

The Excavation of Khok Phanom Di

A Prehistoric Site in Central Thailand

Volume V: The People



By
N.G. Tayles

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The Excavation of Khok Phanom Di

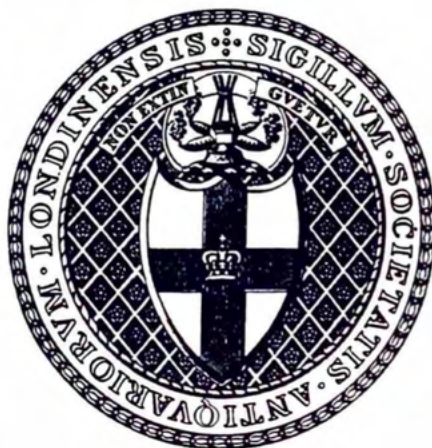
a Prehistoric Site in Central Thailand

Volume V : The People

By

N.G. Tayles, B.A., Ph.D.

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PREFACE

KHOK Phanom Di is a large prehistoric site located in Chonburi Province, Central Thailand. It was occupied between approximately 2000–1500 BC, at a time when the sea level was higher than at present, and the occupants experienced a series of significant environmental changes due to sedimentation and an oscillating sea level. The range of biological and material finds was considerable, and was augmented by the flotation of cultural samples from the beginning to the end of the sequence. Hence, we have an unusually clear picture of the conditions within which the twenty or so generations lived. The mound accumulated rapidly to its ultimate height of about 12 metres, largely as a result of vigorous cultural activity in pottery making and the consumption of shellfish. In consequence, we encountered an unparalleled mortuary assemblage in which the dead were interred successively over their ancestors. The pattern of the cemetery also includes interment in a number of distinct clusters.

The excellent preservation of organic material, from the wood in postholes to partially digested food, coprolites and human bone, provide a rare opportunity to trace interaction between the people who lived at this site and their environment. Nancy Tayles presents the portrait of a community living in one of the world's richest habitats in terms of biological vigour, but which suffered significant infant mortality. One of the great advantages conferred by the data, is the control over time. She has minutely traced the pattern of health, showing how early men had powerful upper bodies, but then became smaller and less muscular. The changing environment, which saw an estuarine hunter gatherer community adopt local rice cultivation, and then again turn to gathering and fishing with the return of saline conditions, is traced through oral disease and tooth wear. The physical appearance of the people, the fecundity of the women, the nature of the diet and the life span of individuals are all vital components of any attempt to synthesise this society. We are deeply indebted to the dedication and the intense scholarship which Dr Tayles has brought to this study, which in our view sets a new standard in the consideration of the people not only of this site, but of South East Asia as a whole.

I. INTRODUCTION

IN 1987 Pisit Charoenwongsa, Director of the Research Section (Archaeology) of the Royal Thai Fine Arts Department, listed his assessment of the major deficiencies in current knowledge of Thai prehistory. The list included the following comment:

We must make progress in solving the problem of the long-term genetic and medical history of human populations in Thailand. Much physical anthropological data from excavated skeletal material is now available, but we still cannot describe prehistoric populations in this country with any confidence. (Charoenwongsa 1987:87)

This statement indicates that there is a recognised need for continued research on the biology of prehistoric South East Asians. Most of the reports available at that time had the principal research aim of determining the genetic relationships between South East Asians and other populations from Asia and the Pacific (e.g. Pietrusewsky 1974a, 1978) or describing the demography of the skeletal collections (Pietrusewsky 1974c, 1984).

Skeletal biology reflects the complex interaction of genetics and environment and as such, in addition to the genetic composition of a population, the effects of subsistence mode, social structure, environment, and survival success are all potentially discernible. Skeletal material permits the reconstruction of a dynamic impression of the people represented, both as individuals and as a community. The biocultural approach to the analysis of human skeletal remains involves the integration of all data to form a comprehensive overview of the biology of the population, rather than being viewed as a set of independent characteristics. As an example, the importance of skeletal pathology lies not in its presence on a particular bone, but in the implications for the health of the individual, and, where the pathology indicates nutritional inadequacies or exposure to infection, the health of the population. Some bone pathology is easily recognisable, resulting in its overemphasis. A well-healed simple fracture of a small bone, for example, is unlikely to have represented more than a temporary, painful, inconvenience for the individual but is of minor importance in understanding the biology of the population, whereas a pattern of similar fractures in a population may indicate widespread involvement in a hazardous activity.

