17:387–405.

Heinemann, S. 1974. Xeroradiography of a Pre-Columbian Mummy. *Journal of the American Medical Association*, 230:1256.

Jaffe, H.L. 1958. *Tumors and Tumerous Conditions of the Bones and Joints*. Philadelphia: Lea and Febiger.

Medawar, P.B. 1974. Some Follies of Quantification. *Hospital Practice*, July:179–180.

Notman, D.N.H., J. Tashjian, A.C. Aufderheide, O.W. Cass, O.C. Shane, T.H. Berquist, J.E. Gray, and E. Gedgaudas. 1986. Modern Imaging and Endoscopic Biopsy Techniques in Egyptian Mummies. *American Journal of Roentgenology*, 146:93–96.

Ortner, D.J., and W.G.J. Putschar. 1981. Identification of Pathological Conditions in Human Skeletal Remains. *Smithsonian Contributions to Anthropology*, 28. Washington, D.C.: Smithsonian Institution Press.

Pahl, W.M. 1980. Computed Tomography—A New Radiodiagnostical Technique Applied to Medico-Archaeological Investigation of Egyptian Mummies. *Ossa*, 7:189–198.

———. 1986. Tumors of Bone and Soft Tissue in Ancient Egypt and Nubia: A Synopsis of Detected Cases. *International Journal of Physical Anthropology*, 3:267–276.

Stout, A.P., and R. Lattes. 1967. *Tumors of the Soft Tissues*. Washington, D.C.: Armed Forces Institute of Pathology.

Wells, C. 1967. Pseudopathology. In D. Brothwell and A.T. Sandison, eds., *Diseases in Antiquity*, 5–19. Springfield, Ill.: Charles C Thomas.

Wong, P.A. 1981. Computed Tomography in Paleopathology: Technique and Case Study. *American Journal of Physical Anthropology*, 55:101–110.

Zimmerman, M.R. 1977. An Experimental Study of Mummification Pertinent to the Antiquity of Cancer. *Cancer*, 40:1358–1362.

———. 1981. A Possible Histiocytoma in an Egyptian Mummy. *Archives of Dermatology*, 117:364–365.

SUMMARY OF AUDIENCE DISCUSSION: Estimates of the modern frequency of bone involvement in cancer and other diseases are based on autopsy, clinical, and radiological data. Examination of skeletal tissues in routine autopsies is limited, while clinical attention with consequent radiological study is usually dependent on patient complaint of pain and dysfunction. Thus, the available bone involvement patterns are highly selective and no doubt overlook many lesions which would be evident in osteological examinations if such bodies were skeletonized. Total body x-rays of, say, 100 patients dying of each of several common cancers and subsequently autopsied could provide a database against which patterns of bone involvement found in ancient skeletons could be measured.

A recent study demonstrated that the lytic lesions of bone found in erosive arthropathies actually accommodate fat-producing cells capable of providing lubricating fat in joints that have lost their cartilage. Histological studies of the content of lytic lesions found in an autopsy study might reveal similar "surprises."

Dental Disease

Interpretations of general problems in amelogenesis

Albert A. Dahlberg

The microscopic and gross morphological details of tooth parts have been recognized by paleontologists and biologists for a long time and have been used as differentiation markers in classification; many of them have been the significant points of taxonomical considerations. However, these same important properties of tooth part details are a frequent source of confusion because of the complexities of the amelogenesis and calcification process.

The simplest forms of enamel hypoplasias are those that are the result of nutritional deficiencies or pathological conditions accompanied by high fevers or infections, and are easily recognized by the matching occurrence of a circle or ring of pits and adjacent defects on the enamel. This dyscrasia occurs at the level of calcification that was in progress at the time of the insult, differing in position from tooth to tooth as they were developing. The key to this category of enamel developmental display is the timing-location factor. Chemical hypoplasia from high fluorosis or circumstances such as phosphate deficiencies may be similarly expressed. Other individual or combined effects involving enamel formation, rate of growth, and variations in morphological size and proportions have been recorded in the literature. Crenulated enamel surfaces on some of the fossils and similarly disturbed surfaces of later forms having disorganized patterns of enamel have, at times, invited erroneous diagnoses. This is particularly true in regard to some of the mulberry molars of the congenital syphilis triad (Bradlaw 1953).

The biochemical control of the phylogenetic architecture of occlusal cusp patterns is sometimes disturbed to the extent of losing almost all the identity of the surface. A long list of authors hold differing views and explanations of these occurrences (Weidenreich 1937; Sarnat and Schour 1941–1942; Dahlberg 1960; Taylor 1978; White 1978; Goodman and Armelagos 1980; White and Johanson 1982; Pindborg 1982; Mayhall and Saunders 1986; Cook and Buikstra 1979; Tobias 1986) and present a variety of perspectives in many directions.

Explanations of ontogenetic and genetic interactions have been useful in explaining some of the literature reports. Evocative interpretations can be made from observations of the terminal growth of such structures as the deflecting wrinkle of the metaconid ridge of the lower molar occlusal surfaces, the enamel extensions between the roots of the lower molars, enamel occlusal pearls, enamel molar cusp bulges (Kirveskari et al. 1972), and the structural relationship of the outer crown patterns compared to the endocast topography of those same teeth (Korenhof 1960, 1982). Both Korenhof (1960) and Tobias (1986) discussed the crests, wrinkles and crenulations of the outer crown surface and the relationship to the dentin endocast, some of which perhaps may be due to the developmental events of the enamel growth and direction taken within the enamel organ itself. Similar comments can be made relating to the enamel bulges of the buccal cusps described by Kirveskari et al. (1972). Events in the growth processes are vulnerable to environmental input and interact with the sequence and nature of induction responses (Kollar 1975).

Growth of enamel occurs in directions away from the first initiated ameloblasts, a site which becomes known as the tips of the cusps, with the mitotic divisions favoring the direction of the adjacent cusps. The mitotic divisions follow the same pattern of succession in all the lower teeth: from the protoconid to metaconid, to hypoconid, to entoconid, to hypoconulid, and lastly to the accessory sixth, seventh and other peripheral cusplets. A similar succession of initiation, division, further division, and calcification occurs in the maxillary teeth from paracone to protocone, to metacone, to hypocone, to Carabelli's cusp, and so on. In all the teeth the ameloblast activity changes course to conform with the tooth architecture as the growth activity approaches the line angle limits of the buccal, lingual, mesial, or distal outer surfaces. How this occurs is assumed to be a biochemical problem such as Kollar (1975) and others have discussed in tissue culture demonstrations.

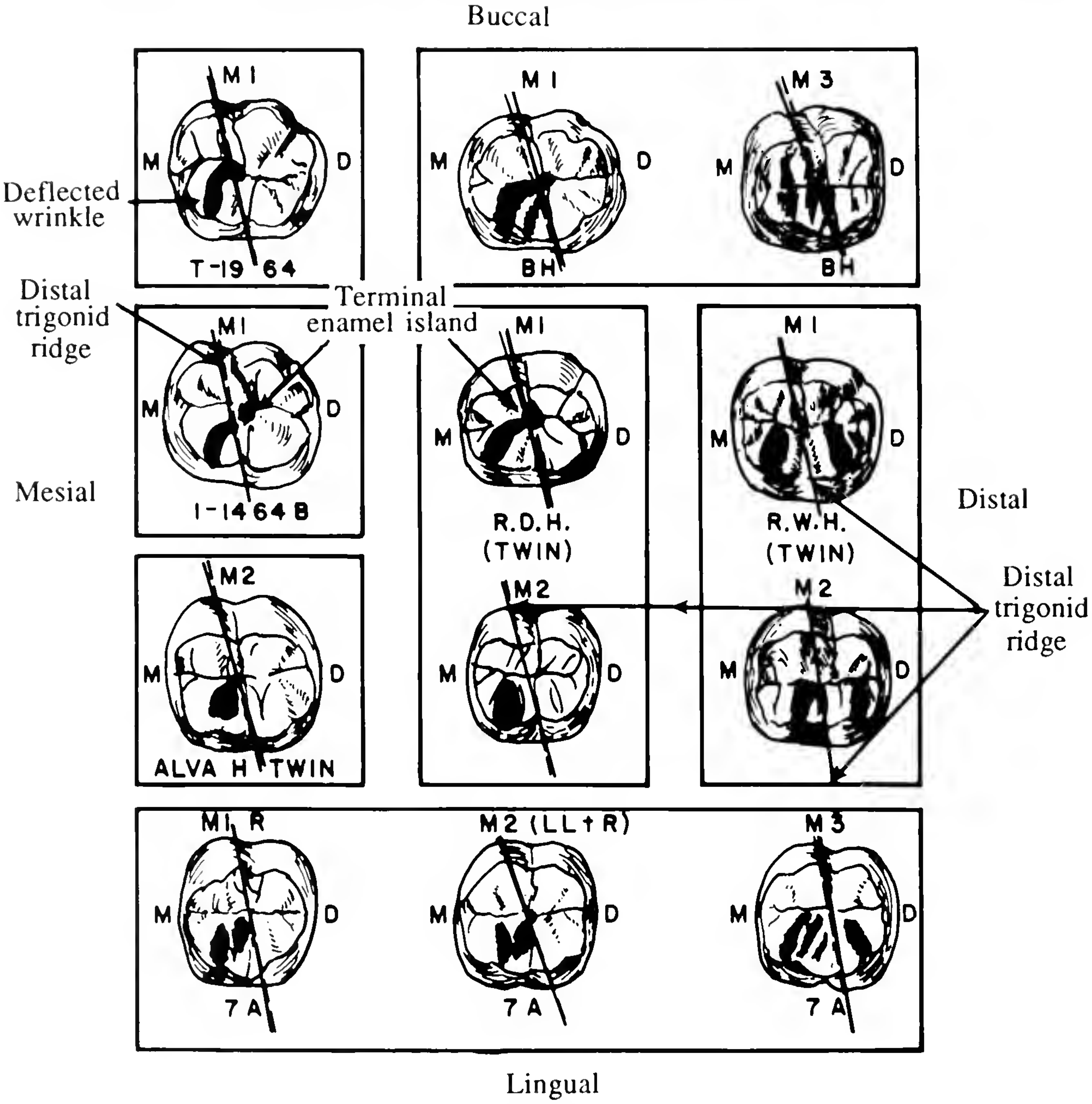

Buccal
Deflected wrinkle
Distal trigonid ridge
Terminal enamel island
Mesial
Distal
Distal trigonid ridge
Lingual
M1
M
D
T-19 64
M1
M
D
BH
M3
M
D
BH
M1
M
D
1-1464 B
M1
M
D
R.D.H. (TWIN)
M1
M
D
R.W.H. (TWIN)
M2
M
D
ALVA H TWIN
M2
M
D
M2
M
D
M1 R
M
D
7 A
M2 (LL+R)
M
D
7 A
M3
M
D
7 A

FIGURE 1. Twelve occlusal views of lower right molar teeth; heavy black indicates various patterns of "deflecting wrinkle" on buccal aspects of metaconids (mesiolingual cusps). Deflecting wrinkle seen as a heavy crest (T-19 64) extending buccally from occlusal tip toward protoconid and deflecting distally as it reaches central fossa. Deflecting part generally continuous in deciduous second molar and permanent first molar, but can be separated from a terminal island of enamel. Frequently, separation is incomplete and occurs in vicinity of midpoint of distal trigonid ridge, as seen in sketches BH, 1-1464B, R.D.H., and M2 of 7A. (Figure drawing courtesy of Marcia Bakry, Smithsonian Institution, Department of Anthropology, Washington, D.C.)

Cusp growth, for the most part, terminates in the central part of the occlusal surface, at grooves, pits, or at the advancing termini of the adjacent cusps. The size and shapes of these cusps are determined to a considerable extent by the rate and extent of growth of the neighboring cusps. In the lower molars, for example, the grooves adapt to form a Y or X configuration, with other minor adjustments. In most instances in man, the lower molars present a larger prominence of the central crest on the buccal surface of the metaconids (mesiolingual cusps). This crest has been the subject of interest to odontologists because of its size and its tendency to deflect distally as it projects buccaly. This structure has been labeled the "deflecting wrinkle" (T-19 64 and 1-1464B of Figure 1), and varies in frequency and details of form and direction in any given dentition, from dm2 to M1 to M2 and to M3, following the succession patterns of Butler's Field Concept of development (Butler 1939; Dahlberg 1945).

The deflecting wrinkle variations are seen in other sketches of Figure 1 as thickened and shorter (in M2 of Alva H, in M1 of R.W.H.) and as divided (in M1, M2, and M3 of 7A). The wrinkle of M1 of R.D.H. shows a beginning of separation but not a complete severance of the termini of the wrinkle, as on the M1s of 1-1464B. The deflection of the terminal end is almost always confined to the M1s, and is very rare on the stubby, heavier or divided M2s. Additional to the areas or pathways not open for the full expression of the developing wrinkle, there is a deterrent of phylogenetic origin, the distal trigonid ridge or crest, a distal ridge remnant of the original trigonid (three-cusp tooth formed in evolution) (Korenhof 1978). Zoubov (1973) reports a high frequency of distal trigonid ridges in lower molars of some eastern populations of the Soviet Union, and Hanihara (1966) records high frequencies of deflecting wrinkles in mongoloid-complex dentitions. However, a substantial number of terminal ends of this wrinkle are physically separated as an enamel island, especially on M2s and occasionally on M1s, as seen on M1 of BH, M1 of 1-1464B and M1 of R.D.H. of Figure 1. These terminal enamel islands are similar to the islands seen in the crenulations and wrinkles of some of the fossils discussed by Weidenreich (1937) and Tobias (1986). Also, a similar suggestion of some morphological changes induced by spirochetes in the enamel islands and some other conformations (Bradlaw 1953; Sarnat and Schour 1941–1942) can be seen in Figure 2 (Dahlberg 1986; Mann et al. 1990).

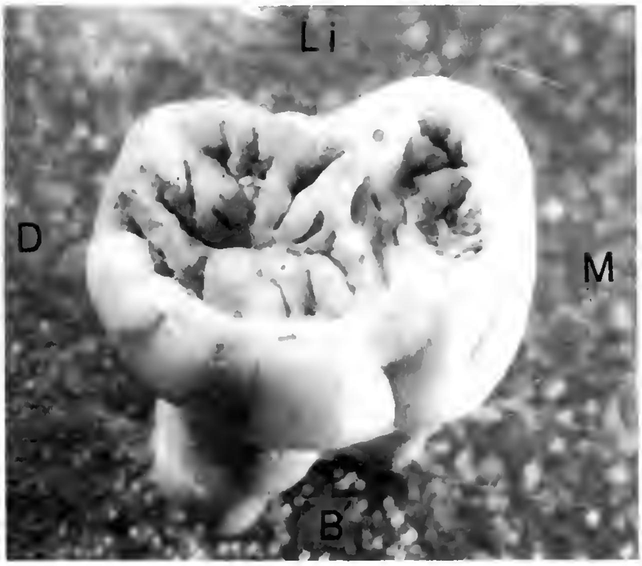


FIGURE 2. Bucco-lingual view of deciduous lower right second molar showing many disorganized groupings of enamel islands on occlusal surface. Buccal pit and protostylid seen on buccal surface. Several divided islands visible on this tooth from 5-year-old male American Indian, William and Mary collection of the Chickahominy tribe. (Mann et al. 1990).

The major problem in understanding and interpreting amelogenesis arises from the fact that several separate processes are going on at one time. These are involved in a recording sequence of calcification and growth of other centers of similar activity. Also, there is an interplay of environment, genes, and interactions with delays or arrests of procedures. This works out well for the normal programming of tooth formation. However, it compounds the picture for observers (Boyde 1970) who already are faced with myriads of performances of the polygenic tooth production scheme or background. The introduction of insults or new factors of major potential in themselves do not cause the same effects on such things as islands of enamel, other tissues, or time- and chemically regulated interactions. Not all morphologically similar productions of enamel rings, pits, or surface enamel island arrays and patterns have the same causation. Those affecting the ontogenetic sequences can be very misleading. Many such cases have been considered the systemic effects of congenital syphilis or other factors, without sufficient substantiation. A simple example would be the occurrence of only one instance of a mulberry molar in a large population. Other causes can be possible triggers for such metabolic events. Pits and defects are good markers, but need additional corroborating evidence for proper evaluation.

Literature cited

Boyde, E.A. 1970. Surface of the Enamel in Human Hypoplastic Teeth. *Archives of Oral Biology,* 15:897–898.

Bradlaw, R.V. 1953. The Dental Stigmata of Prenatal Syphilis. *Oral Surgery, Oral Medicine, Oral Pathology,* 6:147–158.

Butler, P. 1939. Studies of the Mammalian Dentition. Differentiation of the Postcanine Dentition. *Proceedings of the Zoological Society of London,* 109:1–36.

Cook, D.C., and J.E. Buikstra. 1979. Health and Differential Survival in Prehistoric Populations. Presented Dental Defects. *American Journal of Physical Anthropology,* 51:649–664.

Dahlberg, A.A. 1945. Changing Dentition of Man. *American Dental Association,* 32:676–690.

———. 1960. The Olduvai Giant Hominid Tooth. *Nature,* 188: 962.

———. 1986. *Variation in the Dentition.* Paris: Memoires of Meseed l'Homme.

Goodman, A.H., and G.J. Armelagos. 1980. Chronology of Enamel Hypoplastic Growth Disruptions in Prehistoric, Historic and Modern Population. *American Journal of Physical Anthropology,* 52:232.

Hanihara, K. 1966. Mongoloid Dental Complex in the Deciduous Dentition. *Journal of the Anthropological Society of Nippon,* 74:61–72.

Kirveskari, P.E., B. Heslegard, and A.A. Dahlberg. 1972. Bulging of the Lingual Aspects of Buccal cusps in Posterior Teeth of Skolt Lapps from Northern Finland. *Journal of Dental Research,* 51, 5(2):1513.

Kollar, E.J. 1975. Gene-Environment Interactions during Tooth Development. *Clinics of North America,* 19:141–146.

Korenhof, C.A.W. 1960. *Morphogenetical Aspects of the Human Upper Molar: A Comparative Study.* Utrecht, Netherlands: Uitgeversmaatschappij, Neerlandia.

———. 1978. Remnants of the Triconial Crests in Medieval Molars of Man of Java in Development, Function and Evolution of Teeth. In P.M. Butler and K.A. Joysey, eds., *Development, Function and Evolution of Teeth,* 157–170. 4th International Symposium on Dental Morphology, Cambridge, England, 1974. London: Academic Press.

———. 1982. Evolutionary Trends of the Inner Enamel Anatomy of Deciduous Molars from Sangrivien (Java, Indonesia). In B. Kurten, ed., *Teeth—Form, Function and Evolution,* 350–365. New York: Columbia University Press.

Mann, R.W., A.A. Dahlberg, and T.D. Stewart. 1990. Anomalous Morphologic Formation of Deciduous and Permanent Teeth in a 5-Year Old 15th-Century Child: A Variant of the Ekman-Westborg-Julin Syndrome. *Oral Surgery Oral Medicine Oral Pathology,* 70:90–94.

Mayhall, J.T., and S.R. Saunders. 1986. Dimensional and Discrete Dental Trait Asymmetry Relationship. *American Journal of Physical Anthropology,* 69:403–411.

Pindborg, J.J. 1982. Etiology of Developmental Enamel Defects Not Related to Fluorosis. *International Dental Journal,* 32:123–134.

Sarnat, B.G., and I. Schour. 1941–1942. Enamel Hypoplasia in Relation to Systemic Disease. *Journal of the American Dental Association,* 28:1989–2000, 67–75.

Taylor, R.M.S. 1978. *Variation in Morphology of Teeth: Anthropologic and Forensic Aspects.* Springfield, Ill.: Charles C Thomas.

Tobias, T. 1986. Enamel Crenulations and Wrinkles on Hominid Molar Teeth. *Anthropos,* 23:63–77. Brno, Czechoslovakia.

Weidenreich, F. 1937. Dentition of *Sinanthropus pekinensis. Paleontologica Sinica,* 10(1):1–180.

White, T.D. 1978. Early Hominid Enamel Hypoplasia. *American Journal of Physical Anthropology,* 49:79–83.

White, T.D., and D.C. Johanson. 1982. Pliocene Hominid Mandibles from the Hadar Formation, Ethiopia: 1974–1977 Collections. *American Journal of Physical Anthropology,* 57:501–544.

Zoubov, A. 1973. *Etnicheskaya ondostologia.* Moscow: Nauka.

Two developmental anomalies of the teeth and resulting secondary pathosis

Gábor Kocsis and Antonia Marcsik

Among dental developmental anomalies are alterations of shape and size. These include, among others, the dens invaginatus and the palato-gingival groove. The latter is considered a form of invagination (Lee et al. 1968:18; Walker and Glyn Jones 1983:34). The clinical importance of both is that they promote the formation of periodontal and periapical pathosis (Lee et al. 1968:16; Simon et al. 1971:823; Aboyans and Ghaemmaghami 1976:65).

The purpose of our study was to determine the prevalence of these two developmental abnormalities. We examined the difference in frequencies between archeological material and recent samples. In archeological material from the seventh–eighth centuries A.D. we investigated their simultaneous occurrence and their prevalence on one or two sides, their sex dimorphism, as well as their incidences in the teeth of Europoid and Mongoloid skulls. We also present pathologic phenomena induced by the palato-gingival groove.

Dens invaginatus (invaginated odontome or dens in dente) is a well-known developmental anomaly occurring as a result of invagination of the tooth germ before its calcification, creating a pouchlike defect. It may be found in any tooth germ but the maxillary incisors are most frequently affected (Aboyans and Ghaemmaghami 1976:63). According to Hallett (1953:496) the invagination is over eight times more frequent in the upper lateral incisors than in the central ones.

There are two types of invagination: coronal and radicular. It has also become customary to divide coronal invaginations into superficial and deep ones (Schulze 1970:108–109). Hallett's classification (1953:492) is most widely accepted: (1) A definite cleft is formed in the palatal enamel at the cervical level. This cleft runs vertically and there is no expansion or dilatation; (2) the invagination extends toward the pulp chamber and a definite pit is formed in the cingulum; (3) the invagination extends deeply into the pulp chamber and is dilated; (4) the invagination apparently occludes the whole of the coronal pulp chamber and may extend beyond the amelo-cemental junction level.

Schranz's study (1987:78) is also remarkable. He differentiated the following invaginations on the palatal surface of the upper incisors: "blind pit" (foramen caecum dentis), imperfect invagination (pouch or funnel-like tooth), perfect invagination (dens in dente, dens invaginatus) and palatal invagination.

The pathogenesis of the coronal invagination is not entirely known. Schulze (1970:110–111) wrote about a single, active proliferation or passive retardation of a circumscribed area of epithelium, or that possibly it is formed by union of adjacent teeth. Grahnen et al. (1959:131–132) drew attention to the fact that dens invaginatus is genetically determined. Photographs and x-rays of dens invaginatus are shown in Figures 1–3.

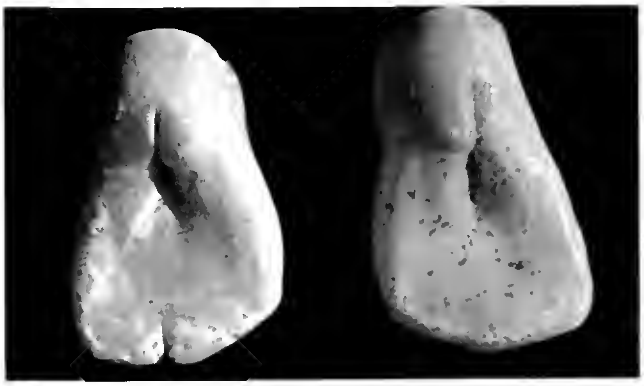


FIGURE 1. Palatal invagination in maxillary lateral incisors. Szegvár-Oromdülö, Grave 83, 7th century.

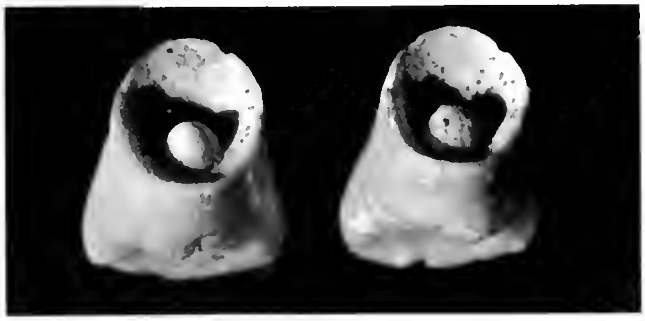


FIGURE 2. Palatal invagination from apical direction in maxillary lateral incisors. Szegvár-Oromdülö, Grave 83, 7th century.

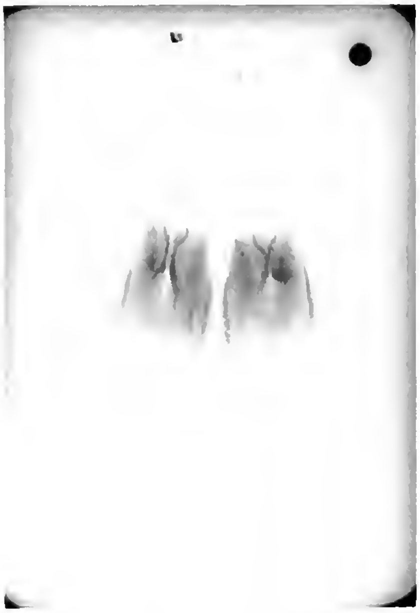


FIGURE 3. X-rays of palatal invagination (Hallett's type III) in maxillary lateral incisors. Szegvár-Oromdülő, Grave 83, 7th century.

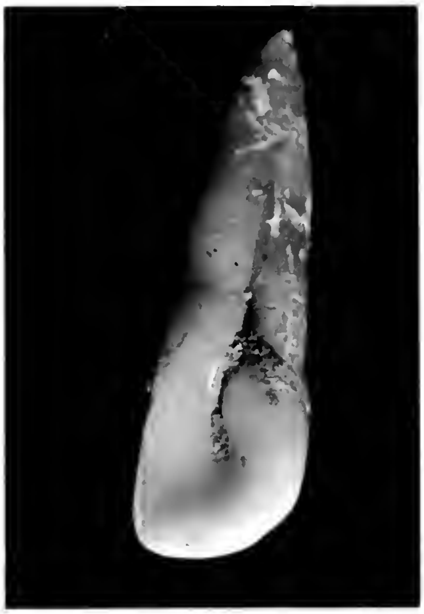


FIGURE 4. Palato-gingival groove in maxillary right lateral incisor. Szegvár-Oromdülő, Grave 128, 7th century.

FIGURE 5. X-rays of palato-gingival groove in maxillary right lateral incisor. Szegvár-Oromdülő, Grave 128, 7th century.

The palato-gingival (Figures 4,5) groove begins in the central fossa area, principally in the crown of upper incisors, crosses over the cingulum, and continues apically down the root for varying lengths (Simon et al. 1971:823). This groove occurs mostly in the upper lateral incisors, rarely in the upper central incisors (Withers et al. 1981:42). The phenomenon has been termed in the literature as radicular anomaly, disto-lingual groove, and radicular lingual groove (Withers et al. 1981:41).

It is important from the point of view of differential diagnosis that the palato-gingival groove should be differentiated from the vertical fracture and crack of the root (August 1978:1038).

The direct cause of the groove is unknown. Mechanical effects on the tooth germ are considered as probable causes, which can also induce dental invagination (Lee et al. 1968:18; Everett and Kramer 1972:357). On the basis of the investigations of Bruszt (1950:536–537) it is also possible that the cause of the malformation is fusion of teeth, as in the case of dens invaginatus.

Material and methods

We studied the prevalence of the two developmental anomalies on x-rays of 500 upper lateral incisors of unknown persons and on 803 upper incisors of 229 persons at the Department of Dentistry and Oral Surgery (Albert Szent-Györgyi University Medical School). We also investigated 796 upper incisors of 282 skulls from the seventh–eighth centuries A.D. and 84 upper incisors of 36 skulls from the Neolithic age from the paleoanthropological collection of the Department of Anthropology, Attila József University, Szeged. The period of the seventh–eighth centuries is the so-called "Avar Period." One of the components of the Avar tribes, which originated from the Altaic region, is of Mongoloid character, but in the population of the Avar Period of course, the Europoid types predominate (85.5%; Lipták 1983:48–49,85–89). On the skulls from the seventh–eighth centuries we could examine the association of the two developmental anomalies with the Europoid and Mongoloid traits.

The classical, anthropological elaboration of the larger part of the studied paleoanthropological series has already been reported in the literature (Lipták and Marcsik 1966; Lipták and Vámos 1969; Kőhegyi and Marcsik 1971; Vámos 1973; Lipták and Varga 1974; Farkas 1975; Lipták and Marcsik 1976).

The study of the two developmental anomalies was performed by direct observation and evaluation of x-rays. The results were analyzed using two-dimensional contingency tables.

Results and discussion

FREQUENCY

In our recent material, on the basis of the x-rays of 500 upper lateral incisors, we found the invagination in dilated form (Hallett's type III) in 12 cases, which translates to a frequency of 2.4% (Table 1). The incidence of dens invaginatus was

TABLE 1. The prevalence of foramen cecum, dens invaginatus and palato-gingival groove in recent and paleoanthropological material

	Neolithic		*7-8th centuries*		*Recent*	
No. individuals	36		282		-	
foramen cecum	7		92 (32.6%)			
No. individuals	36		282		-	
dens invaginatus	2		15 (5.3%)			
No. individuals	36		282		229	
palato-gingival groove	5		52 (18.4%)		7 (3.1%)	
	11+21	*12+22*	*11+21*	*12+22*	*11+21*	*12+22*
No. teeth	38	46	374	422	-	-
foramen cecum	-	9	9 (2.4%)	137 (32.5%)	-	-
No. teeth	38	46	374	422	-	500
dens invaginatus	-	2	-	22 (5.2%)	-	12 (2.4%)
No. teeth	38	46	374	422	401	402
palato-gingival groove	1	4	7 (1.9%)	52 (12.3%)	1	6 (1.5%)

NOTE: 11 = maxillary right central incisor; 21 = maxillary left central incisor; 12 = maxillary right lateral incisor; 22 = maxillary left lateral incisor.

TABLE 2. Simultaneous occurrence of anomalies

	Lateral incisor (% FC+PG)	*Central incisor*
No. FC	137 (13.9%)	9
No. PG	52 (36.5%)	7
FC+PG	19	1
No. DI	22	0
DI+PG	2	0

NOTE: FC, foramen cecum; PG, palato-gingival groove; DI, dens invaginatus.

reported as 0.25–5.1% by Pindborg (1970:58–59), as 3–10% by Schulze (1970:108), and the foramen cecum was present in 9.6% in the material of Fujiki et al. (1974:344).

From the seventh–eighth centuries the prevalence of the dilated invagination was 5.2%, that of the foramen cecum was 32.5% in the upper lateral incisors. In the material of the Neolithic age, 2 of the 46 teeth had invagination, and 9 teeth a foramen cecum (Table 1). Brabant and Sahly (1962:304) reported invagination in 4.2% from the Neolithic material, and Árkövy (1904:23) found the foramen cecum in 39.3% from the Avar Period.

The incidence of the palato-gingival groove in the upper lateral incisors of our recent material was 1.5% (Table 1). It was 4.4% in the material of Withers et al. (1981:42), and 2.9% in Everett and Kramer (1972:352).

The palato-gingival groove was present in 12.3% of the teeth from the seventh–eighth centuries, and in 4 of the 46 teeth from the Neolithic age (Table 1). Brabant reported 6.3–14.2% from the Neolithic age (1969:448), and 5.5% from the Gallo-Roman ages (1973:250).

The simultaneous occurrence of the two developmental anomalies, their symmetry-asymmetry relation, sex dimorphism and racial connection could be studied only in teeth of skulls from the seventh–eighth centuries.

SIMULTANEOUS OCCURRENCE

Several studies have shown (Lee et al. 1968:18; Everett and Kramer 1972:352; Walker and Glyn Jones 1983:33–34) that the occurrence of the palato-gingival groove is associated with the dens invaginatus. In Table 2 we give the number of foramen cecum and invagination cases occurring together with the groove, compared with the total number of malformations.

TABLE 3. Symmetry-asymmetry relation in maxillary incisors

	Symmetry 11+21	*Asymmetry 11 or 21*	*Symmetry 12+22*	*Asymmetry 12 or 22*
No. skulls	140		159	
FC	3	3	51	9
DI	-	-	7	5
PG	0	6	7	20

NOTE: For tooth notation, see note to Table 1. For abbreviations, see Table 2.

TABLE 4. Sexual dimorphism

	Male		*Female*	
No. skulls	105		93	
	Central incisors	Lateral incisors	Central incisors	Lateral incisors
FC	1	25 (23.8%)	3	28 (30.0%)
DI	-	4	-	5
PG	3	16 (15.2%)	2	14 (15.1%)

NOTE: For abbreviations, see Table 2.

SYMMETRY-ASYMMETRY RELATION

The number of skulls having tooth malformations is shown in Table 3. This number is lower than that in Table 1. For symmetry-asymmetry relationships, we could investigate only those skulls in which both central incisors and/or both lateral incisors were present. The presence of foramen cecum was more frequent on both sides simultaneously, while in the case of dens invaginatus no such difference could be identified. Végh's results (1974:370) are similar to ours. Amos (1955:33) found 22 symmetric and 29 asymmetric cases, while Grahnen et al. (1959:122) observed 35 bilateral and 23 unilateral ones.

The palato-gingival groove was more frequently asymmetric in our material. Withers et al. (1981:42) reported a similar relationship but stated that the difference is minimal and therefore unimportant.

SEXUAL DIMORPHISM

In the case of skulls of known sex we investigated the sex dimorphism of the malformations (Table 4). The frequency of dens invaginatus and that of the palatal-gingival groove is the same in each of the two sexes. The foramen cecum in the teeth of females is more frequent, but no statistically significant difference was found between the two sexes. In Amos's work (1955:32) the dens invaginatus was more frequent in females, but Grahnen et al. (1959:122) found it more commonly in males (boys). Frequency of the palato-gingival groove was also the same in the two sexes, both in our material and in the studies of Withers et al. (1981:42).

DIFFERENCES BETWEEN EUROPOIDS AND MONGOLOIDS

Table 5 gives the occurrences of the malformations in skulls of Europoid and Mongoloid types. The foramen cecum occurs three times more frequently in the Europoid skulls than in the Mongoloid ones, but the difference is not significant statistically. At the same time, the frequency of the palato-gingival groove was higher in the Mongoloid skulls, and the difference was statistically significant ($p < .01$ from chi square tests). According to Amos (1955:31), the dens invaginatus cannot be observed in Negroes. Lee et al. (1968:18) called attention to the relationship of the palato-gingival groove with the races, although they do not consider it to be a racial variation. Withers et al. (1981:42) found the frequency of the palato-gingival groove was 9.1 percent in Caucasians and 5.2 percent in blacks, but this difference was not significant.

PATHOLOGIC CONSEQUENCES OF THE TWO DEVELOPMENTAL ANOMALIES

Schranz (1987:78) described the developmental anomalies of tooth shape from the point of the caries, pointing out that the different invaginations are accessory factors in the formation of the dental caries. Fujiki et al. (1974:344) reported caries in 15 (6.5%) and periapical lesions in 7 (3%) out of 230 teeth having dens invaginatus. According to Aboyans and Ghaemmaghami (1976:63), palatal cingulum pits were frequently attacked by caries, sometimes causing periapical pathosis, with or without clinical signs. In permanent maxillary lateral incisors showing minor palatal invaginations, pulp death occurs in the absence of clinical or histological evidence of caries (Stephens 1953:502; Kramer 1953:504–506).

TABLE 5. Differences between Europoids and Mongoloids

	Europoid	*Mongoloid*
No. skulls	119	75
FC	33 (27.7%)	6 (8.0%)
DI	4 (3.4%)	
PG	16 (13.4%)	27 (36.0%)
PG + FC	6 (5.0%)	6 (8.0%)

NOTE: For abbreviations, see Table 2.

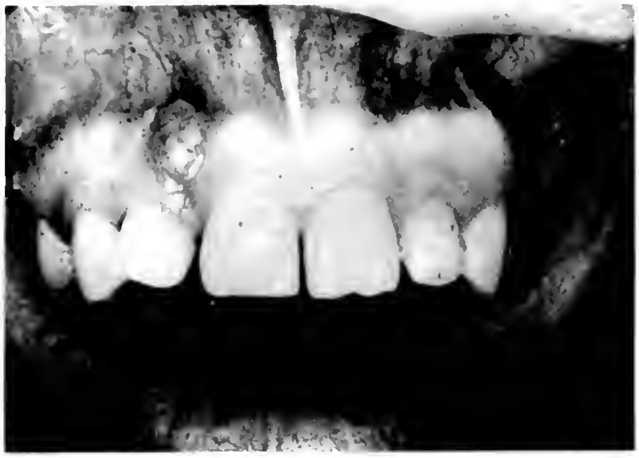


FIGURE 6. Periodontal abscess (clinical case).

Creaven (1975:79–80) also mentioned periapical lesions caused by dens invaginatus in teeth where the pulp was viable.

According to Everett (1968:287–288), Prichard was the first to state that the palato-gingival grooves on maxillary incisor teeth are a predisposing factor to localized severe periodontal destruction. However, Brabant et al. (1958:87) called attention to the fact that as early as 1949, Dechaume reported that the palato-gingival groove may be responsible for the infection of the palatal gingiva and for inducing retrograde apical pulpitis.

In a deep palato-gingival groove the surface is covered by bacterial plaque and infected granulation tissue (Lee et al. 1968:17). On the basis of the investigation of Withers et al. (1981:42) the Plaque, Gingival and Periodontal Disease Index is statistically significant in the case of this anomaly. Tooth mobility is also increased, but there is no connection between the mobility and the groove. The pathomechanism of the gingivitis predisposed by this anomaly is similar to that known in the case of enamel formation (Simon et al. 1971:824). The periodontal lesion, developing locally, leads to recession, abscess formation and, from time to time, pulp death. In the case of deep grooves the pulp disease is in a direct or indirect association with the formation of the periapical pathosis (Lee et al. 1968:16; Simon et al. 1971:824; Aboyans and Ghaemmaghami 1976:63; Walker and Glyn Jones 1983:33).

CASE REPORT

A 19-year-old female patient was presented at the Department of Dentistry and Oral Surgery with a swelling on the area of the roots of the upper right incisors (Figure 6). At the clinical examination the teeth in question showed a vital dental reaction, recession formation and periodontal abscess vestibularly, and a groove vestibularly and palatinally. Radiography revealed that the upper right lateral incisor had two

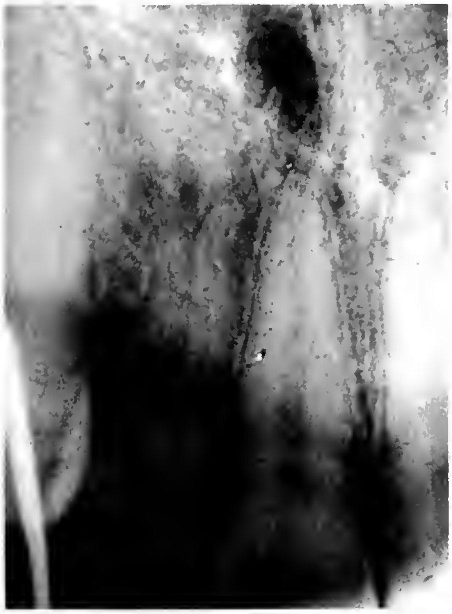


FIGURE 7. X-rays of two roots with palato-gingival groove in maxillary right lateral incisor (clinical case).

roots mesiodistally (Figure 7), and the upper left lateral incisor had a palato-gingival groove distally (Figure 8). The periodontal abscess formed by developmental anomaly was removed surgically. Three months later periapical pathosis was observed. The pulp in the distal chamber was necrotic (Figure 9). The tooth was treated endodontally and provided with root filling (Figure 10).

Investigating paleoanthropological material we have observed bone defects (granuloma and cyst cavities) in the area of incisors. These teeth showed less abrasion and no caries (Figure 11). In such cases we can think of trauma as an etiological factor, but we have to think of developmental anomalies too, as predisposing factors to the above-mentioned pathological processes. On the other hand, we have also seen some localized alveolar bone resorption with palato-gingival groove (Figure 12); presumably the palato-gingival groove caused the localized bone resorption in this case.

Summary

Two developmental anomalies, dens invaginatus and palato-gingival groove, both occurring on upper incisors, are reviewed. The purpose of our investigation was mainly to determine the prevalence of these anomalies in maxillary lateral incisors and to draw attention to the fact that these conditions may cause periodontic and pulpal pathosis.

Neither in our recent nor in our paleoanthropological material do the frequencies of the dens invaginatus and of the palato-gingival groove differ from the literature data. It is

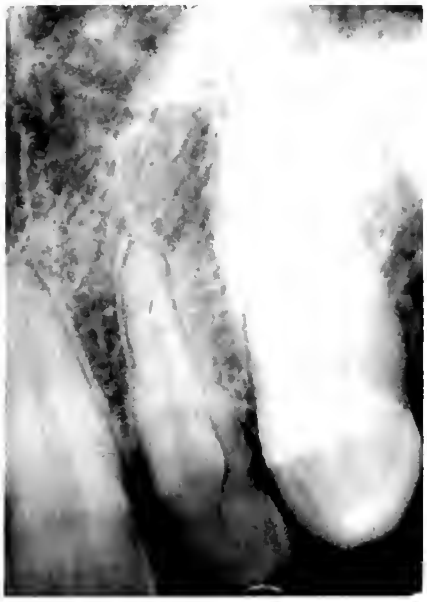


FIGURE 8. X-rays of palato-gingival groove in maxillary left lateral incisor (clinical case).

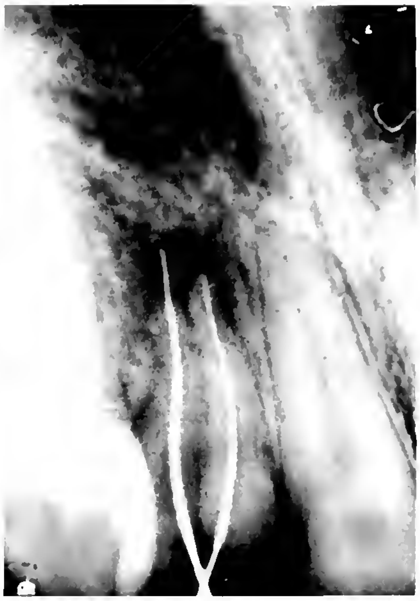


FIGURE 9. Root chambers are probed in maxillary right lateral incisor, osteoporosis at apex (clinical case).

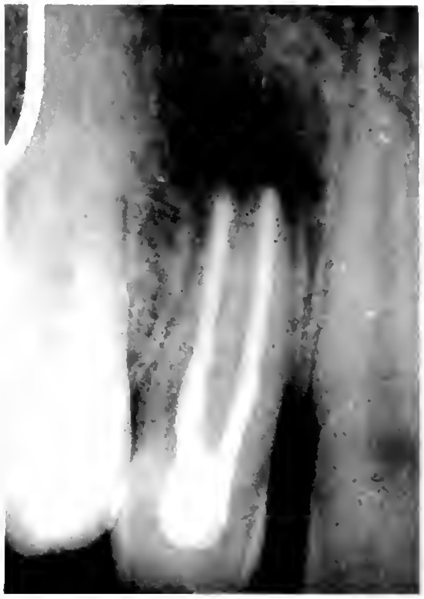


FIGURE 10. X-rays of roots after filling and resection in maxillary right lateral incisor (clinical case).

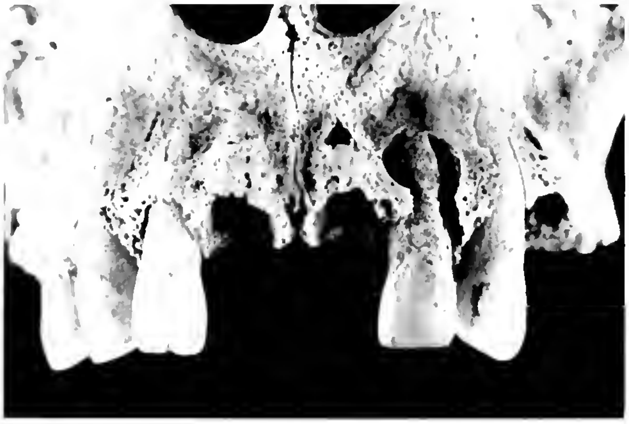


FIGURE 11. Bone defect by periostitis and fistula cavity formation in area of upper left lateral incisor. Székkutas-Kápolnaülö, Grave 61, 8th century.