There has been a history of research on human skeletal remains being treated as if the individuals represented were somehow separate from the site itself, with the reports appended to the main analyses. The people who lived at a site and were responsible for producing the artefacts and modifying the environment are central to the interpretation of all data. The primary evidence for past populations is the people themselves. The integration of evidence from human biology with environmental and cultural information is essential. The extent and complexity of analysis and interpretation from a skeletal collection will be determined by the completeness and condition of the material and the information available from associated spatial, artefactual and environmental data.

This volume is based on research carried out in the Departments of Anatomy and Anthropology at the University of Otago between 1987 and 1992, using the skeletal remains of the sample of

the people from the prehistoric South East Asian site of Khok Phanom Di, Central Thailand. The principal research aim was to use the approach outlined above to contribute to an understanding of prehistoric South East Asians by addressing the question "what were the lives of the people like?". This global question was approached through an examination and explanation of patterns of health and disease and the interpretation of this evidence for the quality of life of the population and their survival success. The research was presented as a thesis in fulfilment of the requirements for the degree of Doctor of Philosophy at the University of Otago, Dunedin, New Zealand, in 1992. This volume is based almost entirely on that thesis, with some updating of references and additional work undertaken in subsequent years. The latter provided the basis for the physical description of the people in particular.

A previous project examined the metric and non-metric dental and skeletal phenotypic features, to investigate biological relationships within the group and ascertain the pattern of genetic homogeneity or heterogeneity (Choosiri 1988, 1992). A series of smaller research projects, covering metacarpal cortical thickness as evidence of health (McGill 1986), shovel-shaped incisors (Roy 1986) and pelvic evidence for pregnancy and fecundity (Pollock 1987) were also undertaken by graduate students. These have been superseded by work incorporated in the current report where appropriate.

The research does not cover all aspects of biology of the Khok Phanom Di people. The number of skeletons recovered from the excavation and the quality of preservation, both in terms of completeness of individual skeletons and of condition of the bone tissue, is such that the potential of the collection is vast. The work which has been undertaken so far does not represent a definitive description of the population but this report is a presentation and interpretation of the results so far.

THE SITE OF KHOK PHANOM DI

The site of Khok Phanom Di was excavated in 1985 as part of a major research project in the Bang Pakong Valley. This is a long-term joint project between the Archaeology Division of the Fine Arts Department of Thailand and the University of Otago, with the aim of tracing the course of prehistoric and early historic human adaptation and settlement in the area. The Bang Pakong Valley was chosen as an example of adaptation to a coastal, estuarine environment, as a contrast to previous research in Thailand which had focussed on inland sites. Details of the research project, the excavation, mortuary practices, the environment and the material culture of the people are given in previous volumes of this series (Higham and Bannanurag 1990, 1991, Higham and Thosarat 1993, Thompson 1996). These are briefly summarised here.

Khok Phanom Di is located c. 100 km south east of Bangkok and 22 km from the coast of the Gulf of Siam (Fig. 1). It is a 12 m high mound, c. 5 ha in size, standing in rice fields in the vicinity of the Bang Pakong River. A Buddhist temple complex occupies the centre of the mound and there are a few houses around the sides. There are also trees and garden crops growing on the mound, virtually all of which is culturally deposited (Aitken 1993).

The excavation was sited close to the centre of the mound, although not at its highest point (Fig. 2). This site was chosen for its proximity to an earlier, smaller excavation (Pisnupong 1984)