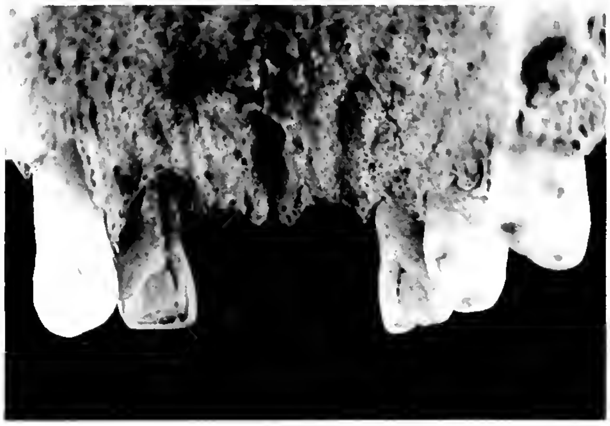


FIGURE 12. Palato-gingival groove in maxillary left lateral incisor and localized alveolar bone resorption. Székkutas-Kápolnaülö, Grave 61, 8th century.

remarkable that in the paleoanthropological material the frequency is higher as compared with the recent material, which may be the result of the closed communities of people in early times. It is worthy to note that the palato-gingival groove was more frequent in teeth of skulls with Mongoloid character.

Sexual dimorphism, symmetry and asymmetry relationships, and simultaneous occurrence of the two developmental anomalies show no important differences.

Based on clinical reports and our clinical case herein reported, caries and the localized bone lesions in paleoanthropological material might have been caused by developmental anomalies (dens invaginatus and palato-gingival groove).

Literature cited

Aboyans, V., and A. Ghaemmaghami. 1976. Two Developmental Anomalies as Potential Causes of Periapical Pathosis. *Journal of Oral Medicine*, 31:63–66.

Amos, E.R. 1955. Incidence of the Small Dens in Dente. *Journal of the American Dental Association*, 51:31–33.

Arkovy, J. 1904. Die Bedeutung des Diverticulum Tomes-Zsigmondyi, des Cingulum an den oberen lateralen Schneidezähnen und des Foramen coecum molarium (Milleri) in phylogenetischer Beziehung. *sterreich-ungarische Vierteljahrschrift für Zahnheilkunde*, 20:13–34.

August, D.S. 1978. The Radicular Lingual Groove, an Overlooked Differential Diagnosis. *Journal of the American Dental Association*, 96:1037–1039.

Brabant, H. 1969. Observations sur les Dents des Populations Megalithiques d'Europe Occidentale. *Bulletin du Groupement International pour la Recherche Scientifique en Stomatologie*, 12:429–460.

______. 1973. Etude Odontologique des Restes Humains Découverts dans la Nécropole Gallo-Romaine et Mérovingienne de Dieue (Meuse) en France. *Bulletin du Groupement International pour la Recherche Scientifique en Stomatologie*, 16:239–261.

Brabant, H., L. Klees, and R.J. Werelds. 1958. *Anomalies Mutilations et Tumeurs des Dents Humaines*. Paris: Julien Prélat, Liege, Sciences et Lettres.

Brabant, H., and A. Sahly. 1962. La Paleostomatologie en Belgique et en France. *Acta Stomatologica Belgica*, 59:285–355.

Bruszt, P. 1950. Fber die Entstehung des "Dens in dente." *Schweizerische Monatsschrift für Zahnheilkunde*, 60:534–542.

Creaven, J. 1975. Dens Invaginatus Type Malformation without Pulpal Involvement. *Journal of Endodontics*, 1:79–80.

Everett, F.G. 1968. Predisposing Factor to Localized Periodontal Disease. *British Dental Journal*, 125:287–288.

Everett, F.G., and G.M. Kramer. 1972. The Disto-Lingual Groove in the Maxillary Incisors. A Periodontal Hazard. *Journal of Periodontology*, 43:352–361.

Farkas, Gy. 1975. A Délalföld öskorának paleoantropológiája. (Palaoanthropology of Prehistoric Age in the Hungarian Great Plain). Ph.D. dissertation.

Fujiki, Y., N. Tamaki, K. Kawahara, and M. Nabae. 1974. Clinical and Radiographic Observation of Dens Invaginatus. In Y. Fujiki, H. Fuchihata, and T. Wada, eds., *Proceedings of the 3d International Congress of Maxillofacial Radiology*, 343–345. Kyoto, Japan.

Grahnén, H., B. Lindahl, and K. Omnell. 1959. Dens Invaginatus I. A Clinical Roentgenological and Genetical Study of Permanent Upper Lateral Incisors. *Odontologisk Revy*, 10:115–137.

Hallett, G.E.M. 1953. The Incidence, Nature, and Clinical Significance of Palatal Invaginations in the Maxillary Incisor Teeth. *Proceedings of the Royal Society of Medicine*, 46:491–499.

Köhegyi, M., and A. Marcsik. 1971. The Avar-Age cemetery at Sükösd. *Acta Antiqua et Archeologica*, 14:87–94.

Kramer, R.H. 1953. The Pathology of Pulp Death in Non-Carious Maxillary Incisors with Minor Palatal Invaginations. *Proceedings of the Royal Society of Medicine*, 46:503–507.

Lee, K.W., E.C. Lee, and K.Y. Poon. 1968. Palato-Gingival Grooves in Maxillary Incisors. *British Dental Jour.*, 124:14–18.

Lipták, P. 1983. *Avars and Ancient Hungarians*. Budapest: Publishing House of the Hungarian Academy of Sciences.

Lipták, P., and A. Marcsik. 1966. Szeged-Kundomb avarkori népességének embertani vizsgálata. (Die anthropologische Untersuchung des Gräberfeldes Szeged-Kundomb aus der Awarenperiode). *Anthropologiai Közlemények*, 10:13–56.

______. 1976. A Madaras-Téglavetö avar temető csontvázmaradványainak embertani jellemzése. (Anthropologische Charakteristik der Skelettreste aus dem awarischen Gräberfeld bei Madaras- Téglavetö). *Cumania*, 4:115–140.

Lipták, P., and K. Vámos. 1969. A "Fehértó-A" megnevezésü avarkori temetö csontvázanyagának embertani vizsgálata. (Anthropologische Untersuchung des Skelettmaterials des awarenzeitlichen Gräberfeldes "Fehértó-A"). *Anthropologiai Közlemények*, 13:3–30.

Lipták, P., and I. Varga. 1974. Charakterisierung des anthropologischen Materials des awarischen Gräberfeldes von Kunszallas. *Móra Ferenc Muzeum Évkönyve*, (1971) 2:71–85.

Pindborg, J.J. 1970. *Pathology of the Dental Hard Tissues*. Copenhagen: Munksgaard.

Schranz, D. 1987. A fogkorona alakjának fejlödési rendellenességei és a kárieszhajlam. (Development Anomalies and Caries Tendency of the Tooth Crown Shape). *Fogorvosi Szemle*, 80:77–79.

Schulze, C. 1970. Developmental Abnormalities of the Teeth and Jaws. Anomalies of the Tooth Shape and Size. In F.R. Gorlin and H.M. Goldman, eds., *Thoma's Oral Pathology*, 96–112. St. Louis: C.V. Mosby.

Simon, H.S., D.H. Glick, and A.L. Frank. 1971. Predictable Endodontic and Periodontic Failures as a Result of Radicular Anomalies. *Oral Surgery, Oral Medicine, Oral Pathology*, 31:823–826.

Stephens, R. 1953. The Diagnosis, Clinical Significance and Treatment of Minor Palatal Invaginations in Maxillary Incisors. *Proceedings of the Royal Society of Medicine*, 46:499–503.

Vámos, K. 1973. "Szeged-Makkoserdö" avarkori népességének embertani vizsgálata. (Die anthropologische Untersuchung der awarenzeitlichen Bevölkerung von "Szeged-Makkoserdö). *Anthropologiai Közlemények*, 17:29–39.

Végh, T. 1974. A fogak koronai invaginatiojának röntgenvizsgálatához. (Röntgenuntersuchung der Invagination der Zahnkronen). *Fogorvosi Szemle*, 68:370–373.

Walker, R.T., and J.C. Glyn Jones. 1983. The Palato-Gingival Groove and Pulpitis: A Case Report. *International Endodontic Journal*, 16:33–34.

Withers, J.A., M.A. Brunsvold, W.J. Killoy, and A.J. Rahe. 1981. The Relationship of Palato-Gingival Grooves to Localized Periodontal Disease. *Journal of Periodontology*, 52:41–44.

SUMMARY OF AUDIENCE DISCUSSION: These examples were found among 300 specimens from about A.D. 800. The palato-gingival groove is higher in Mongoloids than Europoids. Certain traits may have dental survival value. For example, an elongated pulp cavity has the opportunity of filling in more dentine, delaying the time when attrition reaches the pulp and thus prolonging the tooth life. The dental survival value of other traits is not always obvious and deserves further study. For example, a buccal pit almost invariably becomes carious. On the other hand, animals (including man) surviving in an environment challenging many of their protective mechanisms frequently demonstrate involvement of more than one system in the reestablishment of equilibrium following insult. Studies directed at identification of such "backup" systems could enhance our understanding of past processes.

Stress, adaptation, and enamel developmental defects

Alan H. Goodman

In recent years we have witnessed an impressive growth in research on the evolution of disease. While the ultimate aims of paleopathologists were once merely to describe the occurrence of lesions and infirmities in relationship to the time and location of those long dead, most researchers now strive to understand the significance of their data for the lives of the individuals and populations under investigation. Temporal changes in the pattern of health and disease are now seen as important keys to understanding human evolution, and the distribution of disease over ecological and socioeconomic landscapes is increasingly seen as critical to understanding the human condition, past and present.

With these broader and deeper questions on disease, evolution, and the human condition comes an increased need to understand the context of our paleopathological data. In order to understand who dies and who gets sick, and the impact of these events on individuals and populations, one must understand the available ecological and social resources, population sizes, social stratification, systems of power, resource distribution and the like. At present, I hope it will suffice to say that the need for context—not just ecological and demographic, but broadly social, political, and economic—forces paleopathologists to pay greater attention to colleagues in anthropology and archeology who share the goal of knowing something of the daily lives and struggles of past populations.

With the asking of questions about the effects of lesions on peoples' lives comes a well-placed need to understand firmly the biobehavioral significance of the underlying condition causing these scars. How chronic and long lasting might the underlying illness have been? How often does a bony scar signify a condition which leads to reduced work capacity, reproductive potential, or length of life?

The potential disparity in interpretation of the adaptive significance of lesions has recently been highlighted by Ortner (1979:596, and this volume) with particular reference to infectious lesions. Most researchers have assumed that lesions generally are signs of stress and, by extension, problems in adaptation (c.f. Goodman, Martin et al. 1984). Their questions have revolved around understanding the degree of stress and adaptive constraint signified by lesions. Ortner, on the other hand, has challenged the basic assumption that those with lesions are less biologically adapted. He proposes that skeletal lesions may be indications of the individual's ability to "rally" from insults. This proposition in based on the notion that lesions are found paradoxically on individuals that survived with the underlying perturbation. These bony scars, therefore, are signs of survival.

The above variability in perspective is particularly critical to the analysis and interpretation of enamel hypoplasias and other developmental defects of dental enamel. Linear or chronologic enamel hypoplasias, deficiencies in enamel thickness resulting from a temporary cessation in enamel matrix apposition (Sarnat and Schour 1941; Goodman, Armelagos, and Rose 1980), have long been considered to reflect nonspecific physiological stress during tooth crown formation (Sarnat and Schour 1941; Kreshover 1960). Hypoplasias are, without denial, a marker of a condition which the individual survived.

Based on clinical and experimental data (see recent reviews by Cutress and Suckling 1982, Jontel and Linde 1986, and Pindborg 1982) paleopathologists have frequently and increasingly interpreted those with hypoplasias to be more stressed and less well adapted than those without hypoplasias. For example, Swardstedt (1966) has shown that the frequency of enamel hypoplasias decreases significantly from a slave class to a land-owning class in Westerhus, a Medieval Swedish population. These data are consistent with increased cultural buffering of the stresses of disease and malnutrition in families owning land. In a recent volume on health changes at the origins of agriculture (Cohen and Armelagos 1984), 15 of 19 (79%) regional case studies reported on changes in frequency of hypoplasias from gathering-hunting to agriculture. All participants considered increased hypoplasia frequencies to signify increased stress. Ortner's comments notwithstanding, hypoplasias have been reified as prime indicators of stress in paleopathological studies.

Are we justified in considering hypoplasias to be general indicators of stress? If so, what is the degree of disruption and limit to function signified by hypoplasias? The purpose of this paper is to begin to consider the meaning of hypoplasias in an adaptive context. I will do this by reviewing two recent studies of enamel hypoplasias in relationship to other indicators of stress. One of these studies involves a prehistoric population. If hypoplasias are indications of increased stress and adaptive constraint then we should expect to see them associated with other indicators of stress. Thus, in the prehistoric study we focus on the relationship between enamel hypoplasias and life expectancy, a universally accepted measure of stress or failure to rally from insults (Goodman, Martin et al. 1984). Finally, because of the limitations of understanding the adaptive context of dental defects in prehistoric studies, I have begun to examine the pattern of defects in modern, mild-to-moderately malnourished children. In the subsequent section I report on the association of defects with socioeconomic status and stature.

Enamel hypoplasias and longevity at Dickson Mounds

In order to evaluate the long-term consequences of stress during childhood we have examined the relationship between enamel hypoplasias of permanent teeth and mean age at death at Dickson Mounds (Goodman and Armelagos 1988). The sample consists of 111 adults and adolescents from the Dickson Mounds, a multicomponent habitation-burial complex located in Lewiston, Illinois (Goodman, Armelagos, and Rose 1980; Goodman, Lallo et al. 1984). The mounds are associated with three cultural horizons: Late Woodland (LW), Mississippian Acculturated Late Woodland (MALW), and an early and late Middle Mississippian (MM) (Harn 1980). During the Late Woodland period (ca. A.D. 900–1050) the area was occupied by a small (75–125) and semi-sedentary hunting and gathering population with seasonal camp sites and an economy directed toward the use of a wide spectrum of local fauna and flora. During the MALW (ca. A.D. 1050–1175) local populations began to come under the influence of Mississippian cultures farther to the south. During the MM period (ca. A.D. 1175–1250) the Mississippianization of local populations became complete with the culmination of trends toward extended and intensified trade networks, increased population density, size, and sedentarism, and greater reliance on maize agriculture (Harn 1978).

These changes have been associated with an increase in nutritional and infectious pathological conditions and a decrease in life expectancy (Goodman, Lallo et al. 1984). Porotic hyperostosis typically found on the flat bones of the skull and the superior half the orbits, an indication of iron deficiency anemia, is four times as prevalent among MM subadults as compared to LW subadults (64% to 16%) (Lallo et al. 1977). Periosteal infections in subadults increase from 27% in the LW to 81% in the MM (Lallo et al. 1978), and the frequency of enamel hypoplasias doubles in adults and adolescents (Goodman, Armelagos, and Rose 1980). Life expectancy is lower at all age intervals in the MM when compared to the combined LW and MALW samples (Moore et al. 1975).

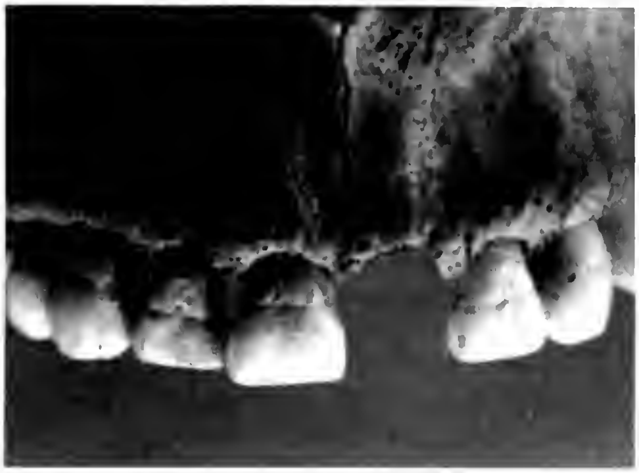


FIGURE 1. Chronologic enamel hypoplasias (stress-hypoplasias) on anterior, maxillary permanent teeth. Hypoplasias are observable on right central incisor, right and left lateral incisors, and right and left canines. All hypoplasias occur between 3.0 and 3.5 years developmental age and appear to be the result of the same systemic disruption (stress).

Enamel hypoplasias were recorded on all permanent teeth except third molars as circumferential lines, bands, or pitting of decreased enamel thickness (Goodman, Armelagos, and Rose 1980) (Figure 1). The distance of the hypoplasia from the cemento-enamel junction was measured to 0.1 mm using a thin-tipped caliper. This distance measure was converted to the individual's dental age when the disruption occurred, based on the developmental standard of Massler et al. (1941).

Each half-year period between birth and 7.0 years was rated as either stress-positive, stress-negative, or undetermined, based on the following criteria: (1) a half-year period was rated as stress-positive if there were two or more teeth with hypoplasias occurring during the half-year period; (2) a half-year period was rated as stress-negative if less than two hypoplasias occurred during this period and at least four tooth crowns were available for scoring whose development includes this period; (3) a half-year period was rated as undetermined if one or no hypoplasias occurred during this period and there were less than four tooth crowns available for scoring whose development included this period. By using this method, a chronology of stress by half-year periods was developed for each individual from birth to 7 years of age (Goodman, Armelagos, and Rose 1980, 1984).

This study is based on the evidence for stress between 3.5 and 7.0 years of age. The extensive dental attrition limits ability to observe the enamel record of stress from birth to 3.5

TABLE 1. Comparison of mean ages at death for individuals by cultural horizon and number of hypoplasias-stress periods at 3.5–7.0 years developmental age (see text)

	Sample size	*Mean*	*S.D.*	*1-way ANOVA (F-ratio)*	*A priori contrasts (T-values)*		
					A vs B	*A vs C*	*A vs B + C*
Late Woodland	20	33.0	11.5	.35	.55	--	.55
0 Hypoplasias (A)	11	31.6	10.4				
1 Hypoplasia (B)	9	34.7	13.0				
2-3 Hypoplasias (C)	-	--	--				
M. A. L. W.	45	33.3	13.4	1.44	1.22	1.53	1.69
0 Hypoplasias (A)	22	36.6	12.8				
1 Hypoplasia (B)	14	31.1	14.7				
2-3 Hypoplasias (C)	9	28.6	11.7				
Middle Mississippian	46	31.6	11.22	6.52**	2.25*	3.50***	3.52***
0 Hypoplasias (A)	17	37.5	9.0				
1 Hypoplasia (B)	22	30.2	11.0				
2-3 Hypoplasias (C)	7	21.8	8.7				
TOTAL SAMPLE	111	32.5	12.1	4.99**	1.84^{+}	3.04**	3.08**
0 Hypoplasias (A)	50	35.8	10.1				
1 Hypoplasia (B)	45	31.4	12.7				
2-3 Hypoplasias (C)	16	25.6	10.8				

NOTES: + = 2-tailed $p \leq .10$; * = 2-tailed $p \leq .05$; ** = 2-tailed $p \leq .01$; *** = 2-tailed $p \leq .001$

years (Goodman, Armelagos, and Rose 1984). However, all individuals yielded a complete record of stress-hypoplasias for the seven half-year periods from 3.5 to 7.0 years.

The mean age at death of individuals in the three cultural horizons and in the total sample is compared for those individuals with no defects-stress period (total n = 50, 45.0%), one defect-stress period (total n = 45, 40.5%), and two or more defects-stress periods (two defects: total n = 14, 12.6%; three defects: total n = 2, 1.8%) (Table 1, Figure 2). The lowest overall frequency of defects is found in the LW group. All individuals in this sample have either one or no hypoplasias-stress periods between 3.5 and 7.0 years. Individuals with one hypoplasia-stress period have a slightly greater mean age at death (34.7 years) than individuals with no hypoplasias-stress periods (31.6 years). This difference, however, is not statistically significant (F-ratio = .35).

This association between hypoplasias-stress periods and longevity is reversed during the MALW periods. The mean age at death of individuals without hypoplasias-stress periods is 36.6 years, or 5.5 years greater than those with one hypoplasia-stress period (31.1 years) and 8.0 years greater than those with two or more stress periods (Figure 2).

This inverse association between stress periods and mean age at death is most pronounced during the Middle Mississippian. The mean age at death of individuals without hypoplasias-stress periods is 37.5 years, or 7.3 years longer than those with one hypoplasia-stress period and 15.7 years longer than those with two or more hypoplasias-stress periods (Figure 2). A one-way ANOVA, testing for the statistical significance of differences in ages at death among hypoplasia-stress period groups in the MM (Nie et al. 1975) yielded an F-ratio of 6.52 (Table 1; $p < .01$).

Furthermore, I tested for thz specificity of differences between groups in the MM with a series of a priori contrasts (Table 1). These provide a comparison of the mean age at death in group A (no stress periods) with: (1) group B (one stress period), (2) group C (two or three stress periods), and (3) group B + C combined (one or more stress periods). For the MM group, all a priori contrasts yielded statistically significant results at the .05 level of confidence. The most significant differences are found in comparing individuals without any stress periods with those with one or more or two or more stress periods ($t = 3.50$ and 3.52, $p < .001$ and $< .001$).

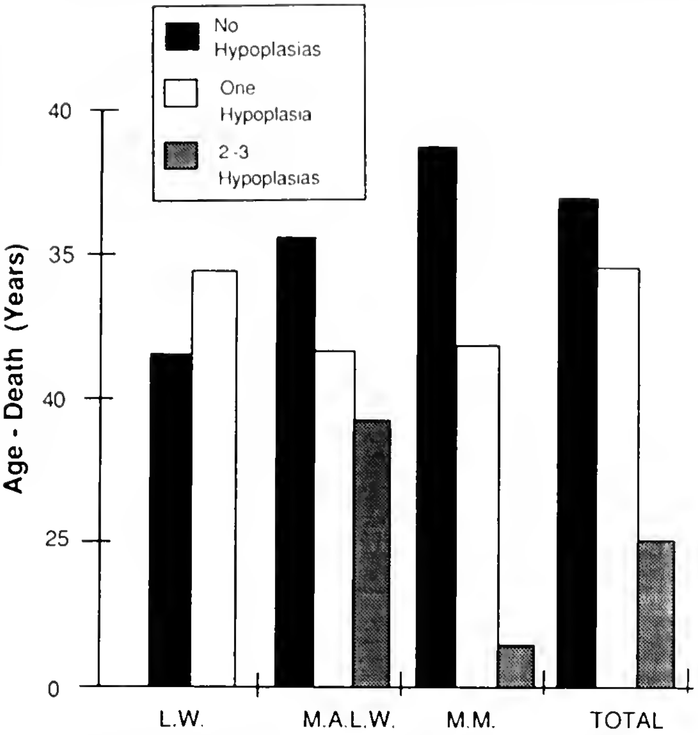


FIGURE 2. Mean ages at death of Dickson Mounds adolescents/adults by number of hypoplasias-stress periods between 3.5 and 7.0 years developmental age. LW = Late Woodland, MALW = Mississippian Acculturated Late Woodland, MM = Middle Mississippian.

Finally, there is a significant decrease in longevity with childhood stress periods in the total sample (Table 1, Figure 2). The mean age at death of individuals without hypoplasias-stress periods is 35.8 years, or 4.4 years greater than for those with one hypoplasia-stress period (31.4 years) and 10.2 years greater than for those with two or more hypoplasia-stress periods (25.6 years). The ANOVA yields a significant F-ratio (4.99, $p < .01$) and all a priori contrasts are significant at the .10 level of confidence. Again, the most significant contrasts are between the no-stress period group and the one or more stress period and two or more stress period groups ($t = 3.04$ and 3.08, $p < .01$, $< .01$).

At least three prior studies of archeological groups have also noted an inverse relationship between the occurrence of enamel hypoplasias and longevity. White (1978) assessed hypoplasias on permanent maxillary first molars from South African Plio-Pleistocene Australopithecines (ca. 1.5–3.0 M.Y.B.P.). He noted that individuals with maxillary first molar hypoplasias from the Swartkrans site ($n = 6$) had "lower-than-expected" ages at death. These individuals die between 4 and 13 years of age, while individuals with non-hypoplastic first molars ($n = 110$) die between 8 and 31 years of age. Goodman and Armelagos (1988) have calculated the mean age at death of individuals in these groups as 7.8 and 19.6 years, respectively. While White's study suffers from a restricted sample size and lack of precision in assigning ages of death to fragmentary paleontological materials, the data nonetheless demonstrate a decrease in fitness associated with hypoplasias.

Cook and Buikstra (1979) compared the mean age at death of infants and children with and without postnatal defects on deciduous tooth crowns from Middle and Late Woodland skeletal samples from Illinois. They conclude that postnatal dental defects are associated with decreased longevity during both the Middle and Late Woodland periods.

Rose and co-workers (1978) studied areas of disturbed enamel formation visible in thin-section (Wilson bands) in Middle Woodland, Mississippian Acculturated Late Woodland, and Middle Mississippian samples from Illinois. They found that individuals with Wilson bands died at an earlier mean age at death in all samples. Overall, we calculated that the average age at death of the 21 individuals with Wilson bands to be 26.7 years, or 15.4 years less than the average age of the 66 individuals without Wilson bands (42.1 years). In summary, these studies support our contention that dental developmental defects may predispose to an earlier age at death.

There are at least three processes which may account for the association between childhood stress and decreased life expectancy. First, these data may result from differential lifelong patterns of biological susceptibility to physiological disruptions and their adverse effects. An increased susceptibility to stress may be causative of both an increased frequency of childhood hypoplasias and an earlier age at death. That is, individuals who are ill during childhood may continue to fall ill as adults due to a "weak constitution." Second, individuals who were exposed to and survived a period of severe childhood stress may suffer a loss in ability to respond to other stresses. In a sense, these individuals are "biologically damaged" by the early stress. The wear and tear of stresses during development may render them less fit to respond to and survive subsequent stresses. For example, suboptimal early nutrition has been proposed as a mechanism for later immune dysfunction (Chandra 1975; Miler 1982). Third, these data may result from differential lifelong patterns of behaviorally and culturally based exposure to stressors. An increased lifelong potential for exposure to stressors may be causative of both an increased frequency of childhood stress and earlier ages at death. Barker and Osmond (1986a,b) have shown an association between poor nutrition and respiratory infections in childhood and coronary disease and chronic bronchitis in adulthood. They propose that this relationship is mainly due to social conditions present in childhood, which are likely to persist into adult life.

It is not possible to rule out any of these processes. All may contribute to the associations which we have observed. However, the wide variation in the degree of association between stress and longevity supports the view that the association is not solely a function of biological factors, since these samples appear to be genetically continuous (Cohen 1974; Goodman, Lallo et al. 1984). Furthermore, the greatest difference between stressed and nonstressed group mean ages at death occurs in the MM period. Since this is also the horizon in which status differences are likely to be greatest (Rothschild 1979), these data suggest that lifelong differences in social status, and therefore differential cultural buffering from stress, may be important. Unfortunately, it is difficult to assess cultural buffering in archeological populations. Goodman, Rothschild, and Armelagos (1983) have tested to see if differences in type of grave offering, an indicator of status differences, might explain the association between stress and longevity. While individuals with no grave goods are more likely to have multiple hypoplasias (17.6%) as compared to individuals with no nonutilitarian offerings (8.7%), their relationship does not explain the association between hypoplasias and age at death. The inability of grave goods to explain the observed association, however, is probably more a function of their uncertainty as indicators of status than of the insignificance of status differences in the etiology of childhood stress and adult mortality.

Although these data have largely been unable to distinguish among these mechanisms, it is suggested that a cultural buffering hypothesis is most congruent with the pattern of associations. Furthermore, this hypothesis is not exclusive of a "biological damage" mechanism. Low status during childhood may promote disease and undernutrition which leaves individuals less able to rally from future insults. Most importantly however, the data are strongly in support of the notion that enamel defects are indicators of stress and that this stress is highly meaningful in terms of life expectancy.

Socioeconomic and nutritional correlates of dental defects in contemporary Mexican children

The second study is part of the Collaborative Research Support Project, or CRSP. The purpose of CRSP is to understand better the sequence and severity of functional effects of mild-to-moderate undernutrition, the most prevalent but least understood form of malnutrition. The research site is in the Tamascalsingo region of the Mexican highlands, about 170 km northwest of Mexico City. The immediate area, the Solis Valley, includes 13 villages, 5 of which were included in the study. Living conditions are "typical" for a rural community in the Third World. Roads are unpaved and there is little sanitation. Houses are small, usually consisting of from one to three rooms. The dominant food item is tortillas, though this traditional food is rapidly giving way to pasta and coca-cola. Relevant to this study, the fluoride content of the central well's water is low, 0.20 ppm.

The dental data are from 7–9-year-old children, targeted for inclusion in the CRSP study, and their school age siblings. The sample includes 300 children, ages 5 to 15, with slightly more females than males. Defects were scored on anterior teeth by developmental zones approximating sixths of tooth crowns from the incisal to the cervical border. Classification followed the FDI index of developmental defects of dental enamel (1982). Five types of defects were found from most to least prevalent: white opacities, generally mild hypoplastic lines, hypoplastic pits, yellow opacities, and missing enamel. In the permanent incisors most hypoplasias developed in the second to fourth zones, suggesting a peak age at formation of from about 8 to 30 months. Hypocalcifications or opacities tended to occur in the first to third zones.

The following analysis includes only permanent dentition defects, divided into two general classes—enamel hypocalcification, or all opacities, and enamel hypoplasias, or all deficiencies in thickness. Patterns of association are presented between enamel defect and socioeconomic status and height-for-age. Height percentiles were calculated relative to National Center for Health Statistics Standards (Hamill et al. 1977). Socioeconomic status is based on household characteristics and material wealth (Allen et al. 1987).

Mean height percentile for those with no defect, with hypocalcifications and with hypoplasias, by anterior tooth, are presented in Figure 3. As is to be expected for a rural Mexican sample, the mean percentile height-for-age is generally low, around 10–15%.

The pattern for incisors is of highest mean height percentiles associated with opacities and the lowest with hypoplasias. For example, the hypoplasia group mean percentile for the upper lateral incisor (3.1%) is significantly less than either of the other groups (no defects = 12.3%; hypocalcifications = 17.9%). While this pattern varies slightly for the other teeth it is clear that enamel hypocalcifications are not associated with a reduced height-for-age and enamel hypoplasias are so associated.

No significant mean differences in the socioeconomic scores (SES) are observed for those with and without hypocalcifications (Figure 4). However, a consistent decrease in SES is seen in those with hypoplasias versus those without defects. This decrease in hypoplasia mean is significant at the .05 level or greater for the upper central incisor, the lower lateral incisor, the lower canines and the total. In sum, the SES data are similar to the above stature data in that decreased SES is not associated with hypocalcification, but is associated with the appearance of hypoplasias.

The lack of association between enamel hypocalcifcations and SES or nutritional status suggests that these defects are not related to general conditions of life. This is consistent

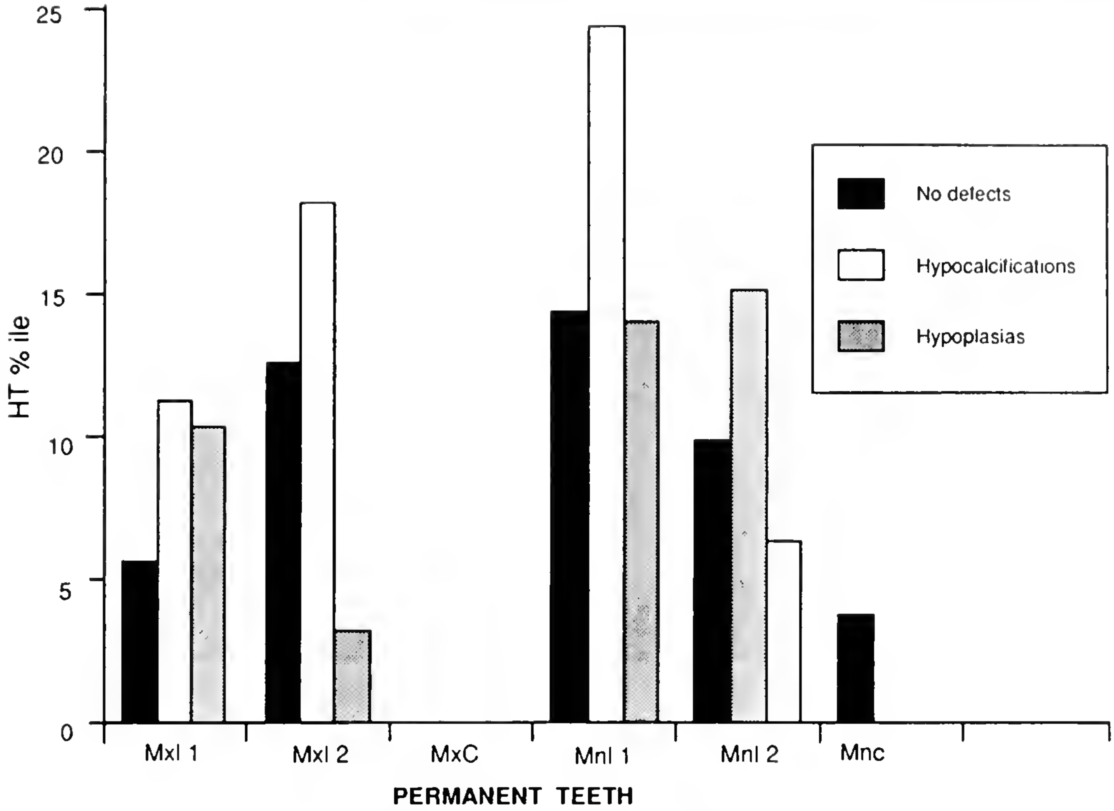


FIGURE 3. Mean height percentile (HT%ile) for those with no defects, hypocalcification, or hypoplasias for the six anterior permanent teeth.

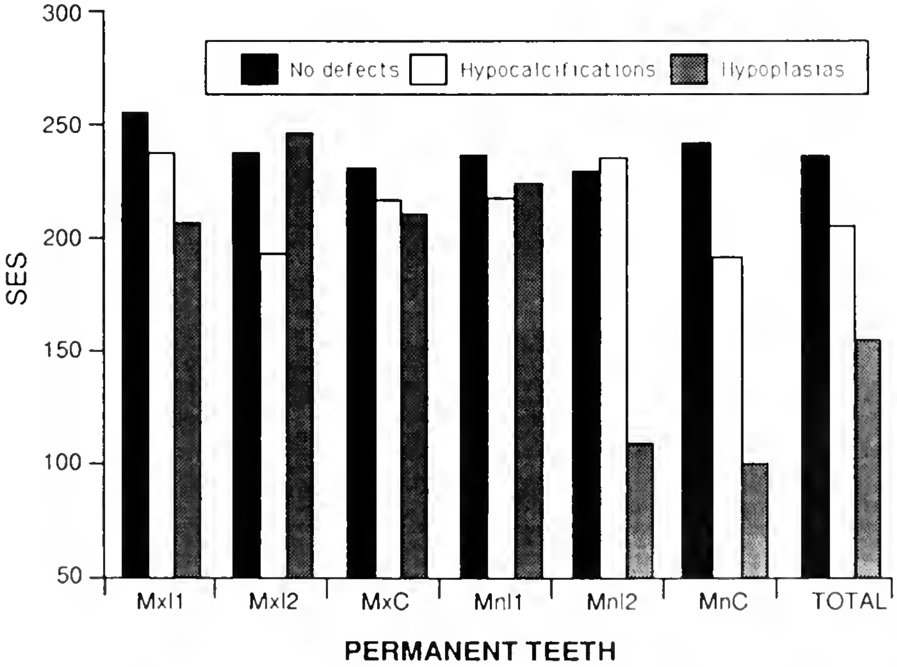


FIGURE 4. Mean socioeconomic score (SES—based on household characteristics and material style of life) for those with no defects, hypocalcifications, or hypoplasias for the six anterior permanent teeth.

with the fact that the hypocalcification rates found in Solis are in line with other low-fluoride areas with much greater access to resources.

The degree of association between hypoplasias and nutrition and socioeconomic status, however, reaffirms the notion that they are related to general conditions of life. These data are consistent with prior experimental and epidemiological data on hypoplasias and nutritional status. Sweeney and coworkers (1971), for example, have linked deciduous hypoplasias to second and third degree malnutrition, measured by growth status, in Guatemalan children. Our study extends from Sweeney's in focusing on permanent tooth defects, a less severe degree of malnutrition and, we believe, less severe hypoplasias.

Discussion

Based on the results of these two studies it is suggested that enamel hypoplasias are indicators of increased stress and adaptative constraint. The general assumption that those with hypoplasias are exposed to more stress than those without (or are less able to rally from this stress) is warranted. While these indicators do not cause death, they are associated with hardship and, therefore, are important factors in elucidation of the human condition.

But, what level of infirmity is associated with enamel hypoplasias? Based on the Mexico data, it is clear that enamel hypoplasias are common under typical conditions of endemic mild-to-moderate malnutrition. Under these conditions one can expect decreases in functions, such as work capacity, sociability, and immune resistance (Chavez and Martinez 1982). The prevalence of defects found in prehistoric populations, which is usually greater than that found in the Mexico study, projects to similar functional effects.

While enamel hypoplasias suggest an important stress, can we say the same for other paleopathological conditions? Do Harris's lines connote an increase in the burden of morbidity? Do arthritic and osteoporotic lesions dictate losses in ability to work and in other functional capacities? Do the infectious and nutritional conditions thought to be causative, at least in part, of periosteal lesions and porotic hyperostosis signify a decrease in adaptation?

In order to answer these questions researchers need to address fundamental issues regarding the cause of lesions in both environmental and biological terms. In order to better appreciate the functional and adaptive meaning of any growth disturbance (Harris' lines, enamel hypocalcifications, skull base height, etc.) the environmental conditions causative of these lesions must be clearly understood. If they are secondary to rather rapid infectious episodes, then the impact on function may be equally short lived. However, if the conditions strike at critical periods and are relatively longer lasting, then permanent damage might result.

The effect of a stressor on an organism is due not only to the characteristics of the stressor, but to the organism's biological response to the stressor (Selye 1973). Therefore, it is equally critical to understand the severity, duration, and quality of the response summoned by the organism (Thomas et al. 1979). Some infections, perhaps causative of periosteal reactions, might elicit an appropriate immunological response leading to little or no lasting and detrimental effects (Ortner 1979). Other conditions, such as endemic parasitism, might lead to disruptions in work and reproductive capacities, but will leave no indications on hard tissue. Still other conditions such as endemic undernutrition might cause a mild porotic hyperostosis, which does not match the severity with which a wide spectrum of functional capacities are inhibited. In short, paleopathological conditions are likely to differ in their adaptive and functional meaning depending on the characteristics of the stressors causative of the lesion, the underlying condition of the organism, and the responses elicited by the organism to the stressful condition. Furthermore, the severity of a lesion might not be easily equated with a severity of functional effects. If we wish to understand the adaptive and functional meaning of paleopathological lesions then we need to better understand the environmental contexts in which they occur and the biological responses which lead to their formation.

Given the magnitude of the problem of undernutrition and associated lesions, both now and in prehistory, it is reasonable to work toward the development of easily implementable measures of nutritional status. Enamel defects have unique strengths of being time specific and indelible, contrasting with more standard nutritional status indicators. Researchers are encouraged to continue to study these defects in prehistory. While there are large gaps in our understanding of these defects, one can make important inferences based on patterns of dental defects. Finally, studies of these defects in experimental and epidemiological contexts will help to elucidate contemporary and prehistoric conditions.

In conclusion, this study has reaffirmed the notion that enamel hypoplasias are related to general conditions of life in marginal environments. It is hoped that studies of enamel defects in living populations will both help us to understand the significance of these defects in prehistory and become important in their own right as tools for understanding the stresses of contemporary conditions. Fifteen years ago Jelliffe and Jelliffe concluded that "further study of its (enamel hypoplasias') etiology and public health consequences seems overdue" (1971:893). This plea remains appropriate.

Literature cited

Allen, L.H., A. Chavez, and G.H. Pelto. 1987. *Collaborative Research and Support Project on Food Intake and Function. Mexico Project Final Report.* Washington, D.C.: United States Agency for International Development.

Barker, D.J.P., and C. Osmond. 1986a. Childhood Respiratory Infection and Adult Chronic Bronchitis in England and Wales. *British Medical Journal*, 293:1271–1275.

———. 1986b. Infant Mortality, Childhood Nutrition, and Ischaemic Heart Disease in England and Wales. *Lancet*, 1:1077–1081.

Chandra, R.K. 1975. Fetal Malnutrition and Postnatal Immunocompetence. *American Journal of Diseases of Childhood*, 129:450–454.

Chavez, A., and C. Martinez. 1982. *Growing Up in a Developing Country.* Mexico City: Instituto Nacional de la Nutricion.

Cohen, J. 1974. Population Differences in Dental Morphology Viewed in Terms of High and Low Heritability. *American Journal of Physical Anthropology*, 41:473.

Cohen, M.N., and G.J. Armelagos, eds. 1984. *Paleopathology at the Origins of Agriculture*. New York: Academic Press.

Cook, D.C., and J.E. Buikstra. 1979. Health and Differential Survival in Prehistoric Populations: Prenatal Dental Defects. *American Journal of Physical Anthropology*, 51:649–664.

Cutress, T.W., and G.W. Suckling. 1982. The Assessment of Non-Carious Defects of Enamel. *International Dental Journal*, 32: 117–122.

Federation Dentaire Internationale (FDI). 1982. An Epidemiological Index of Developmental Defects of Dental Enamel (DDE Index). *International Dental Journal*, 32:159–167.

Goodman, A.H., and G.J. Armelagos. 1988. Childhood Stress and Decreased Longevity in a Prehistoric Population. *American Anthropologist* (in press).

Goodman, A.H., G.J. Armelagos, and J.C. Rose. 1980. Enamel Hypoplasias as Indicators of Stress in Three Prehistoric Populations from Illinois. *Human Biology*, 52:515–528.

———. 1984. The Chronological Distribution of Enamel Hypoplasias from Prehistoric Dickson Mounds Populations. *American Journal of Physical Anthropology*, 65:259–266.

Goodman, A.H., J. Lallo, G.J. Armelagos, and J.C. Rose. 1984. Health Changes at Dickson Mounds, Illinois (A.D. 950–1300). In M.N. Cohen and G.J. Armelagos, eds., *Paleopathology at the Origins of Agriculture*, 271–305. New York: Academic Press.

Goodman, A.H., D.L. Martin, G.J. Armelagos, and G. Clark. 1984. Indications of Stress from Bone and Teeth. In M.N. Cohen and G.J. Armelagos, eds., *Paleopathology at the Origins of Agriculture*, 13–44. New York: Academic Press.

Goodman, A.H., N.A. Rothschild, and G.J. Armelagos. 1983. Social Status and Health in Three Prehistoric Populations from Dickson Mounds, Illinois. *American Journal of Physical Anthropology*, 60:199.

Hamill, P.U., T.A. Drizd, C.L. Johnson, R.B. Reed, and A.F. Roche. 1977. NCHS Growth Curves for Children, Birth–18 Years, United States. *Vital and Health Statistics*, series 2, no. 165. Department of Health, Education, and Welfare Publication Phs 78–1650.

Harn, A. 1978. Mississippian Settlement Patterns in the Central Illinois River Valley. In B. Smith, ed., *Mississippian Settlement Patterns*, 233–268. Academic Press: New York.

———. 1980. The Prehistory of Dickson Mounds: The Dickson Excavation. *Illinois State Museum Report*, 36. Springfield.

Jelliffe, D.B., and E.F.P. Jelliffe. 1971. Linear Enamel Hypoplasia of Deciduous Incisor Teeth of Malnourished Children. *American Journal of Clinical Nutrition*, 24:893.

Jontell, M., and A. Linde. 1986. Nutritional Aspects of Tooth Formation. *World Review of Nutrition and Dietetics*, 48:114–136.

Kreshover, S. 1960. Metabolic Disturbances in Tooth Formation. *Annals of the New York Academy of Science*, 85:161–167.

Lallo, J., G.J. Armelagos, and R.P. Mensforth. 1977. The Role of Diet, Disease and Physiology in the Origin of Porotic Hyperostosis. *Human Biology*, 49:471–483.

Lallo, J., G.J. Armelagos, and J.C. Rose. 1978. Paleopathology of Infectious Disease in the Dickson Mounds Population. *Medical College of Virginia Quarterly*, 14:12–23.

Massler, M., I. Schour, and B. Sarnat. 1941. Developmental Pattern of the Child as Reflected in the Calcification Pattern of the Teeth. *American Journal of Diseases of Childhood*, 62:33–67.

Miler, Ivo. 1982. Nutrition in Early Life and the Development of Resistance and Immunity. *Bibltheca Nutritio et Dieta*, 31:55–60.

Moore, J., A.C. Swedlund, and G.J. Armelagos. 1975. The Use of Life Tables in Paleodemography. *American Antiquity Memoir*, 30:57–70.

Nie, N.H., C.H. Hull, J.G. Jenkins, K. Steinbrenner, and D.H. Bent. 1975. *SPSS Statistical Package for the Social Sciences*. 2d edition. New York: McGraw Hill.

Ortner, D.J. 1979. Disease and Mortality in the Early Bronze Age People of Bab edh-Dhra, Jordan. *American Journal of Physical Anthropology*, 51:589–598.