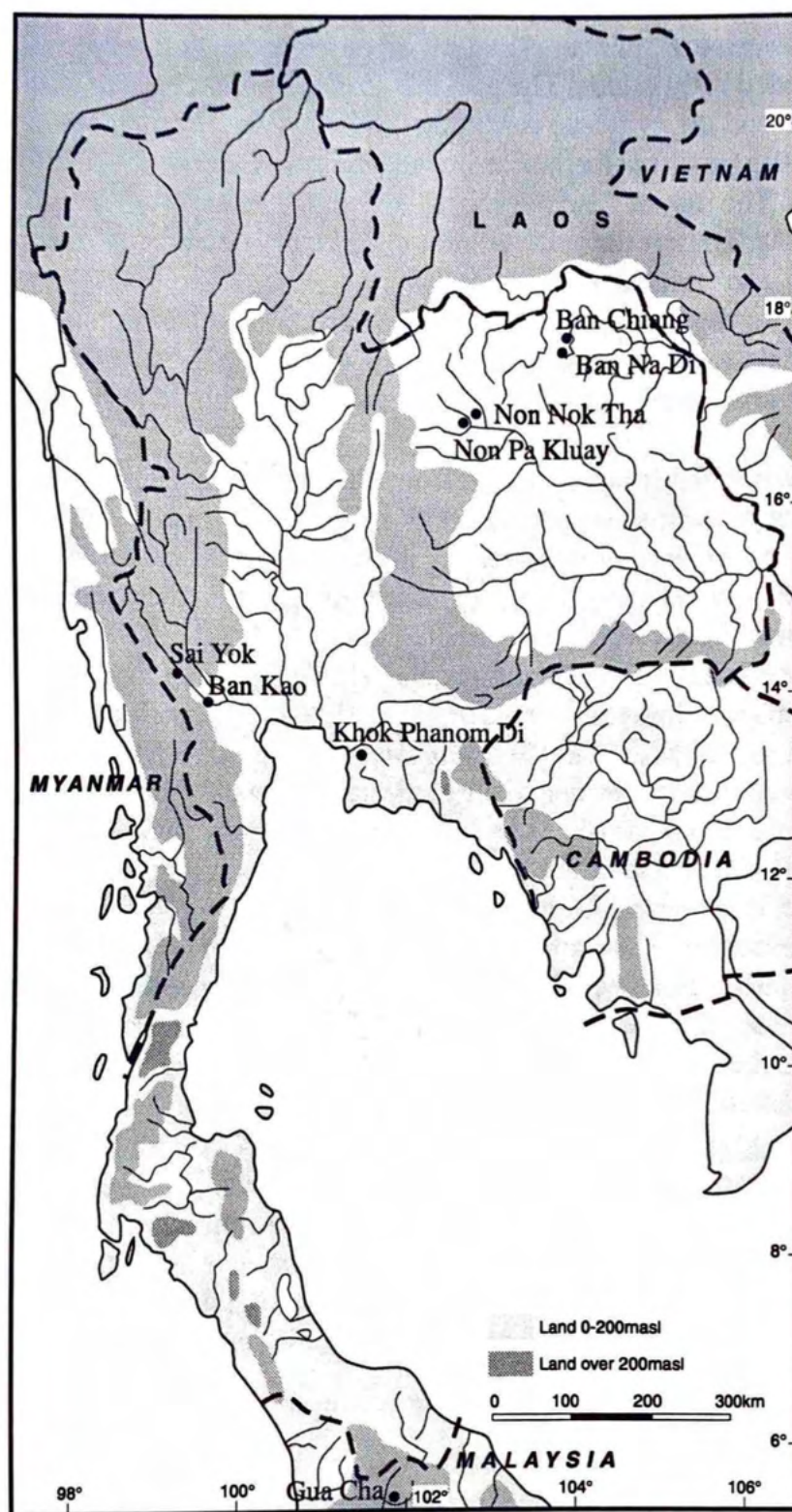


FIG. 1. Map of South East Asia, showing the location of Khok Phanom Di and other prehistoric sites referred to in this volume (adapted from Higham and Bannanurag 1990)

and because the area did not interfere with the temple complex. Earlier excavations (Noksakul 1981, Pisupong 1984) recovered 22 human skeletons among other cultural material but the skeletal remains were not included in this study. The previous excavations were sited to the north and south of the major excavation and the evidence from these, together with some human bone exposed in an earlier road cutting, suggests that the burials extended further across the site for at least part of the period of occupation. The mound, and therefore the depth of cultural deposits, varies in height from seven to twelve metres. The depths and duration of cemetery deposits, as distinct from occupation and industrial debris, are variable and unknown.