Pindborg, J.J. 1982. Aetiology of Developmental Enamel Defects Not Related to Fluorosis. *International Dental Journal*, 32:123–134.

Rose, J.C., G.J. Armelagos, and J. Lallo. 1978. Histological Enamel Indicators of Childhood Stress in Prehistoric Skeletal Samples. *American Journal of Physical Anthropology*, 49:511–516.

Rothschild, N.A. 1979. Mortuary Behavior and Social Organization at Indian Knoll and Dickson Mounds. *American Antiquity*, 44:658–675.

Sarnat, B.G., and I. Schour. 1941. Enamel Hypoplasias (Chronic Enamel Aplasia) in Relationship to Systemic Diseases: A Chronological, Morphological and Etiological Classification. *Journal of the American Dental Association*, 28:1989–2000.

Selye, H. 1973. The Evolution of the Stress Concept. *American Scientist*, 61:692–699.

Swardstedt, T. 1966. *Odontological Aspects of a Medieval Population from the Province of Jamtland/Mid-Sweden*. Stockholm: Tiden Barnangen A.B.

Sweeney, E.A., J.A. Saffir, and R. de Leon. 1971. Linear Enamel Hypoplasias of Deciduous Incisor Teeth in Malnourished Children. *American Journal of Clinical Nutrition*, 24:29–31.

Thomas, R.B., B. Winterhalder, and S.D. McRae. 1979. An Anthropological Approach to Human Ecology and Adaptive Dynamics. *Yearbook of Physical Anthropology*, 22:1–46.

White, T. 1978. Early Hominid Enamel Hypoplasia. *American Journal of Physical Anthropology*, 49:79–83.

SUMMARY OF AUDIENCE DISCUSSION: Dental hypoplasia appears to reflect an acute episode of ill health occurring in the presence of and superimposed upon a chronic one.

Miscellaneous Conditions

Secondary hyperparathyroidism in an Andean mummy

James Blackman, Marvin J. Allison, Arthur C. Aufderheide, Norman Oldroyd, and R. Ted Steinbock

While the idiopathic type of bladder stone seems to have been more common in antiquity (Streitz et al. 1981), renal urolithiasis has become more frequent with the industrial revolution in western populations. The metabolic phenomena which led to bilateral, diffuse nephrocalcinosis and renal calculus formation in an adolescent male a millennium ago are presented together with the associated differential diagnosis.

Materials and methods

The spontaneously mummified body was excavated from a burial site near the mouth of the Azapa Valley at Arica in northern Chile. Associated burial goods identified it as a member of the Cabuza culture population (A.D. 350–1000). A total autopsy was carried out at the University of Tarapaca (Arica, Chile) with final removal of all soft tissue and paleopathological examination of the skeletal tissues. Following gross examination all identified soft tissue organs were sampled for histologic and physicochemical studies.

Flat plate bone x-rays were prepared of the skull, mandible, femurs, tibias, humeri, forearms, feet and hands, as well as soft tissue films of both kidneys.

Tissues for histologic studies were rehydrated and fixed in 4% formalin and in Ruffer's solution, after which they were processed in the same manner as routine surgical tissues, dehydrated with organic solvents (alcohols, benzene, xylol), embedded in paraffin and sectioned at four microns thickness. Routine stains included hematoxylin and eosin, Movat's pentachrome and Gomori's silver stain for reticulum (Zimmerman 1976:59–61).

Immunocytochemical studies for thyroid and parathyroid antigens were performed on deparaffinized histologic sections using peroxidase-antiperoxidase methods. Thyroid and parathyroid antibodies and other reagents were supplied by Biogenex Laboratories, Dublin, California. Positive controls consisted of thyroid and parathyroid tissues from recent hospital autopsies.

Oxalate crystal identification studies were carried out on deparaffinized tissue sections as described by Johnson and Pani (1962).

Renal stone analysis was performed by Norman Oldroyd at the Louis C. Herring Laboratory in Orlando, Florida. Methods included infrared spectroscopy, x-ray diffraction, and crystallographic studies.

Polarizing quality of tissue crystals was evaluated by examination of histologic sections with light microscopy using polarizing filters.

Hydrated, small (one-half millimeter) fragments of tissue from the neck masses were studied by transmission electron microscopy after glutaraldehyde fixation on a Phillips 201 transmission electron microscope (TEM). Both gross tissue fragments and unstained histologic sections of the neck masses and kidneys were examined in an Amray 1000 scanning electron microscope (SEM) equipped with an electron probe for element identification by energy dispersion x-ray analysis (EDXA).

Results

GROSS SOFT TISSUE FINDINGS

A large perforation of anthropogenic origin was found in each ear lobe. In the Cabuza culture this signals an elite social status (shaman?). Except for the spleen, the major organs normally present in the thoracic and peritoneal cavities were easily recognized and in their normal positions. Pathological findings included a left lung dried in an expanded condition, held in position by adhesions between the visceral and parietal pleural surfaces over all aspects of both lobes. The right lung was thin (2–3 mm) and collapsed, conforming to the curvature of the posteromedial chest wall interior, an appearance common for a normal lung in spontaneously mummified bodies. The heart was of normal size, but much of the visceral pericardial surface was covered with a thin (1 mm), cream-colored layer of material which could not easily be

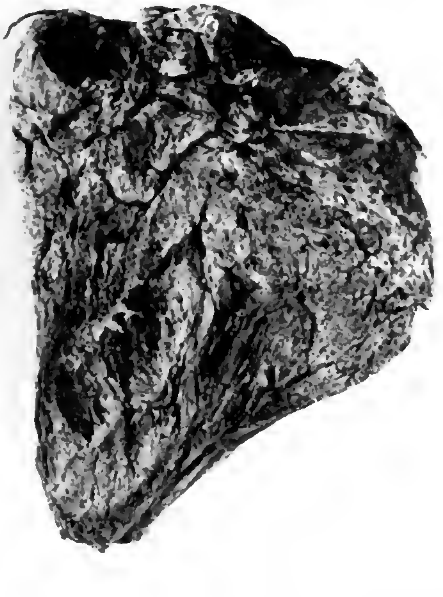


FIGURE 1. Heart demonstrating fibrinous exudate on pericardial surface.

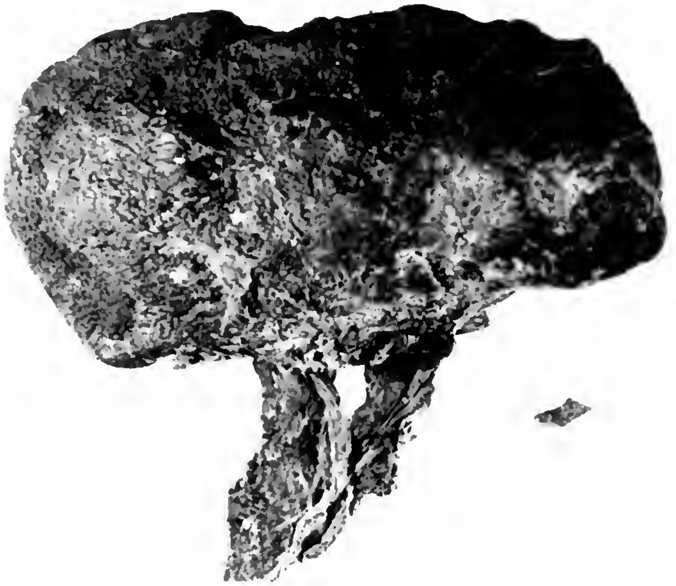


FIGURE 3. Right kidney demonstrating moderate atrophy with granular surface, hydroureter and adjacent calculus removed from calyx in lower pole.

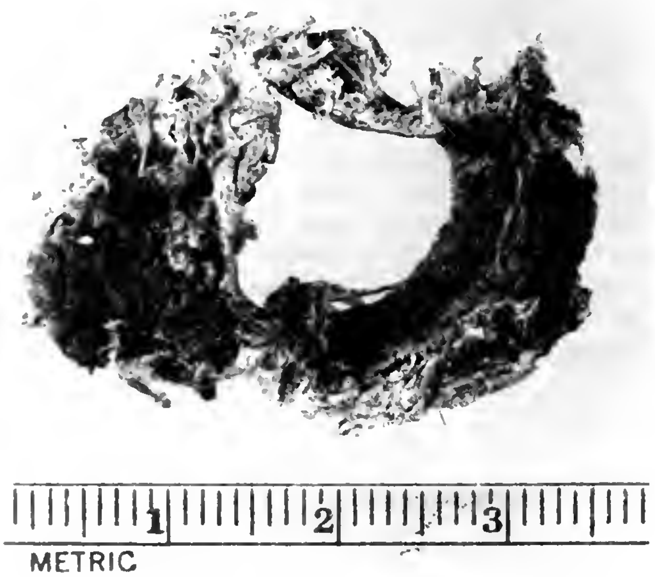


FIGURE 2. Section through the trachea just below the larynx demonstrates bilateral dark-brown masses juxtaposed to the trachea.

separated from the myocardium (Figure 1). At the level of the inferior portion of the larynx bilateral, dark brown masses, roughly spherical and measuring 1.5 cm in diameter, were juxtaposed to the trachea (Figure 2). Long bones appeared lighter than usual and a cross-section of the femur revealed a thin cortex. The right kidney measured 7 × 4 × 2 cm and the left appeared to be about two-thirds as large. The pelves and calyces on each side were well defined and each contained at least five easily recognized calyces. Two gray calculi, measuring 2.5 mm and 6 mm, were found in the right kidney, one in an upper pole (Figure 3) calyx and another in that of the lower pole. Both ureters were moderately dilated to about two or three times their normal diameter from the renal pelvis to the urinary bladder on each side (Figure 4). No abnormalities of the bladder and its trigone area were evident, although the brittle, dehydrated state of the tissues made it impossible to evaluate patency of the ureterovesical junctions. Gross autopsy diagnoses included bilateral hydroureter, right renal calculi, left renal atrophy, pericardial exudate, bilateral neck masses of undetermined nature, and mild, diffuse osteoporosis.

MICROSCOPIC STUDIES

KIDNEYS: The tubular structure of renal cortex was clearly evident in both kidneys, although the architecture of the left kidney was much more extensively destroyed than that of the right. Glomeruli were difficult to find. Irregular masses of calcified tissue were scattered through the parenchyma, the smaller ones revealing residual traces of arterial wall or cortical tubules as their locale of origin. Sheaf-shaped clusters of needlelike crystals were scattered through the parenchyma, some within tubule lumens and others superimposed on the calcified masses.

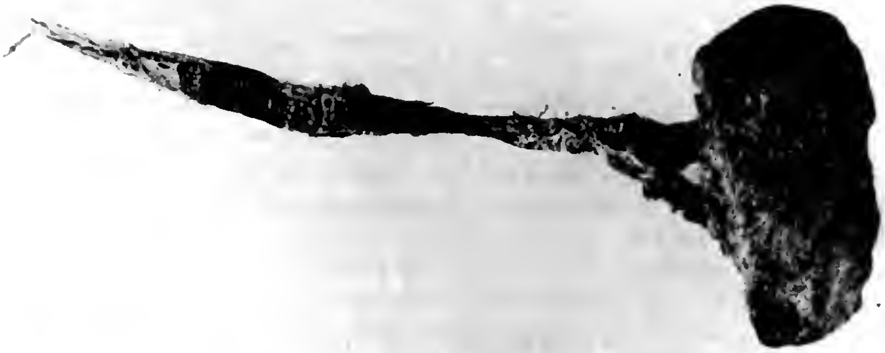


FIGURE 4. Right kidney demonstrating moderate atrophy and moderate hydroureter.

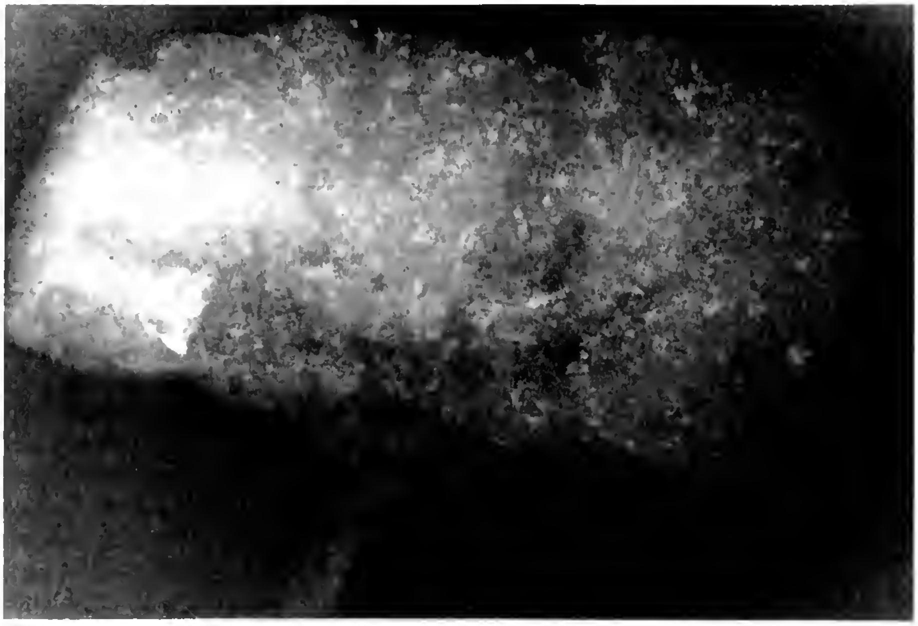


FIGURE 5. X-ray of right kidney demonstrating diffuse nephrocalcinosis in the form of multiple small (1 mm) radiodensities scattered through the parenchyma and a calculus in the lower pole.

LUNGS: Mild fibrosis was present, but no bacteria or pathogenic fungi were seen.

HEART: The light-colored material on the pericardial surface was amorphous, consistent with fibrin. No bacilli were demonstrated.

NECK: Although no epithelium survived, both reticulum and conventional stains identify a well-defined glandular pattern with amorphous material occupying the centers of the glandlike structures.

X-RAYS

KIDNEYS: Many small (1–3 mm), irregular opacities were scattered through both kidneys, more densely in the left one. Films exposed after removal of the larger calculus revealed the smaller one still impacted in the lower pole (Figure 5).

BONES: Mild, diffuse osteoporosis was evident but no subperiosteal erosion or cortical tunneling changes could be identified, and the lamina dura surrounding the teeth was intact. No cystic changes were apparent.

SPECIAL STUDIES

KIDNEYS: Only a few of the crystals became refractile when viewed under polarized light. Electron microprobe EDXA demonstrated high levels of phosphorus and potassium in most, calcium in a few, and small amounts of magnesium in many. Occasionally small amounts of iron, sodium, sulfur and chlorine were present. The larger, calcified masses contained much phosphorus, potassium, and lesser amounts of calcium. Sections treated as outlined by Johnson and Pani (1962) revealed the reactions expected with calcium oxalate crystals except that most did not stain with alizarin red S. Constituent compounds composing the larger calculus are:

TABLE 1. Possible causes and characteristics of bilateral nephrocalcinosis, renal lithiasis and renal failure

	Idiopathic hyper-calciuria	*Primary oxalosis*	*Primary hyperpara-thyroidism*	*Secondary hyperpara-thyroidism*	*Renal distal tubular necrosis*
Usual age	35–55+	Child	25–50	Any	Child, adult
Nature of renal stones	Ca oxalate	Ca oxalate	Ca phosphate	Due to primary disease	Ca phosphate
Nephrocal-cinosis	+	+	+	+	+
Uremia	As complication	Terminally	As complication	+	Mild-to-moderate
Parathyroid hyperplasia	If uremic	+	+	+	?
Osteopenia	+	0	+	+	+
Cause	Hereditary	Congenital	Neoplasia	Primary renal disease	Hereditary
Frequency	Common	Rare	Common	Common	Rare

calcium oxalate monohydrate (20%), calcium oxylate dihydrate (9%), cagnesium ammonium phosphate hexahydrate (10%), carbonate apatite (10%), ammonium hydrogen urate (1%), and proteins, calcified tissue, and degenerated blood (50%).

NECK MASSES: Transmission electron microscopy revealed no residual evidence of intact cellular structure. Immunoperoxidase studies demonstrated no reactivity of any material in the neck masses with antibodies against either thyroid or parathyroid hormone.

LUNGS: Polarized light study of microscopic sections revealed many minute, crystalline structures. Electron microprobe analysis demonstrated an elemental pattern identical with that of soil from the tomb: silicon, aluminum, calcium, chlorine, sulfur and potassium.

Discussion

The diagnostic problem to be resolved is identification of one or more conditions capable of producing bilateral renal nephrocalcinosis with atrophy, bilateral mildly dilated ureters, right renal calculi of a mixed crystalline structure, fibrinous pericarditis, and mild osteoporosis in an 18-year-old male.

Nonbacterial, fibrinous pericarditis most probably reflects a terminal, uremic status secondary to the renal pathology.

The ureters were not dilated to the extreme degree expected in either congenital hydroureters or long-standing, acquired obstruction, yet were two to three times wider than normal with a normal wall structure histologically. Transient obstruction by a calculus, expelled from the kidney and passed subsequently, would appear to be the most likely explanation of this finding.

Principal candidates for explanation of this constellation of findings are listed in Table 1. It includes conditions which produce renal calculi either (1) as a complication of a primary renal disease, or (2) by causing increased excretion of calcium or oxalate through a normal kidney with resulting precipitation of the calcium salt and secondary renal damage by the calculus (infection or obstruction).

The chemical structure of the renal calculi produced varies in the different conditions under consideration. In evaluating these diseases it must be kept in mind that a calculus of almost any structure may eventually cause renal parenchymal damage by obstructing urine flow or by bacterial infection secondary to erosion of the renal pelvic mucosa. The urea-splitting action of the organisms commonly producing kidney infections will then usually cause precipitation of struvite (magnesium ammonium phosphate) regardless of the original chemical and crystalline structure of the calculus. Such a sequence of events, however, is often revealed by separate chemical examinations of the stone's various layers, the innermost ones reflecting the original chemical milieu at the time of the calculus' origin.

OXALOSIS is characterized by an excessive level of oxalate in the blood, tissues and urine. It occurs as a congenital metabolic defect or as an acquired condition with excessive absorption secondary to intestinal pathology. The insoluble salt, calcium oxalate, precipitates in the urine. It is a rare disease. A much higher content of calcium oxalate would be expected in the calculus than was seen in this Cabuza subject. Furthermore, since the oxalate is distributed by the blood throughout the body, widespread calcium oxalate deposits in many extrarenal tissues are expected but were absent in our subject. Oxalosis appears to be a most improbable explanation for our findings.

IDIOPATHIC HYPERCALCIURIA must be considered a serious possibility. In this hereditary condition a poorly defined metabolic aberration causes increased urinary excretion of calcium which may precipitate as calcium oxalate or phosphate. It most frequently affects adults in the fourth and fifth decades, but has been reported in children (Potts 1983:1929–1943). Nephrocalcinosis may also occur.

RENAL TUBULAR ACIDOSIS is an inherited defect in which the distal renal tubule is unable to maintain normal pH control resulting in excretion of an excessively basic urine causing diffuse precipitation of calcium phosphate (nephrocalcinosis). The disease, however, is rare, usually requires a much longer time to destroy the kidneys, and produces discrete calculi (of predominantly calcium phosphate structure) only in older patients. This, too, would seem an unlikely solution to our diagnostic problem.

PRIMARY HYPERPARATHYROIDISM is an obvious consideration. In this rather common condition—1:1000—neoplasia causes excessive production of parathyroid hormone. The neoplastic lesion is usually a benign adenoma, rarely a carcinoma, of one or more parathyroid glands, or sometimes primary hyperplasia of all four glands. One of this hormone's principal effects is bone decalcification. Unregulated hormone production by the tumor floods the kidney with calcium to be excreted which often then precipitates as calcium phosphate. It may do so in the form of diffuse nephrocalcinosis or as discrete macrocalculi (but usually not both in the same individual). The hormone's effect on the bones, some forms of which are more or less unique, may be detected radiologically. These include subperiosteal resorption (especially evident in phalanges), erosion of the lamina dura adjacent to the teeth, and cystic-appearing lesions in long bones reflecting the presence of osteoclastic "tumors." However, these are often most obvious in rather late or advanced conditions and simple osteoporosis may be the only identifiable radiological abnormality in many. Most clinical diagnoses are made on individuals in the third to sixth decades. In our subject calcium phosphate is neither the principal nor the central component of the renal calculi. He is younger than most persons with this diagnosis and has none of the radiological stigmata of primary hyperparathyroidism except the relatively nonspecific presence of diffuse osteopenia.

There is, however, another form of HYPERPARATHYROIDISM—THAT SECONDARY TO HYPOCALCEMIA. The most common condition producing a low blood calcium level is chronic renal failure. In this state, the failing kidney's inability to excrete phosphates results in a relentless rise in blood phosphorus content and, because of calcium's reciprocal relationship to phosphorus, the blood calcium drops to subnormal levels. Hypocalcemia thus provides a perpetual, pathophysiological stimulus to parathyroid hormone production. The consequent release of calcium by parathyroid hormone from the skeleton raises and may even restore the normal blood calcium level. The tissue concentrations of calcium and phosphorus frequently are then in a supersaturation range and soft tissue precipitates of calcium phosphate occur. The kidney itself is particularly vulnerable to such an event (diffuse nephrocalcinosis), but commonly it also occurs in other soft tissues. While such minute calcifications are distributed diffusely through both kidneys, the condition itself does not normally generate urinary calculi; any such renoliths are usually the product of the original renal disease which destroyed the kidneys. In children the chronic, destructive, primary renal condition leading to secondary hyperparathyroidism is by far most commonly chronic pyelonephritis (Kissane and Smith 1969:755).

Other conditions causing nephrocalcinosis seem most improbable. The hypercalcemia of sarcoidosis is almost invariably accompanied by infiltrations in other organs, absent in our subject (Longcope and Freiman 1952). Renal cortical necrosis spares the medulla, which was also destroyed in this mummy's kidneys. Medullary sponge kidney spares the cortex, again not consistent with the total kidney destruction present in the Cabuza's kidneys (Heptinstall 1983). His bones also revealed no discrete osteolytic lesions (carcinoma; blood dyscrasias). Milk alkali syndrome is caused by excessive alkali ingestion for peptic ulcer disease; it may produce mild renal insufficiency. As a presumed shaman (large ear lobe perforations) he undoubtedly indulged in ritual practice of coca leaf chewing accompanied with alkali to enhance the leaf's alkaloid extraction, but it is not probable this would have involved sufficient alkali ingestion to duplicate the effect of the milk alkali syndrome. There is no reason to suspect hypervitaminosis D.

The composition of this mummy's renal calculus is of special interest. While calcium oxalate is certainly the dominant crystal present (nearly 30% of the stone's weight), significant amounts of struvite and carbonate apatite (each 10%) are also noted and seem to be distributed diffusely through the calculus. In addition it is important to emphasize that *half* of the calculus was composed of blood, proteins, and calcified tissue. More so than in purely metabolic conditions, this calculus appears to have been initiated by precipitation of renal salts within necrotic, hemorrhagic tissue debris (a "matrix" stone).

It appears to us that, while several of the discussed conditions are possible, the nature of the calculus together with the pattern of renal destruction in this age group imply that the initiating event was a probably primary, bilateral renal disease with calculus formation resulting from local tissue necrosis. Gradual onset of progressive renal failure produced a uremic state causing fibrinous pericarditis as well as secondary hyperparathyroidism. The latter complication is then responsible for the observed diffuse, bilateral nephrocalcinosis via the mechanism outlined above, as well as for the diffuse osteoporosis.

The nature of the postulated primary renal disease cannot be established histologically; this only reveals diffusely destroyed, fibrotic and calcified parenchyma of end-stage chronic renal disease. Chronic pyelonephritis would be suspected on the basis of frequency, though the inflammatory cells would not be preserved in a spontaneously mummified body. The noted size asymmetry is common in this condition. Glomerulonephritis does not normally produce calculi. No congenital renal deformities are recognizable. Furthermore, the formation of calculi in an infected kidney is a well-known phenomenon. Somewhat disturbing is the fact that struvite accounts for only 10% of the stone's weight, but in chronic pyelonephritis the organisms may be minimal or absent in the late stage of the disease, and many crystals may have been added to the calculus during the terminal stage of secondary hyperparathyroidism.

The bilateral, moderately dilated ureters are most easily explained by assuming the passage of previous calculi with transient, obstructive episodes.

The nature of the bilateral neck masses could not be determined beyond equivocation. While their histologic pattern suggests a glandular nature, immunologic procedures could not differentiate between thyroid or parathyroid tissue. Normal thyroid tissue (and certainly also the small, normal parathyroid glands) cannot usually be identified in spontaneously mummified bodies (Gerszten et al. 1976). Even if positively identified as parathyroid, their multiple, enlarged status would not be useful in separating primary from secondary hyperparathyroidism since 15% of the former and 100% of the latter reveal enlargement of more than one gland.

The mild pulmonary fibrosis is most likely secondary to inhalation of the soil dust visualized in the histologic sections with polarized light and identified by EDXA (El-Najjar et al. 1985:274). It is conceivable, however, that the major episode of left pneumonia (evidenced by massive left pleural adhesions) suffered at some previous time may have caused bacteremia and been the source of the original pyelonephritis episode.

In summary, this 18-year-old Cabuza male most likely suffered renal destruction from chronic pyelonephritis with secondary renal urolithiasis, chronic renal failure, uremic pericarditis, and secondary hyperparathyroidism leading to diffuse, bilateral nephrocalcinosis. Alternative diagnoses appear to be less probable.

Literature cited

El-Najjar, M., A.C. Aufderheide, and D.J. Ortner. 1985. Preserved Human Remains from the Southern Region of the North American Continent: Report of Autopsy Findings. *Human Pathology*, 16:273–276.

Gerszten, E., M.J. Allison, A. Pezzia, and D. Klurfeld. 1976. Thyroid Disease in a Peruvian Mummy. *MCV Quarterly*, 12:52–53.

Heptinstall, R.H. 1983. *Pathology of the Kidney*, 1599–1634. Boston: Little, Brown and Company.

Johnson, F.B., and K. Pani. 1962. Histochemical Identification of Calcium Oxalate. *Archives of Pathology*, 74:347–351.

Kissane, J.M., and M.G. Smith. 1969. *Pathology of Infancy and Childhood*. St. Louis: C.V. Mosby.

Longcope, W.T., and D.G. Freiman. 1952. A Study of Sarcoidosis. *Medicine*, 31:1–132.

Potts, J.T., Jr. 1983. Disorders of Parathyroid Glands. In R.G. Peterson et al., eds., *Harrison's Principles of Internal Medicine*. New York: McGraw-Hill.

Streitz, J.M., A.C. Aufderheide, M. El-Najjar, and D.J. Ortner. 1981. A 1,500-Year-Old Bladder Stone. *Journal of Urology*, 126:452–453.

Zimmerman, M.R. 1976. A Paleopathologic Archeologic Investigation of the Human Remains of the Dra Abu El-Naga Site, Egypt Based on an Experimental Study of Mummification. Ph.D. dissertation, University of Pennsylvania. Ann Arbor: University Microfilms International.

SUMMARY OF AUDIENCE DISCUSSION: The stone consisted of 50% organic matter whose nature is speculative, but could have been a sloughed renal papilla. The absence of osteitis fibrosa cystica (parathormone-induced foci of bone lysis filled with fibrous tissue) is probably the consequence of the shorter time period available in the secondary form of hyperparathyroidism. Morphologic evidence of hyperparathyroidism can, however, still be recognized in ancient skeletal tissues by the "swiss cheese" pattern in trabeculae produced by the tunneling effect of parathormone-stimulated osteoblastic clusters.

Noma—cancer aquaticus: First indication of the skin involving disease in ancient Egypt?

Wolfgang M. Pahl and W. Undeutsch

Paleopathological publications on Egyptian mummies sporadically report skin lesions, which are usually the result of trauma and very often connected with skeletal damage, whereas there is little evidence of pathological changes of the soft tissue only, especially the skin (Sandison 1967; Ruffer and Ferguson 1911; Pahl 1986). This fact cannot be explained by a lower incidence of such diseases in antiquity—ample evidence as well as therapeutic advice is provided, for example, by the Papyrus Ebers, the Hippocratic Corpus, or Celsus's reports. The paucity of cases could be due either to the poor state of tissue preservation or to the lack of special research projects in this field. In a smaller number of cases the embalming substances applied to the body surface of the mummy prevent an adequate evaluation of the object.

Presentation of the following casuistry is justified on the grounds of the rarity of soft tissue pathology and the macroscopic uniqueness and singularity of such a finding in mummy remains from ancient Egypt. As far as we know, no comparable case has been reported from other geographical regions.

Material and methods

The object under investigation is the head of a male, adult mummy about 30 years of age. Provenience: Lower Egypt. Date: Late Period. It is stored in the Egyptian mummy collection of the Institute of Anthropology and Human Genetics at the University of Tübingen, registration No. 1565.

In addition to a detailed macroscopic inspection, microphotographs with different stains were prepared from most of the ulcerations. Radiological investigations, including orthopantomography, were carried out.

Description of lesions

It was possible to identify a total of five skin defects. Based on their exterior appearance it can be stated with almost complete certainty that at least four of these are of the same origin.

Lesion I: Region of the jaw, approximately 10 mm right lateral to the median sagittal line on the level of the lower canine. The lower lip is partly involved (Figures 1,2,7,8).

Lesion II: Region of the infratemporal facies of the right maxilla, immediately cranial to the 2d upper molar. Exposure of the gingiva (Figures 2, 9,10).

Lesion III: Left buccal region at the level of the lower margin of the zygomatic bone (Figures 1,3,11).

Lesion IV: Region of the right inner mandibular angle, near lesion II (Figures 2,9,10).

Lesion V: Region of the right external mandibular angle, immediately on the frontal cord of the sternocleidomastoid muscle (Figures 2,12).

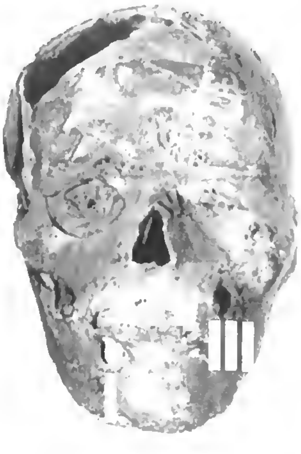
Lesions I–IV are characterized by an irregular, circular, sharply defined, almost punched-out appearance; ulceration of all soft tissue down to the bone; and almost uniform size of ca. 15 mm in diameter. Lesion I shows a steeply sloping, craterlike margin. There is no outer wall and no recognizable color changes or scars. Minute perforations and tunnel formations caused by insects are to be found in the area of lesion III and in other skin regions outside the face. Except for lesion III, in which two perforations of the maxillary bone are observable—most likely caused by an apical tooth abscess, no further osseous destruction traceable pathologically to the soft tissue lesions was detected macroscopically.

Lesion V, located outside the frontal and lateral part of the face, cannot be compared either macroscopically or topographically with the other ulcerations. Rather, it is a partially smooth, oval cavity with a diameter of ca. 12 mm and a depth of ca. 14 mm, with a slightly punctured surface on the bottom connected to the soft tissue of the neck.

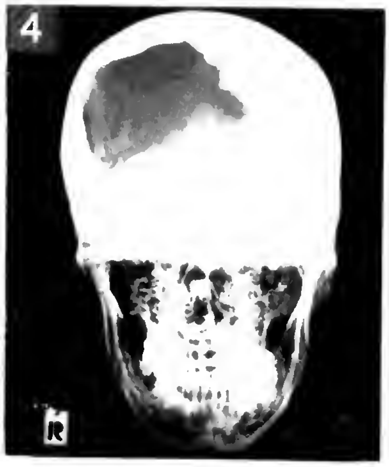
Discussion

Taking into account the missing body of the mummy, which precludes a differentiation between local and whole body involvement of the soft tissue, diagnostic considerations based on the following criteria can be evaluated with all due consideration: localization, number, limitation, shape, and extension of skin lesions I–V. They are supported by radiological and histological findings.

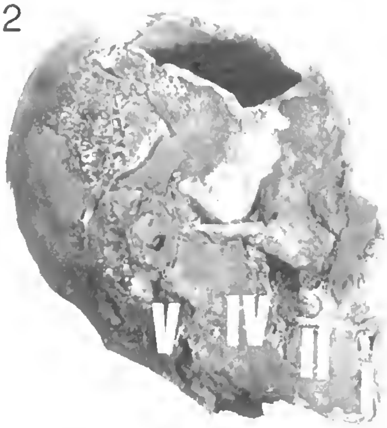
1



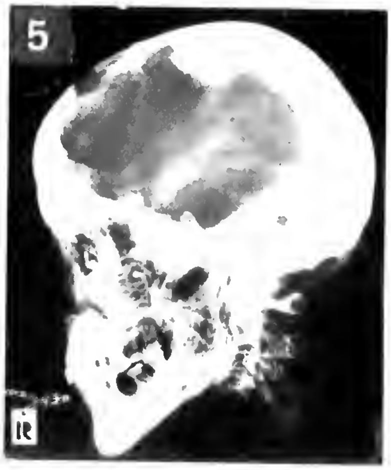
4



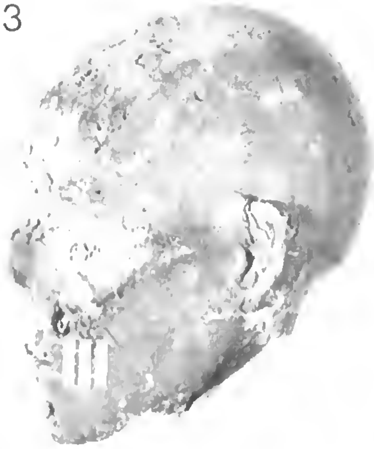
2



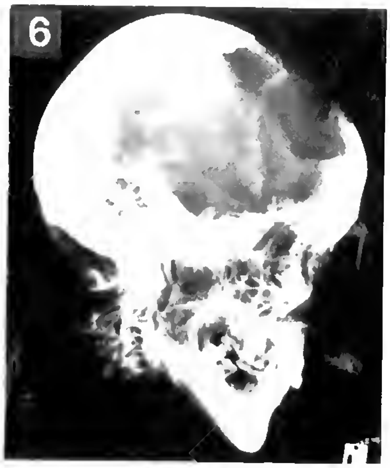
5



3



6



FIGURES 1–3. Specimen No. 1565 (Provenience: Lower Egypt. Date: Late Period) of the Mummy Collection of the Institute of Anthropology and Human Genetics, University of Tübingen (F.R.G.): *1*, anterior view showing skin ulcerations I, III; *2*, right lateral view with skin ulcerations I, II, IV, V; *3*, left lateral view showing skin ulceration III.

FIGURES 4–6. Radiographs of specimen No. 1565: *4*, posteroanterior projection; *5*, right lateral projection; *6*, left lateral projection. In none of these projections is it possible to identify osseous destruction.

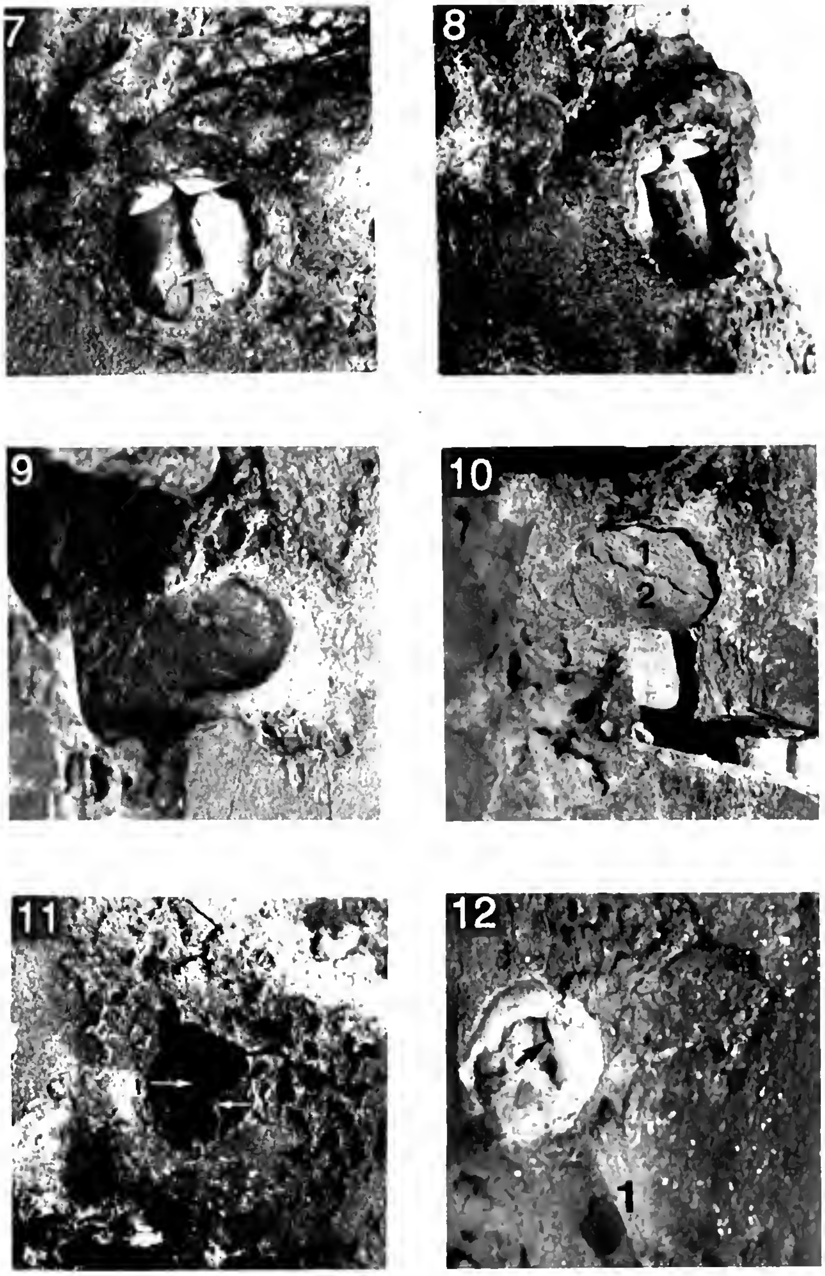
First, a postmortem origin of the defects definitely can be excluded. The radiological investigation in posteroanterior and right and left lateral projections (Figures 4–6) as well as the dental x-ray (orthopantomography) (Figure 13) reveal no signs of osteolytic, metastatic or primary destruction. Instead, a pathological dental process was detected, which corresponds to the already mentioned small perforations of the maxilla at exactly the same level of soft tissue lesion III (Figures 1,3,11 arrows,13 arrow). Another examination with a stereoscopic dissection microscope proved that the osseous defect corresponds to the apparent apical abscesses of the left upper premolars. In all probability, this has no relationship to the cutaneous lesion III. It can therefore be postulated that the skin foci are lesions of the soft tissue only, which, at least to date, have not involved the bone.

Based on the above remarks, the types of diseases relevant for a differential diagnosis of lesions I–IV would be the following:tuberculosis, tropical ulcer, ecthyma, cutaneous leishmaniasis, treponematosis, noma (cancrum oris, cancer aquaticus).

Because of differing characteristic signs, the following diseases should not be considered in the differential diagnosis: nocardiosis, actinomycosis (shape, developing stages), nontuberculous mycobacterial ulcer (irregular limited lesions, localization), skin tumors and secondary deposits of tumors (development, limitation of the foci), and lepromatous ulceration (shape, developing stages, localization, bone involvement).

TUBERCULOSIS

In cases of tuberculosis, the lungs and intestines are the primary sites of infection. Dermatological manifestations are extremely rare. Differences between these and ulcerations I–IV concern the number of defects (few in tuberculosis), their shape (irregular margins in tuberculosis), extension (mostly shallow ulcerations secondary



FIGURES 7–12. Close-up of skin ulcerations I–IV in specimen No. 1565: *7, 8*, different views of ulceration I; teeth and part of mandibula exposed; *9*, soft tissue ulceration II and IV; *10*, ulceration II after biopsy (*1*, periosteum, *2*, maxillary bone); *11*, ulceration III with exposed maxilla; perforation due to an apical tooth abscess on level of first upper premolar *(arrows)*; *12*, ulceration V with exposed right styloid process on bottom of cavity *(arrow)* (*1*, ascended mandibular ramus).

to a papulous initial stage) and morphology of the lesions (Kalkoff 1981). Although there can be no doubt that the ancient Egyptians were affected by at least skeletal tuberculosis (Sandison 1972; Zimmerman 1977), the skin lesions I–IV in case 1565 were not caused by *Mycobacterium tuberculosis* of human or bovine form.

ECTHYMA

Ecthyma simplex is a pyogenic infection caused by alpha-streptococcus (Korting and Denk 1974:549–550) and found in subtropical and tropical areas. Malnutrition and poor hygiene promote its occurrence. After an initial pustulant stage, deep necrotic ulcerations may follow. These can be of polymorphic shape; in addition to irregularly shaped circular lesions, punched-out defects appear. The regressive phase is characterized by formation of scar tissue and hyperpigmentation of the marginal zone. Neither stage is present in the case under discussion. The monomorphic size of the lesions I–IV and the depth of the ulcers to the bone clearly demonstrate the incompatibility of these lesions' appearance with ecthyma.

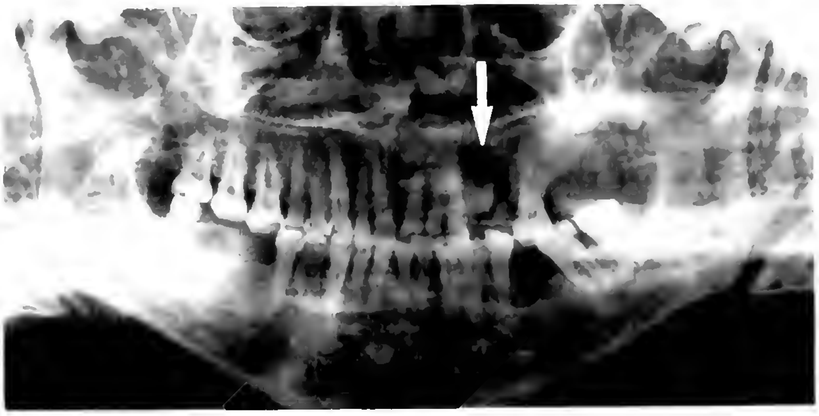


FIGURE 13. Dental x-ray (orthopantomography). Arrow indicates an osteolytic area, corresponding to maxillary tooth abscess mentioned in Figure 11, being in close contact to ulceration III.

TREPONEMATOSIS

It seems that there are no sure indications that diseases caused by *Treponema pallidum* infected the ancient Egyptian population (Sandison 1972: 218). Nevertheless, some of these infections have to be included in the differential diagnosis because the defects in case 1565 are similar to lesions appearing in the extragenital primary manifestation of primary lues, in subcutaneous syphilides of tertiary lues, and occasionally in yaws—a nonvenereal type of treponematosis. However, extragenital manifestations are less frequent than genital ones and usually appear as a single lesion. Normally, they do not reach the degree of soft tissue destruction detected in individual 1565 (Luger 1981). Similar reasoning can be applied in regard to the advanced stage of syphilis. Another, more important argument for not correlating the disease in the investigated specimen with venereal and nonvenereal syphilis is the morphologic uniformity of lesions I–IV. Furthermore, yaws is a disease of subtropical climates, and accordingly its occurrence in Egypt is far less probable than, for example, the nonvenereal endemic syphilis found today in the Nile Delta (Maleville 1976).

TROPICAL ULCER

Tropical ulcer is a phagedenic ulcer primarily found in tropical and humid climates. Established lesions contain fusospirochaetal organisms, but it is unclear whether these are the primary infecting agents. Epidemics have been reported from northern Africa. Following erythema a pustule develops, followed in turn by a sharply limited, circular or oval ulceration with undermined, slightly raised margins. Later the margin hardens, further deepening the crater to the point of exposing the bone (Connor and Neaffie 1976). Malnutrition, inappropriate treatment, unhygenic living conditions, and contamination of the ulcers are factors which prevent healing. Such defects are located primarily on the distal part of the leg above the malleoli (compare Haneveld 1974). The diagnostic difference between tropical ulcer and lesions I–IV consists in the former's raised margins, isolated lesions, and characteristic location on the lower extremities.

CUTANEOUS LEISHMANIASIS

An infection of the skin by a protozoan of the genus *Leishmania* includes three clinical-pathological entities. One of them, the tropical sore, must be included in the differential diagnostic possibilities. It represents a single lesion caused by *Leishmania tropica*. Geographical distribution: tropics and subtropic; sporadic in the southern part of Europe. Predominantly the unclothed regions of the body (mainly face) are involved. The disease shows circular or oval, sharply limited, partly raised margins, and its development begins with a local erythema, followed by pustules and papules, and finally a shallow ulceration (Braun-Falco et al. 1984:178–181). Normally the lesions disappear after one year and a scar remains. Distinctive marks concerning the lesions in case 1565 are the slight outer wall, the depth of the ulcerations, the absence of bone exposure and the stages of the growth process.