The upper 1.2 m of deposit in the 10 x 10 m excavation square contained occupation debris without burials. Burials were distributed through the remaining 5.6 m of deposit. Details of excavation methods and burial distribution are given in Volume I of this series (Higham and Bannanurag 1990).

Eighteen radiocarbon dates are available from charcoal samples. The midpoints of the series of corrected dates range within the period 2000–1500 BC and include almost the full period of the burials, with the upper deposit undated. As the ranges of the individual dates extend over 4–700 years, absolute dating of individual deposits or burials within the deposits is not possible. The superpositioning of interments without disturbance of underlying skeletons, together with the build-up of non-burial deposits has, however, allowed the relative dating of the burials. The chronological ordering of the burials was based on stratigraphic superposition, including the level of the top of the grave cut, which was identified in almost all cases.

The molluscan and botanical evidence suggests that during the initial period of occupation Khok Phanom Di was near a major estuary (Mason 1991, Thompson 1996). The sedimentological evidence suggests that it was on a distributary (Aitken 1993). The first occupants of the site had access to marine, mangrove, riverine and freshwater swamp habitats. This combination offered a rich variety of quality food resources. The abundance of fishhooks and net floats, together with the nature of biological remains, indicates an economy centred on fishing and collecting. The evidence for diet is discussed in more detail in chapter VII. Environmental changes which occurred during the period of occupation of the site are discussed in Mason (1991), Aitken (1993) and Thompson (1996). Although the exact nature or cause of the environmental changes have not been determined, during the middle of the period of use of the cemetery the sea regressed, with loss of ease of access to the resources of the river and coast. At this period, there is material evidence for local rice cultivation in the form of granite hoes and shell harvesting knives. Towards the end of the use of the cemetery, marine resources again became available and material evidence for rice cultivation ceased. The inter-related changes in the environment, subsistence, social organisation and material culture over time will be fully described in the final volume of this series.

Mortuary practices

The condition of both the graves and the skeletons, and the disposition of burials has allowed the temporal and spatial variation in mortuary practices to be studied in detail. Seven mortuary phases (MP 1–7) have been identified on the basis of variation in the cultural evidence such as mortuary practices, the stratigraphic relationships of the burials, and non-burial deposits. The number and density of interments varied over time, as listed in table 1, and the disposition of burials in each