NOMA (CANCRUM ORIS)

Noma is an acute, progressive, necroinflammatory process of unknown origin. It involves the soft tissue of the face and during later stages the facial skeleton as well. Although spread throughout the world, noma is rare in western Europe and North America (Joseph and Duncan 1976). In recent years, it has been reported in Africa and Asia. The disease corresponds to the so-called cancer aquaticus of the Middle Ages and was prevalent during that era. Predisposing factors include immune deficiency due to malnutrition or consumptive diseases. Bacteria isolated from the base of the lesions often include spirochetes, corynebacteria and others, but it is more likely that these represent secondary contamination. The prognosis of the disease was fatal in the preantibiotic era and is still severe today (Tempest 1966:949). The macroscopic appearance of noma corresponds in its essential features to lesions I–IV of subject 1565. The localization (facial region), number of foci (multiple lesions are reported), extension (exposure of the bone), shape and size of the ulcers are in absolute agreement with lesions I–IV. Differences do exist, such as those concerning the age of the involved patients, which are mainly infants in present-day clinical medicine (Figure 14).

In addition to the hitherto diagnostic considerations, lesion V (Figure 12), located in the right mandibular angle and clearly defined, should be discussed. Obviously there is no morphological similarity between lesion V and lesions I–IV. Nevertheless a patho-

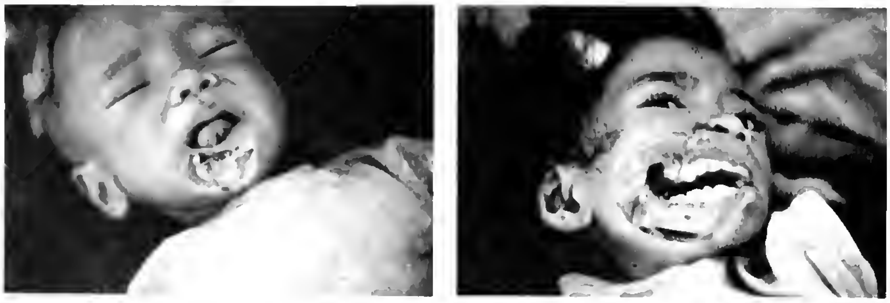
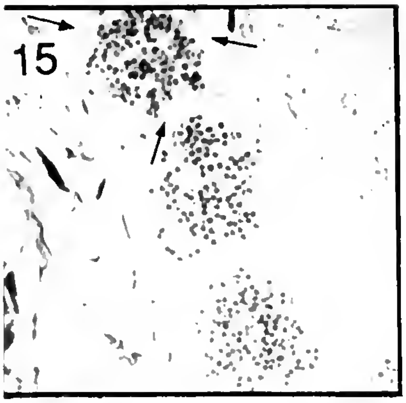
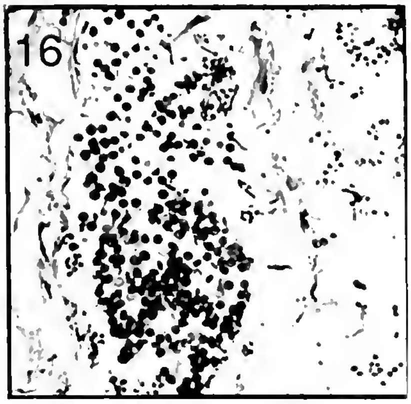


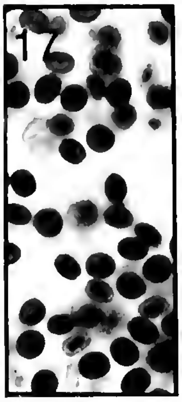
FIGURE 14. Different degrees of noma in clinical patients. Photos courtesy of Armed Forces Institute of Pathology, Washington, D.C.

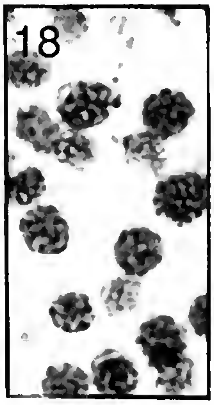
genic connection could be postulated for the following reasons: (1) most of the lesions (I, II, IV) are located in the right part of the face; (2) lesion V is located on the level of the middle group of the nodi lymphatici cervicales profundi which collect the lymph coming from the head and neck region; (3) the discussed diseases cause a more or less severe reaction of the lymphatic system, that is, lymphold hyperplasia or ulcerative lymphangitis.

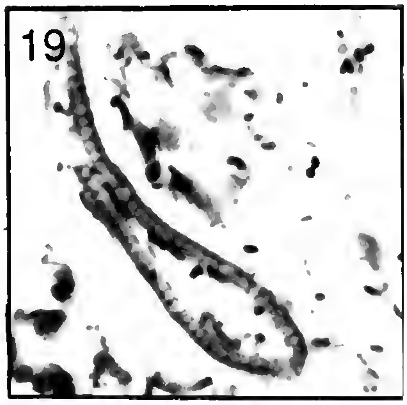
However, investigation of the margin of lesion V by means of a stereoscopic dissecting microscope reveals that no inflammatory or healing process can be detected. Furthermore, it has not been sufficiently proven whether the penetrating defect on the bottom of the cavity exposing the styloid process was caused either *intra vitam* or *post mortem*. We assume that lesion V corresponds to a calcified lymph node cavity (as it is occasionally diagnosed relative to tuberculosis) or to a cystic benign tumor. Any connection with lesions I–IV is conceivable, but not necessary. Due to the brittle surrounding tissue, histological sections could not be prepared from this anatomical region.

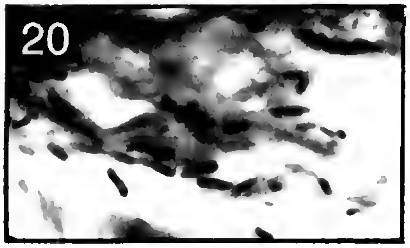


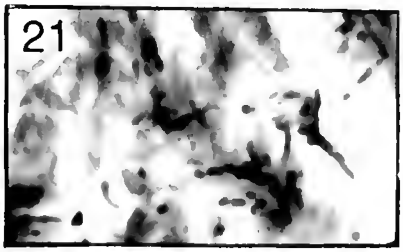












FIGURES 15–18. Microphotographs (specimen 1565) taken from histological sections (ulceration III): *15*, different cell types partly conglomerated into oval cell clusters, Giemsa, ×68; *16*, cell clusters, Giemsa, ×243; *17*, close-up of cells from Figure 16, showing possible erythrocytes, Giemsa, ×243; *18*, close-up of cell-types out of Figure 16, most probably showing leucocytes, Giemsa, ×243.

FIGURE 19. Nonidentified organic structure with regular internal septations, parasitic infection? Giemsa, ×243.

FIGURES 20,21. Oblong, bacterial (?) elements in chainlike arrangement. Giemsa, ×400.

Instead of this, and with the aim of a more detailed diagnosis, biopsies of ulcerations I, II and III were investigated histologically. Besides the partly preserved epidermis, groups of cellular elements could be observed, which consisted of clusters of either spindle-formed, oval or circular cells (Figures 15,16). Erythrocytes and most probably leukocytic infiltrates were identified (Figures 17,18). In Figure 15, tissue surrounds the circular cell cluster (arrows). Most likely it is a vascular wall; therefore, the formation could be interpreted as a cross-section of blood vessels. Despite the application of diverse staining methods such as Ziehl-Neelsen, Gram, Giemsa, PAS, haematoxilin-eosin and Azan, it was impossible to get clear indications for the involvement of the ulcers by microorganisms relating to one of the discussed pathological conditions. One of the problems is that there is no verification of the antemortem origin of the wormlike, organic structure and bacterial (?) elements demonstrated in Figures 19–21. It is remarkable, however, that comparative, cutaneous examinations of the same subject as well as our studies of mummified skin preparations from other samples of the same mummy collection did not disclose parallel findings. Unfortunately, there are insufficient, satisfactory histopathological investigations of Egyptian mummies' soft tissues at the present time, so that an analysis of the above-mentioned findings cannot offer a conclusive, scientifically proven diagnosis. As in so many areas of Egyptian paleopathology, a systematic research program is recommended here (see below). Until then it will be difficult to refute such claims as those in Sandison's paper on infectious diseases in antiquity, that "macroscopic examination and radiographic studies are more likely to give useful information than histological preparations" (1972:222).

Conclusions

Compared with the clinical possibilities of dermatological or internal medicine conditions, the identification of pathologically caused soft tissue lesions in mummies represents simply a minimal diagnostic. In addition to absent color, the typical soft tissue character of the skin is lacking and the skin itself is more or less destroyed by postmortem decomposition, mummification substances, and often by long-term storage. The normal arrangement of layers, color and cell structure has often been altered. In a limited number of cases parasitic infection is demonstrated histologically. Nevertheless it remains a risky enterprise to diagnose soft tissue involvement resulting from any disease of nontraumatic and nonparasitic origin.

With caution, yet supported by the well-preserved and characteristic lesions, it was possible to arrive at a probable diagnosis. In the process it became increasingly clear that, except for one particular disease, the other mentioned conditions exhibit distinct differences relative to lesions I–IV. In light of the available criteria (particularly the typical configuration of the ulcers, their localization, and the exposure of skeletal parts) it seems highly probable that the cause of ulcerations in mummy 1565 is noma. The fact that noma occurs more often in children in present-day patients does not contradict the diagnosis, since adults are affected as well, although in fewer numbers. It can be assumed that noma was known in the Egypt of the pharaohs when one takes the living conditions of the masses into account. Because the harvest was dependent on the degree of Nile flooding, famines were common and contributed to one of the predisposing factors of phagedenic ulcer: malnutrition, poor hygienic conditions, and the accompanying immune deficiency.

Attempts to identify noma in written medical sources from Egypt have been undertaken by Ebbell (1939), who concluded that case 15 of the Edwin Smith surgical papyrus is a description of noma:

> Case 15 (6.14–17). Instructions concerning a perforation in his cheek: "If thou examinest a man having a perforation in his cheek, shouldst thou find there is a swelling, protruding and black, [and] diseased tissue upon his cheek, [conclusion in diagnosis].
>
> "Thou shouldst say concerning him: 'One having a perforation in his cheek. An ailment which I will treat.' Thou shouldst bind it with [ymrw] and treat afterward with grease [and] honey every day until he recovers."

From the viewpoint of modern clinical medicine, this presentation would seem to be too sparse to assign the respective symptoms—consisting of only three items: a hole in the cheek, a kind of swelling, and a black coloring—to the family of phagedenic ulcers, much less to the disease "noma."

James H. Breasted (1930), in his translation of the Smith papyrus, states that case 15 describes a traumatic perforation of the maxilla (according to Breasted, "cheek" is a designation for the maxillary bone, zygoma, and part of the temporal bone) resulting in an infection of the soft tissue wound involving swelling and blackening of the injured region. This could possibly be diagnosed as a type of gangrenous or necrotizing pyoderma. Due to its determinative, the word [ ] ('imrw) may denote a mineral substance which was part of the ancient Egyptian materia medica, perhaps a disinfectant (compare Grapow 1956:129). Thus, because many pathological aspects of case 15 remain ambiguous, the papyrus text does not confirm Ebbell's diagnosis sufficiently.

In spite of this, it seems rather certain that noma was well known in antiquity. In Book VI of "de medicina," A. Cornelius Celsus reports a disease which Patrick regards as noma and not as stomatitis aphthosa (Patrick 1967:243):

> But those ulcers, which the Greek call aphthae, are by far the most dangerous, that is, in children, for they often kill them; in men and women there is not the same danger. They begin at the gums, next possess the palate and the whole mouth, then descend to the uvula and fauces. When these are affected, it is not easy for the child to recover (A.C. Celsus, book VI, translation according to Greive 1756).

Finally, we cite an epigram of the Latin writer Martial, describing a disease which could have been noma:

> Canace, the daughter of Aeolis, lies buried in this tomb; little Canace, whose seventh winter was her last. Alas for the guilt and the crime of it! Thou, passer-by, who art quick weep, may lament here, not the shortness of life, but something sadder than death, the way death came. A dreadful canker wasted her face and settled on her tender mouth, and consumed her very lips before they were surrendered to the smoky pyre. If it had to come with so ill-timed a flight, fate should have come by another path. But death hastened to close the channel of her charming speech, lest her tongue might have power to bend the stern goddesses (Patrick 1967:243).

General remarks

We would first like to state that the ideas and considerations expressed here should not be understood as an appeal for a return to the descriptive style of pathology which, especially in the area of paleopathology, has fallen into discredit in the last few years. The aversion to it may have drawn its justification from the habit, common in the area of paleopathology, to merely describe "cases" without including any subsequent discussion of the global features of a particular disease (i.e., relating a single case to the frequency of a particular disease in a defined geographical region, or to project a certain disease against the background of all diseases endemic to a certain locality). Our understanding of the term "descriptive" is not limited to "merely describing" visible, exterior characteristics. In addition to comprehending "visual" or "macroscopic" phenomena, scientific description, as the word implies, also encompasses the entire range of what can be perceived, that is, what remains hidden to the unaided eye and can only be "seen" with the help of analytical methods. The case presented here from the field of soft tissue pathology provides an opportunity to refer to description as the *conditio sine qua non* of scientific work and interpretation. It is of fundamental importance for any individual as well as comparative study and essential for every experimental model. Contrary to the widespread misconception, however, description must be regarded as a *pars pro toto* and not as an exclusive whole. To be of any scientific relevance, it must necessarily serve a defined goal, strive for knowledge and, if it is not to become an end in itself, be complemented by interpretation.

The study presented above is by necessity a descriptive one. Using various methodological procedures, it is a selective description of "externa" as well as "interna." Due to the unique character of its findings, at least for the paleopathology of ancient Egypt, it conceivably could be concluded *at this point* and presented for subsequent discussion. Above all, this means that the opportunity to compare its findings with related anthropological cases is lost and creates problems in locating relevant, diagnostic information concerning the alleged disease "noma" in modern medical literature. In terms of a histopathological account of the findings, there is no unanimous consensus concerning the origin of microorganisms occasionally seen in the lesions. The question of whether these colonies of microorganisms represent secondary contamination or a primary colonization by a pathogen has yet to be answered satisfactorily.

In our study we have attempted to go beyond a merely descriptive method and by the process of elimination formulate a differential diagnosis based on the macroscopic appearance of the defects as well as additional information obtained from x-ray procedures and conventional histology. Furthermore medical texts from ancient Egypt relating to the clinical picture of "noma" were examined in detail. This resulted in a probable diagnosis and the emergence of new, fundamental questions relating to the field of soft tissue pathology. For our purposes the question of prime importance is the paradox that medical papyri from Egypt contain a large volume of direct and indirect reports concerning skin diseases or pathological skin manifestations, but the amount of anthropological evidence is very scarce indeed. To state it very simply, this means that although Egypt has provided more subjects for anthropological study than any other single culture, no more than a handful of dermatological clinical cases are known. What are the reasons for this pathetic situation which discourages and even handicaps us in formulating any concrete statements concerning an individual case other than a purely descriptive one? Are they due to insufficient technical-diagnostic possibilities? To an inadequate inspection of the subjects under study? To the state of preservation of organs or soft tissue? Or do the written records constitute an inappropriate yardstick for calculating the frequency of diseases which may have occurred to a much lesser extent than the historical sources suggest?

It is difficult to provide an explanation for this phenomenon. In our view, the primary reason for this is the fact that up to now qualified specialists have not conducted selective studies on the paleopathology of skin and soft tissue. Based on our own experiences during archeological excavations in Egypt the superficial examination of bodies (at least those which are found without wrappings) is not sufficient in itself. One must also conduct a precise inspection of darkened skin with definite questions in mind and employ all appropriate instruments. At the same time the necessary arrangements should be made concerning the taking of samples and their preservation until they reach the laboratory.

As another important aspect for future research in the field of soft tissue pathology (as well as in the area of osteopathology), we would like to consider ways to increase the effectiveness of specimen evaluation. In the past this has been carried out mainly by individuals without consulting a group of specialists which should have included not only paleopathologists, who are familiar with the characteristic properties and appearance of mummified soft tissue, but also clinicians. Such a committee should be made up of experts regardless of nationality. Criteria for their participation in diagnostic evaluations should be based on proven competence, prior experience in investigating mummified tissue, and the ability to apply methods best suited for a specific analysis. The publication of the results thus represents a joint effort of all persons and institutions involved. This proposal means that scientists should be willing to sacrifice their egotistical interests for the sake of group consultation, that is, that each individual recognizes the limits of his or her own capabilities when investigating a body and has the courage to delegate the corresponding tasks and decisions to those more authorized, regardless of geographical or political boundaries. This proposal requires explicit and defined game rules which nonetheless should not deviate too much from those unwritten laws of fairness and consideration already common among scientists in their dealings with one another. Yet it is also essential that the mammoth congresses of today be replaced by smaller, more comprehensible gatherings with the possibility for effective group work and discussion of current problems. It seems to us that the symposium "Human Paleopathology: Current Syntheses and Future Options" of this congress has taken the first steps in this direction and that this represents a refreshing contrast to similar congresses in the past.

The future tasks of paleopathology have become too varied and complicated to be left to the exclusive judgment of individuals. They require a cooperative plan which is considerably free of personal egos and which goes beyond that positive development generally referred to as "interdisciplinary" research. "Interdisciplinary" not only means consulting a certain specialist from an area outside one's own, but also calling in a specialist from one's own field of paleopathology. It not only means consulting the nearest specialist to solve a particular problem but also involves the necessary orientation toward the international arena, regardless of rivalries, for the sake of meaningful research. Only then will such combinations promise the optimal utilization of facts and methods on one hand, and the largest possible accumulation of knowledge, for the question at hand as well as for the entire field of study, on the other. What is needed is more concerted action and less insulation of interests in the hopes of acquiring personal fame.

The case we have presented represents an exhaustion of all applicable methods at our disposal. We do not feel completely qualified to apply immunological techniques such as those used successfully by Rothschild and Turnbull (1987) in their recent diagnosis of syphilis. We are also not sure how effective such methods would be in our case, that is, concerning nonspecific contamination. For this reason and in the spirit of the preceding proposal, we have presented our case for discussion in the hope of finding a more concrete basis for our submitted diagnosis of noma. Skin and soft tissue samples are available upon request. However, due to the limited quantity of research material, we reserve the right to select the applicants correspondingly.

Acknowledgments

The authors are indebted to Profs. E. Grosshans, Clinique Dermatologique, Strasbourg, and J. Maleville, Service de Dermatologie, Centre Hospitalier et Universitaire, Bordeaux, to D.H. Connor, M.D., Chairman of the Department of Infectious and Parasitic Diseases Pathology, Armed Forces Institute of Pathology, Washington, D.C. We also appreciate the help of M. Tomsky, Department of Dermatology, University of Tübingen, and S. Pääbo, Department of Cell Research, Wallenberg Laboratory, University of Upsala.

Literature cited

Braun-Falco, O., G. Plewing, and H.H. Wolff. 1984. *Dermatologie und Venerologie*. Berlin: Springer-Verlag.

Breasted, J.H. 1930. *The Edwin Smith Surgical Papyrus*, vol. 1: Hieroglyphic Transliteration, Translation and Commentary. Chicago: University of Chicago Press.

Connor, D.H., and R.C. Neafie. 1976. Tropical Ulcer. In C. Binford and D.H. Connor, eds., *Pathology of Tropical and Extraordinary Diseases*, vol. 1, 199–201. Washington, D.C.: Armed Forces Institute of Pathology.

Ebbell, B. 1939. Die ält-agyptische Chirurgie. Die chirurgischen Abschnitte der Papyrus E Smith und Papyrus Ebers. *Skrifter utgitt av det Norske Videnskaps-Akademi i Oslo, II. Hist.-Filos. Klasse*, No.2.

Grapow, H. 1956. *Kranker, Krankheiten und Arzt*. Berlin: Akademie-Verlag.

Greive, J. 1756. *A. Cornelius Celsus: Of Medicine*. Translated with notes critical and explanatory, in 8 books. Book 6. London.

Haneveld, G.T. 1974. An Egyptian Mummy of the New Kingdom with an Ulceration of the Leg. *Archivum Chirurgicum Neerlandicum*, 26(2):104–107.

Joseph, S.W., and J.F. Duncan. 1976. Noma. In C. Binford and D.H. Connor, eds., *Pathology of Tropical and Extraordinary Diseases*, vol. 1, 202–204. Washington D.C.: Armed Forces Institute of Pathology.

Kalkoff, K.W. 1981. Tuberkulose der Haut. In G.W. Korting, ed., *Spezielle Dermatologie*, Bd. II, 18.36–18.75. Stuttgart, Germany: G. Thieme.

Korting, G.W., and R. Denk. 1974. *Dermatologische Differentialdiagnose*. Stuttgart, Germany: Schattauer.

Luger, A.F. 1981. Syphilis. tiologie, Pathogenese, Klinik, Therapie und Prophylaxe. In G.W. Korting, ed., *Spezielle Dermatologie*, Bd. IV, 45.1–45.43. Stuttgart, Germany: G. Thieme.

Maleville, J. 1976. La Syphilis et les Tréponématosis Endémiques. Distribution Géographique et Écologie. *Les Cahiers d'Outres-Mer*, 113:7–17.

Pahl, W.M. 1986. Tumors of Bone and Soft Tissue in Ancient Egypt and Nubia: A Synopsis of the Detected Cases. *International Journal of Anthropology*, 1:267–276.

Patrick, A. 1967. Disease in Antiquity: Ancient Greece and Rome. In D.R. Brothwell and A.T. Sandison, eds., *Diseases in Antiquity. A Survey of the Diseases, Injuries, and Surgery of Early Populations*, 238–246. Springfield, Ill.: Charles C Thomas.

Rothschild, B.M., and W. Turnbull. 1987. Treponemal Infection in a Pleistocene Bear. *Nature*, 329:61–62.

Ruffer, M.A., and A.R. Ferguson. 1911. Notes on an Eruption Resembling That of Variola in the Skin of a Mummy of the Twentieth Dynasty (1200–1100 B.C.). *Journal of Pathology and Bacteriology*, 15:1–2.

Sandison, A.T. 1967. Diseases of the Skin. In D.R. Brothwell and A.T. Sandison, eds., *Diseases in Antiquity. A Survey of the Diseases, Injuries, and Surgery of Early Populations*, 449–456. Springfield, Ill.: Charles C Thomas.

______. 1972. Evidence of Infective Disease. *Journal of Human Evolution*, 1:213–224.

Tempest, M.N. 1966. Cancrum Oris. *British Journal of Surgery*, 53:949.