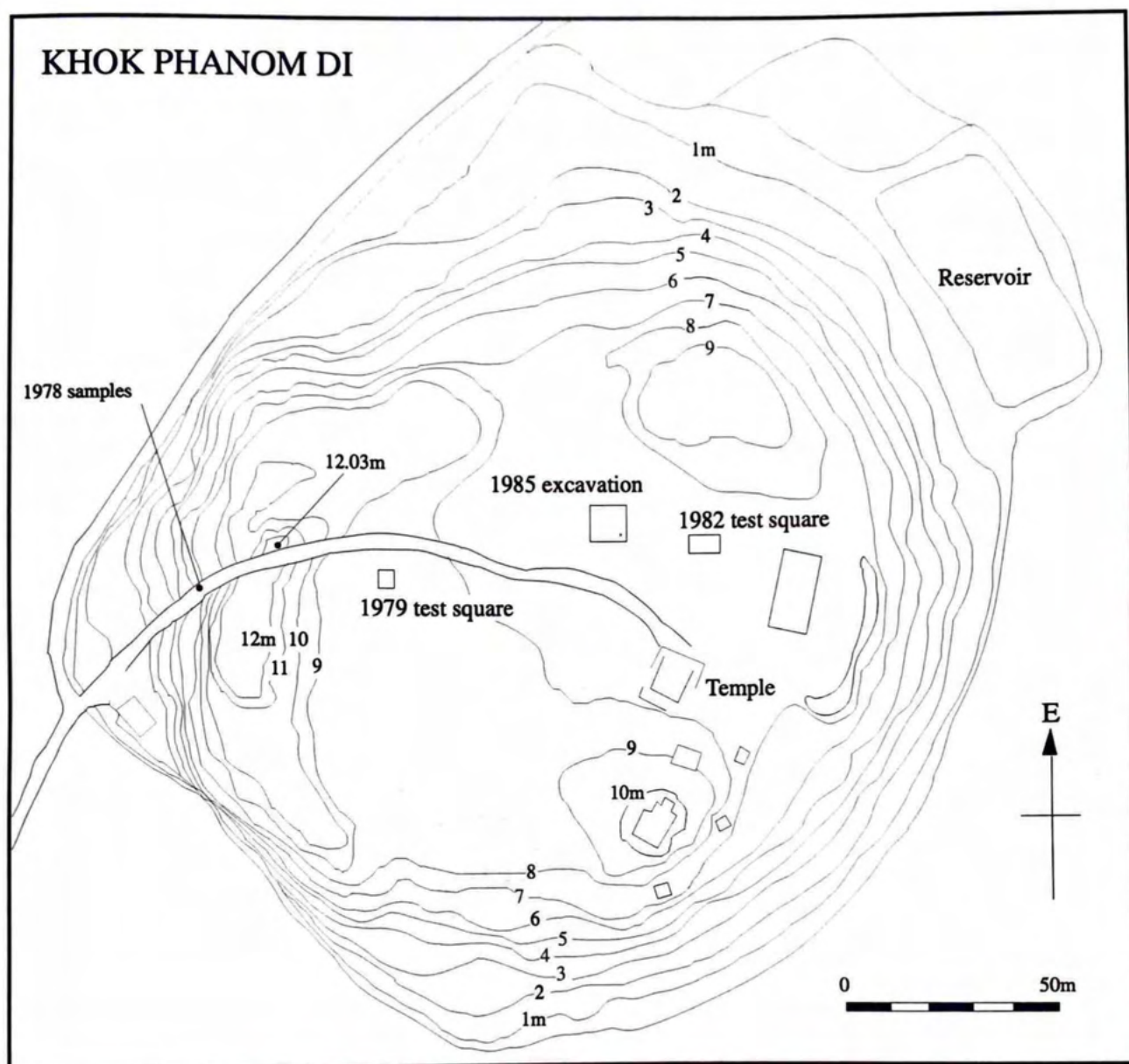


FIG. 2. Plan of Khok Phanom Di, showing the areas excavated (Higham and Bannanurag 1990)

mortuary phase is illustrated in figures 3–9. Stratigraphic relationships are summarised in figure 10. Mortuary practices are described in detail in volume I of this series (Higham and Bannanurag 1990).

The six burials of MP1 were scattered within the lowest 1.2 m of occupation debris. These consisted of a series of lenses of ash and midden, with numerous small hearths and food debris in the form of animal bones and shellfish. There was also evidence of pottery manufacture in the form of

TABLE 1: *Approximate depth of deposits and number and density of burials by mortuary phase*

MP	Depth of deposits (m)	Number of burials	Density burials per m ³
1	1.40	6	0.04
2	0.90	56	0.62
3	1.10	42	0.38
4	1.25	29	0.23
5	0.25	4	0.16
6	0.80	12	0.15
7	0.30	5	0.16
Total	6.00	154	0.26

ash spreads, burnishing pebbles and anvils. Other artefacts found included stone adzes and fishing gear.

Subsequently, the area appears to have been used primarily as a cemetery, with the deposition of the burials being more orderly. During MP2-4 the burials were concentrated in specific areas. For analytical purposes, these concentrations, some of which extended vertically through all three phases, have been identified as 'Clusters' A-G (Higham and Bannanurag 1990). The deposits around the burials and between the clusters contained middens, evidence of fires, pits and postholes. Some of these were disposed in such a way as to suggest that they were the result of activities associated with the burial ritual. In other cases the midden lenses appear to have banked up against a barrier or structure around the graves. The possibility that the deposits represent occupation debris rather than evidence of ceremonial activities has not been discounted, as they also contain artefacts such as fishing gear and stone and shell tools associated with every-day life. The deposits during this period represent a continuum, and the subdivision into three mortuary phases is made on the basis of relatively minor variations in the composition of the non-burial deposits and in mortuary practices, together with a build-up of deposits without graves. These may represent either brief cessations in the use of the cemetery or a relatively rapid deposition of material, giving the appearance of a hiatus in mortuary activity. MP3 is distinguished from MP2 by a hiatus in burials of 10 cm and by greater complexity in the non-burial features. MP3 and MP4 were separated by a 40 cm burial hiatus, and the deposits in MP4 contained less concentrated midden than MP3. The major environmental change occurred at about the time of the MP3-4 hiatus.