Zimmerman, M.R. 1977. The Mummies of the Tomb of Nebwenenef: Paleopathology and Archeology. *Journal of the American Research Center in Egypt*, 14:33–36.

SUMMARY OF AUDIENCE DISCUSSION: Some of the audience members questioned the nature of some histologic structures presented as lymphocytes, feeling their size and shape would more probably suggest they are bacteria.

Synthesis and conclusions

Arthur C. Aufderheide and Donald J. Ortner

Authors of the many excellent manuscripts presented at this symposium were asked to include a special focus on either evaluation of extant support for traditional interpretations ("current synthesis") or indentification of potential new areas of research or improved methods of doing current research ("future options"). Both the depth and the breadth of their response has been a source of special satisfaction to this conference's organizers. The discussed topics can be viewed from several perspectives, including the following:

1. IN WHAT WAYS CAN STUDIES IN PALEOPATHOLOGY CONTRIBUTE TO AN UNDERSTANDING OF THE ORIGIN AND TRANSMISSION OF DISEASES? Clearly, epidemiologic suggestions can be derived from the simple *establishment of the antiquity of a given disease*. In their respective articles, Brothwell traces measles to the Late Neolithic, and Manchester finds convincing descriptions of tuberculosis as early as the fourth millenium B.C. in Europe (Buikstra and Williams by at least A.D. 700 in the New World) and leprosy in Egyptian bones during the second century B.C. (Andersen traces textual evidence to the third century A.D.). Using art sources Dequecker identifies rheumatoid arthritis in paintings nearly three centuries before its description by Sydenham in 1676. Gerszten and Allison moved back the search for histologically documented primary cancer to a facial rhabdomyosarcoma in a South American mummy dated to about A.D. 500.

An alternative approach is embodied in Brothwell's suggestion that *zoonoses may reveal the evolutionary origin of diseases*. He notes that domesticates may transmit diseases (cattle: tuberculosis; birds: ornithosis; cats, sheep and pigs: toxoplasmosis). Furthermore, the dairying activities of early pastoralists exposed them to brucellosis, bovine tuberculosis, and a host of other conditions commonly resident in such animal populations. He suggests that the unravelling of parasite evolution could yield an unexpected harvest of information regarding hominid interactive behavior.

Manchester's meticulous dissection of the chronology of tuberculosis and leprosy prevalence in England demonstrates how paleopathology can provide data not retrievable by any other current methods: *disease interaction producing new disease patterns*. His study reveals how the crowded living conditions of medieval urbanism fueled a rapid rise in pulmonary tuberculosis, generating a population whose tuberculin-positive immune status crossreacted with the leprosy bacillus to suppress expression of the latter disease (an observation of potential therapeutic value in areas of current leprosy endemicity).

Integration of cultural information with physical evidence can both define and become predictive of behavioral aspects of disease susceptibility. Rose and Hartnady clearly spell out the tragic relationship of high infant mortality and infections among post-Reconstruction North American blacks and their socioeconomic misery with accompanying malnutrition. This process is mirrored also in Owsley's documentation of decreasing femur cortex thickness among midwestern Native Americans following contact, and in Goodman's demonstration of dental hypoplasia in disadvantaged subgroups of Mexican children. Kelley also notes that the socially disruptive effect of a major epidemic on an isolated population can, together with other aspects of their life style, be sufficiently profound so as to account for the documented high prevalence and mortality of such infections among Native Americans in the early colonial period without resort to a hypothesized *inherent* biological susceptibility greater than that of immigrating Caucasians.

These observations can spawn a host of research studies: Manchester's reported disease relationships need to be investigated in other locales, both archeological and in modern, living populations. Some of Brothwell's provocative suggestions can be tested with existing methods while evolving technology such as viral DNA probes may become sufficiently sensitive so as to permit tracing specific viruses through past millenia. Goodman's research model seeks evidence that dental enamel defects reflect serious health problems in a simultaneous study of both ancient and modern populations. A similar approach could be applied to test the observations of Kelley, Owsley, Rose and Hartnady, exploiting the fact that appropriately selected modern populations can permit assessment of socioeconomic status, malnutrition and other effects of interest more precisely than is possible in an ancient skeletal population alone.

2. The above observations also provide a partial and positive response to Pfeiffer's question: CAN PALEOPATHOLOGY BE PREDICTIVE OF CONTEMPORARY HEALTH PATTERNS? and vice versa. She points out, however, that there are presently

constraints inherent in the methods of paleopathology which prevent such studies from contributing *novel* data. Principal among these is the difficulty in defining the genetic homogeneity of an ancient population when studying the genetic component of a condition such as the "New World Syndrome" among native North Americans. Now that DNA can be extracted from archeological skeletal tissue and amplified (Tuross, pers. comm.) and with increasing availability of DNA probes for the highly polymorphic HLA system, it is conceivable some of these constraints may be ameliorated.

3. The anatomists' contention of an intimate RELATIONSHIP BETWEEN STRUCTURE AND BIOLOGICAL FUNCTION in normal tissues applies equally to diseased ones. While admitting frequent difficulties, Stirland suggests that enthesiopathies, hypertrophic crests, and skeletal response to repetitive microtrauma can be used to identify *occupationally induced changes* in appropriately selected populations. Martin notes the negative counterpart, pointing out that bone growth arrest secondary to severe matabolic stress is reflected in *histologic evidence* of increased skeletal remodeling with decreased calcification. Microradiography and radiation absorptiometric measurements of bone mineral density reflect similar changes. Both grossly evident and electron microscopical alterations are produced in the enamel of children's developing teeth when such metabolic insults occur in a pediatric population. Routine application of these methods, however, will become predictive at a useful and reliable level only when sufficient human clinical, animal, and paleopathological research studies have defined the precise sensitivity and specificity of these measures.

4. While paleopathology is often labeled a "young science," this symposium has revealed it is old enough to have developed an area of vulnerability sufficiently serious to threaten its potential for flourishing growth: LACK OF METHODOLOGICAL STANDARDIZATION. Since most skeletal collections are of small or modest size, prevalence data can only be computed for many conditions by combining multiple, independent reports. If the authors of the many articles so painstakingly reviewed by Gladykowska-Rzeczycka (documenting tumors in middle and eastern Europe) had all used a standardized reporting form which included a complete bone inventory and other vital data, then their value would have been considerably enhanced by making comparative, quantitative estimates possible. Perhaps even more important is the assumption that *paleopathological taxonomy* is equatable with clinical disease classification, when in fact the observational database of each is shared only in part with the other. The paleopathologist enjoys an unobstructed view of every bone in the skeleton, but lacks clinical symptomatology, soft tissue biopsy and autopsy information as well as results of chemical and metabolic studies. A significant contribution by this symposium is the unveiling of the need for an investigative development of a classification of bone alterations based solely on paleopathological observations, and then testing its utility by using it to develop a differential diagnosis list of diseases classified as clinical conditions.

An additional threat to the intellectual health of this field is the *lack of a general body of theory*, as noted in the introductory section of this volume.

5. The paleopathology database which is traditionally generated largely on the basis of gross skeletal structural features is now being enhanced by NEW AND SOMETIMES EXOTIC STUDY METHODS. Some of these promise to provide the type of data that has been restricted to date to biochemical studies of living individuals' blood samples. Imagine the informational legacy of infectious disease history which could be harvested if Tuross's reported *demonstration of immunoglobulin in bone extract* could be refined through further research into identification of bacteria-specific antibody! In the same manner, the first step in testing Angel's hypothesis of the interdependence of thalassemia and malaria in the Mediterranean would become possible if Ascenzi's *identification of hemoglobin's globin chain structure* could be established on a firm and reproducible basis. While less broadly applicable, Baud's and Kramar's studies dealing with *crystallographic structure* of tissue calcifications already have some etiological predictive value, for example, the presence of apatite and whitlockite predict a tuberculous cause of the calcifying lesion. Wider use of *bone histology*, both light microscopical and ultrastructural, would surely identify a broader range of features useful for its diagnostic applications.

Textual and artistic methods as reported here by Andersen, Chase, Dequecker, and Urteaga can make nonquantitative but specific contributions in spite of their inherent limitations of time and artistic license. Blackman et al.'s elegant reconstruction of a fatal sequence of renal pathophysiological events was made possible by the study of anatomic and chemical changes in *mummified soft tissues*. Newer radiological methods, especially *computerized tomography*, can provide information through uncluttered views of internal structure (Lewin), supplemented by density information which can be particularly helpful in studies of unwrapped human mummies. Investigations designed to determine *preservation of the various proteins of diagnostic interest* in different forms of mummification would be helpful (Aufderheides). Extraction of human DNA from an 8000-year-old brain by Hauswirth et al. promises exciting potential, especially now that the polymerase chain reaction (PRC) can amplify even the smallest quantities of recovered DNA to the point of reactivity with diagnostic nuclear probes. Many technical difficulties remain to be overcome, not the least of which constitutes concern for the degree of postmortem molecular damage with loss or substitution of various bases. Nevertheless, research directed at overcoming these problems is highly justified in view of the anticipated rich rewards, such as diagnostic screening for genes coding for congenital disease conditions (cystic fibrosis, Huntington's, and others already available), ability to demonstrate close genetic relationship through the HLA system, identification of specific infectious pathogens *(Mycobacterium tuberculosis, Salmonella* sp., *Legionella)* including retroviruses such as HIV and others. Completion of the present effort to map the entire human genome can be expected to expand these applications manyfold. All of these methods, however, are in their infancy. Collectively they will require an enormous amount of investigative effort before the contributions of their application have been defined at a useful level.

6. This symposium has also identified new evidence (or critically reviewed existing evidence) which suggests ALTERNATIVES TO THE TRADITIONAL VIEWS OF PATHOGENESIS for a variety of conditions. Stuart-Macadam identifies multiple observations inconsistent with the concept of *porotic hyperostosis* as a product of congenital anemia. She reminds us that a departure from the norm may be adaptive, when she points out the iron-deficiency anemia so common in milk-fed infants may have a protective effect against bacterial infections. While not denying the role of malnutrition in the production of certain *dental enamel defects*, both Dahlberg as well as Kocsis and Marcsik note that developmental defects may be responsible for others. Studies are needed to establish clear separation of these two, divergent pathogenetic mechanisms if interpretational confusion is to be avoided. Vyhnanek and Stloukal also note that accumulating evidence relating to the biological behavior of *transverse (Harris') lines* of bone growth arrest needs to be refined and incorporated into our interpretative process if we are to escape errors inherent in the relatively simplistic application common for this method to date. Kelley's postulate that environmental, not genetic, factors can explain the *high frequency and mortality of certain infectious diseases among native North Americans* also should be tested. This need not necessarily await development of sophisticated methods of DNA analysis. Clinical tuberculosis treatment records already exist which, if appropriately selected and studied, may be capable of identifying whether or not Native American tissue and immune responses to both the disease and treatment differ from those of Caucasians. Coupled with detailed epidemiological studies a database useful in evaluating this question may be possible.

Urteaga warns that the nasal destructive changes attributed to leprosy in Europe may be lacking in Amazonian lepers, and that such changes in the latter region can be duplicated by *leishmaniasis*. Brothwell notes the high probability that *brucellosis* was present among early European pastoralists and argues the need for studies which would define patterns of skeletal changes diagnostic for this disease, which could then be employed for its recognition in archeological skeletons. Roberts makes a good argument for her complaint that the physical evidence of *trauma* contains much more information than is usually derived from it by conventional studies. Her interdisciplinary approach appears to offer anthropologically useful information not only for reconstruction of circumstances leading to the injury and of treatment administered, but also for prediction of specific forms of subsequent dysfunction caused by the lesions.

While the references to *tuberculosis* by many symposium authors have been recorded above in other contexts, it may be useful to concentrate these concerns within this discussion. Buikstra and Williams's *simulation studies* suggest the disease should not have survived in North America under the conditions of their study and stipulations. It would be useful to repeat these, probably for different locales at different periods. Their suggestion that *alternative organisms* may produce identical skeletal changes may be testable with modern laboratory animal studies and perhaps in ancient skeletal tissue after some of the newer diagnostic methods (molecular biology?) discussed above become available. Such methods might also lend themselves very well to pursue the pathogenic role played by *atypical mycobacteria* although useful information could already be derived from thorough current environmental studies with conventional methodology. The ultimate availability of reliable nuclear probes for the various mycobacterial species and variants can be expected to permit studies directed at Brothwell's question *whether M. tuberculosis was derived from the avian or bovine strain*. In tracing the *antiquity of tuberculosis* such methods applied to the earlier examples of Egyptian skeletal lesions (Morse et al. 1964) would also be useful, as well as those of the South American mummies (Allison et al. 1973). Kelley and Micozzi's 1984 assertion that localized *rib periostitis reflects tuberculous empyema* needs to be verified, most easily in mummies where empyemas of both tuberculous and nontuberculous nature occur. Not only do Manchester's noted disease patterns resulting from interaction between tuberculosis and leprosy need to be checked in other geographical areas, but it would be useful also to design investigations for the evaluation of his assumption that during the Early Middle Ages *gastrointestinal tuberculosis* affected both village and urban areas equally because it was dependent upon herd, not human population size.

Paleopathologists have also been criticized for their reluctance to integrate their data with that generated by medical historians. Occasional examples exist which demonstrate its potential usefulness (Handler et al. 1986), and it is not difficult to visualize other possibilities.

In summary, these presentations succeed in achieving the symposium's goals: "Current synthesis and future options." The ultimate adoption of even a fraction of the new methods discussed can be expected to change the nature of paleopathologic study more during the coming decade than it has enjoyed during the past century. The editors hope publication of these manuscripts will serve as a useful guide and stimulant to future paleopathology investigations.

Literature cited

Allison, M.J., D. Mendoza, and A. Pezzia. 1973. Documentation of a Case of Tuberculosis in Pre-Columbian America. *American Review of Respiratory Disease*, 107:985–991.

Handler, J.S., A.C. Aufderheide, R.S. Corruccini, E.M. Brandon, and L.E. Wittmers Jr. 1986. Lead Contact and Poisoning in Barbados Slaves: Historical, Chemical and Biological Evidence. *Social Science History*, 10:399–425.

Kelley, M.A., and M.S. Micozzi. 1984. Rib Lesions in Chronic Pulmonary Tuberculosis. *American Journal of Physical Anthropology*, 65:381–386.

Morse, D., D.R. Brothwell, and P.J. Ucko. 1964. Tuberculosis in Ancient Egypt. *American Review of Respiratory Disease*, 90: 524–541.

Participants

Marvin J. Allison
Medical College of Virginia
Richmond, VA 23298

Johs G. Andersen
Braineparken 85, st. th.,
DK-6100 Haderslev, Denmark

A.J.G. Araujo
National School of Public Health
Oswaldo Cruz Foundation
Rio de Janeiro, Brazil

Antonio Ascenzi
Dipartimento di Biopatologia Umana
Sezione di Anatomia Patologica
Universitá "La Sapienza"
Policlinico Umberto I
Viale Regina Elena 324
I-00161 Rome, Italy

Arthur C. Aufderheide
Department of Pathology
University of Minnesota-Duluth
School of Medicine
Duluth, MN 55812

Mary L. Aufderheide
Paleobiology Laboratory
University of Minnesota-Duluth
School of Medicine
Duluth, MN 55812

C.-A. Baud
Universite de Geneve
Department D'Anthropologie
12, Rue Gustave-Revilliod
1227 Carouge-Geneve, Switzerland

A. Bellelli
Dip. di Scienze Biochimiche
Universitá "La Sapienza"
Rome, Italy

Pia Bennike
Institute of Medical Anatomy B
University of Copenhagen
Blegdamsvej 3
DK-2200 Copenhagen N, Denmark

James Blackman
Associate Director
Family Practice Residency Program
777 North Raymond St.
Boise, ID 83704

Don R. Brothwell
Institute of Archaeology
University of London
31-34 Gordon Square
London, WC1H OPY, England

M. Brunori
Dip. di Scienze Biochimiche
Universitá "La Sapienza"
Rome, Italy

Jane E. Buikstra
Department of Anthropology
University of Chicago
1126 E. 59th St.
Chicago, IL 60637

M. Chame
National School of Public Health
Rio de Janeiro, Brazil

Debra A. Chase
Department of Near Eastern Languages
& Civilizations
Harvard University
Cambridge, MA 02138

G. Citro
Instituto Regina Elena per lo Studio
e la Cura dei Tumori
Rome, Italy

Ulisses E.C. Confalonieri
National School of Public Health
Oswaldo Cruz Foundation
Rio de Janeiro, Brazil

Albert A. Dahlberg
Department of Anthropology
Zoller Memorial Dental Clinic
University of Chicago
Chicago, IL 60637

Jan Dequeker
Arthritis and Metabolic Bone Disease
Research Unit
K.U. Leuven, U.Z. Pellenberg
B-3041 Pellenberg, Belgium

Cynthia D. Dickel
Department of Immunology and Medical
Microbiology
Box J-266 JHMHC
College of Medicine, University of Florida
Gainesville, FL 32610

David N. Dickel
Department of Anthropology
Florida State University
Tallahassee, FL 32306

Glen H. Doran
Department of Anthropology
Florida State University
Tallahassee, FL 32306

Howard Duncan
Division of Rheumatology
Henry Ford Hospital
Detroit, MI 48202

L.F. Ferreira
National School of Public Health
Oswaldo Cruz Foundation
Rio de Janeiro, Brazil

Enrique Gerszten
Department of Pathology, Box 662
Medical College of Virginia
Richmond, VA 23298

Judyta Gladykowska-Rzeczycka
Department of Anatomy
Academy of Physical Education
ul. Wiejska 1
80-336 Gdansk, Poland

Alan H. Goodman
School of Natural Sciences
Hampshire College
Amherst, MA 01002

Philip Hartnady
Department of Anthropology
University of Arkansas
Fayetteville, AR 72707

William W. Hauswirth
Department of Immunology and Medical
Microbiology
Box J-266 JHMHC
College of Medicine, University
of Florida
Gainesville, FL 32610

R. Ippoliti
Dip. di Scienze Biochimiche
Universitá "La Sapienza"
Rome, Italy

Robert D. Jurmain
Department of Anthropology
San Jose State University
125 South Seventh St.
San Jose, CA 95192

Marc A. Kelley
Paleobiology Laboratory
University of Minnesota-Duluth
School of Medicine
Duluth, MN 55812

Gábor Kocsis
Department of Dentistry and Oral Surgery
Albert Szent-Györgyi University
Medical School
6701-Szeged, Hungary

Christiane Kramar
Departement d'Anthropologie
12 rue Gustave-Revilliod
1227 Carouge-Geneve, Switzerland

Philip J. Laipis
Department of Biochemistry
and Molecular Biology
Box J-245 JHMHC
College of Medicine, University
of Florida
Gainesville, FL 32610

James C.C. Leisen
Division of Rheumatology
Henry Ford Hospital
2799 W. Grand Blvd.
Detroit, MI 48202

E. Lendaro
Dip. di Scienze Biochimiche
Universitá "La Sapienza"
Rome, Italy

Peter K. Lewin
Hospital for Sick Children
555 University Ave.
Toronto, Ontario, M5G 1X8, Canada

Keith Manchester
Undergraduate School of Studies
in Archaeological Sciences
University of Bradford
Bradford West Yorkshire BD7 1DP,
England

Antonia Marcsik
Department of Anthropology
Attila Jozsef University
P.O. Box 586
Egyetem-u. 2
6701-Szeged, Hungary

Debra L. Martin
School of Natural Science
Hampshire College
Amherst, MA 01002

Juan R. Munizaga
Universidad de Chile
Santiago, Chile

Norman Oldroyd
Laboratory Supervisor
Louis C. Herring and Company
Orlando, FL 32802

Donald J. Ortner
Department of Anthropology
National Museum of Natural History
Smithsonian Institution
Washington, D.C. 20560

Douglas W. Owsley
Department of Anthropology
National Museum of Natural History
Smithsonian Institution
Washington, D.C. 20560

Wolfgang Michael Pahl
Institut für Anthropologie
und Humangenetik
Universität Tübingen
Wilhelmstr. 27, 7400 Tübingen
West Germany

Susan Pfeiffer
School of Human Biology
University of Guelph
Guelph, Ontario N1G 1W1, Canada

Mary Lucas Powell
Museum of Anthropology
University of Kentucky
Lexington, KY 40506-0024

Graeme L. Pretty
South Australian Museum
Adelaide, Australia

Miroslav Prokopec
Institute of Hygiene and Epidemiology
Prague, Czechoslovakia

B.M. Ribeiro Filho
National School of Public Health
Oswaldo Cruz Foundation
Rio de Janeiro, Brazil

J.M. Riddle
Division of Rheumatology
Henry Ford Hospital
Detroit, MI 48202

Charlotte Roberts
School of Archaeological Sciences
University of Bradford
Bradford West Yorkshire BD7 1DP,
England

Jerome C. Rose
Department of Anthropology
University of Arkansas
Fayetteville, AR 72701

R. Ted Steinbock
X-Ray Associates of Louisville
Suite 117, Mall Office Building
400 Sherburn Lane
Louisville, KY 40207

Ann Stirland
University College, London
The Cottage, Lower Green, Woodend,
Towcester, Northants NN12 8SB, England

Milan Stloukal
Department of Anthropology
National Museum
Prague, Czechoslovakia

Eugen Strouhal
Naprstek Museum
Section of the National Museum
Betlemske n. 1,
11000 Prague 1, Czechoslovakia

Patty Stuart-Macadam
Department of Anthropology
University of Toronto
Toronto, Ontario M5S 1A1, Canada

Takao Suzuki
Department of Epidemiology
Tokyo Metropolitan Institute
of Gerontology
35-2 Sakae-cho, Itabashi-ku
Tokyo 173, Japan

James M. Tenney
Research Associate
Lowie Museum of Anthropology
Berkeley, CA 94720

Noreen Tuross
Conservation Analytical Laboratory
Smithsonian Institution
Suitland, MD 20746

Oscar Urteaga-Ballon
Apartment 905
11 Island Avenue
Miami Beach, FL 33139

W. Undeutsch
Hautklinik der Universität Tübingen
Histologisches Labor
Liebermeisterstr. 25, 7400 Tübingen
West Germany

Lubos Vyhnanek
Radiological Clinic
Medical Faculty
Charles University
U nemocnice 2
128 08 Praha 2, Czechoslovakia

Sloan Williams
Los Alamos National Laboratory
Life Sciences Division
Box 1663
Los Alamos, NM 87545

Joseph Zias
Israel Department of Antiquities
and Museums
Jerusalem, Israel

R. Zito
Instituto Regina Elena per lo Studio
e la Cura dei Tumori
Rome, Italy

SMITHSONIAN INSTITUTION LIBRARIES
3 9088 00911 7060

ISBN 1-56098-039-7