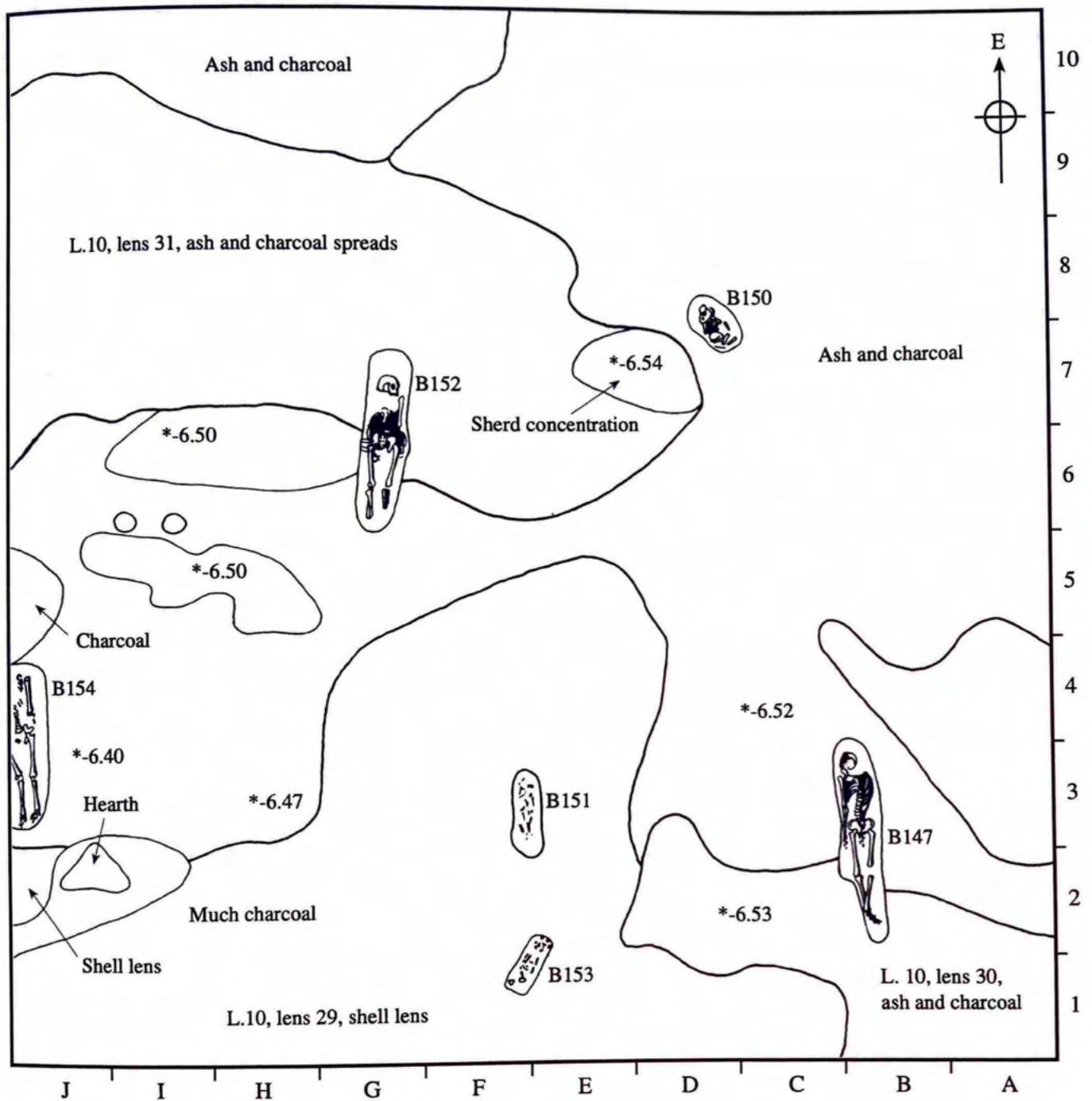


FIG. 3. The distribution of burials and features in MP1. Scale in metres. (Higham and Bannanurag 1990)

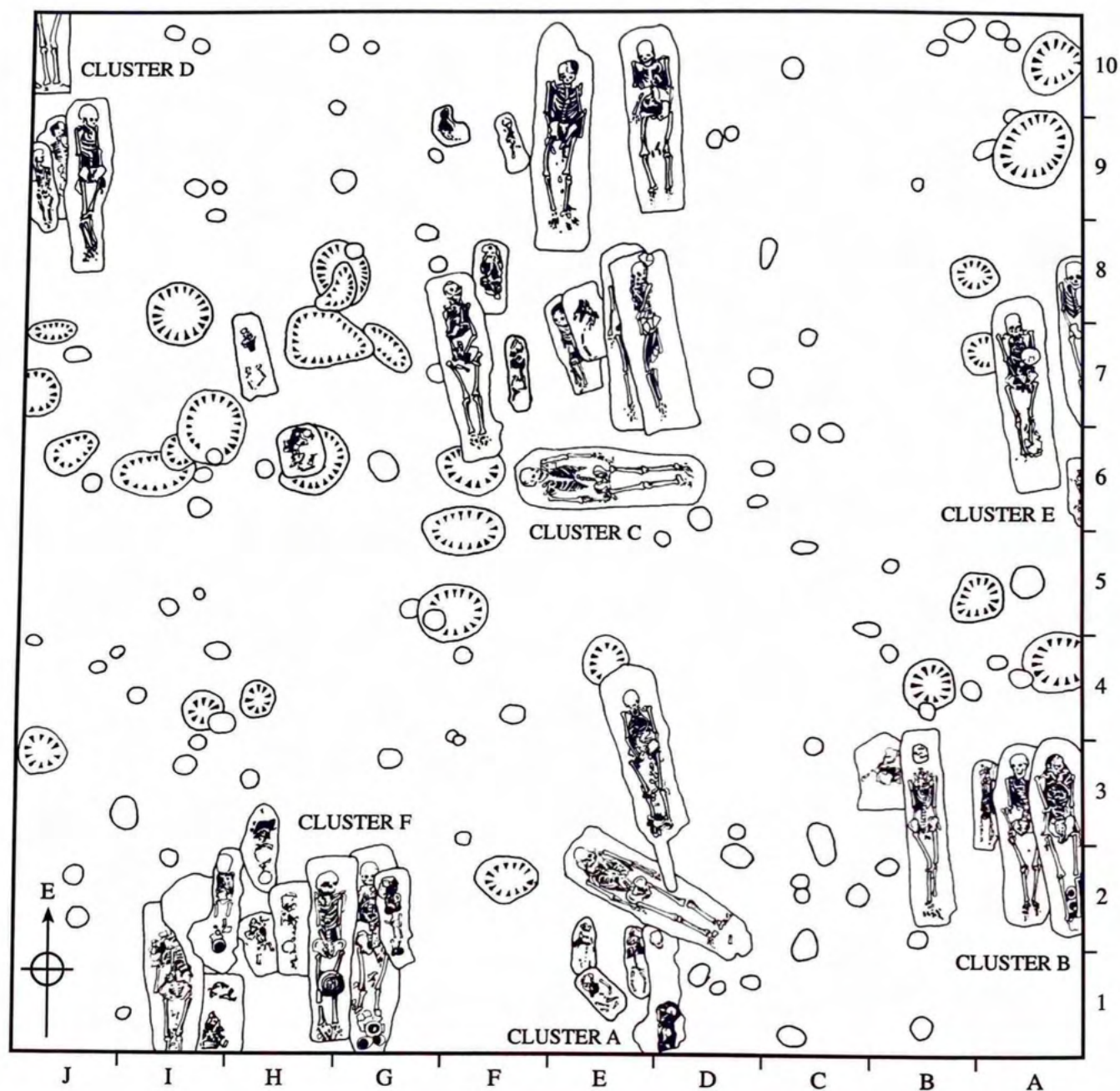


FIG. 4. The distribution of burials and features in MP2. Unfilled circles are postholes or areas of disturbance. Pits are hachured. Scale in metres. (Higham and Bannanurag 1990)

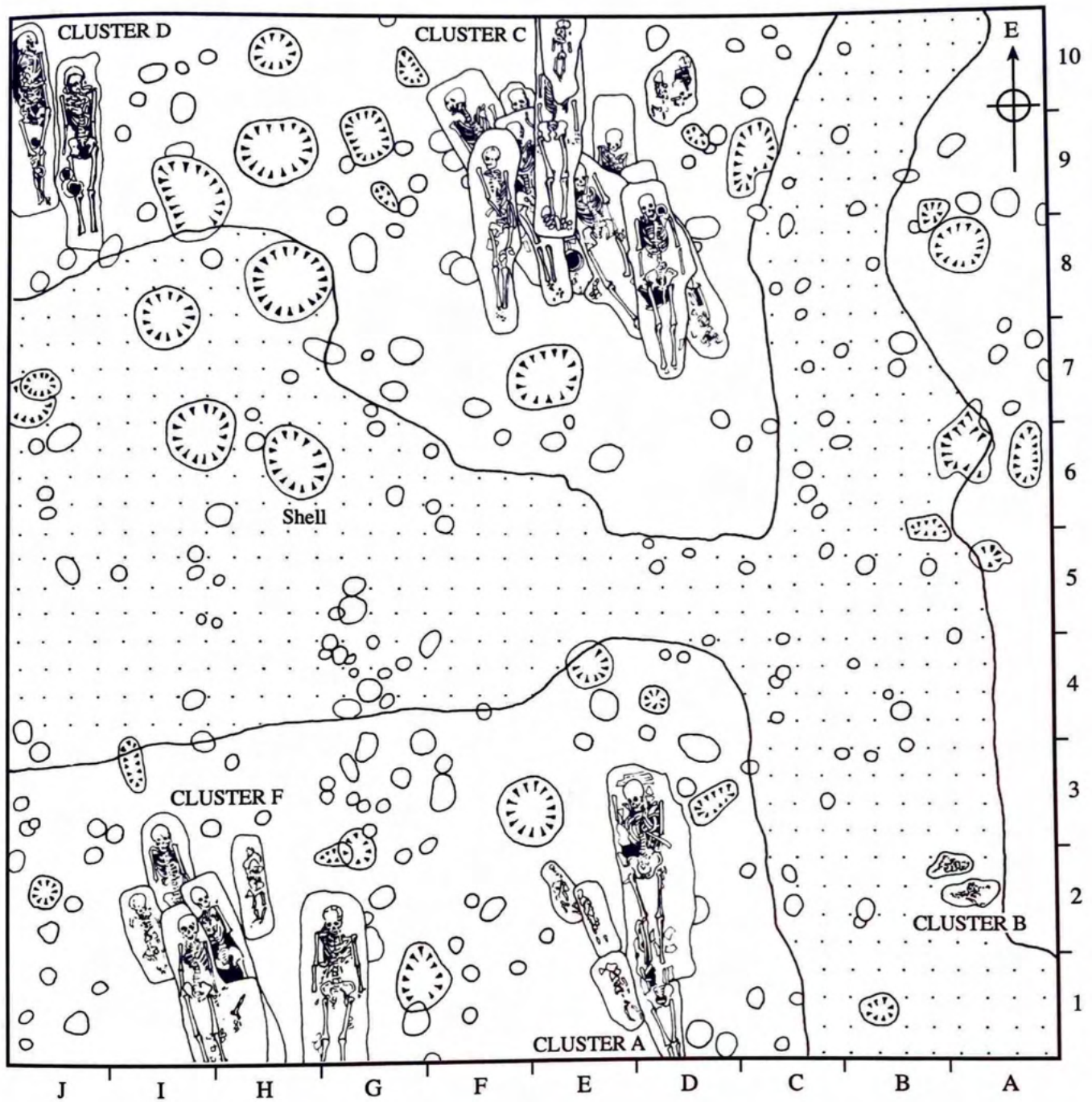


FIG. 5. The distribution of burials and features in MP3. Unfilled circles are postholes or areas of disturbance. Pits are hachured. The stippled area is a shell lens. Scale in metres. (Higham and Bannanurag 1990)

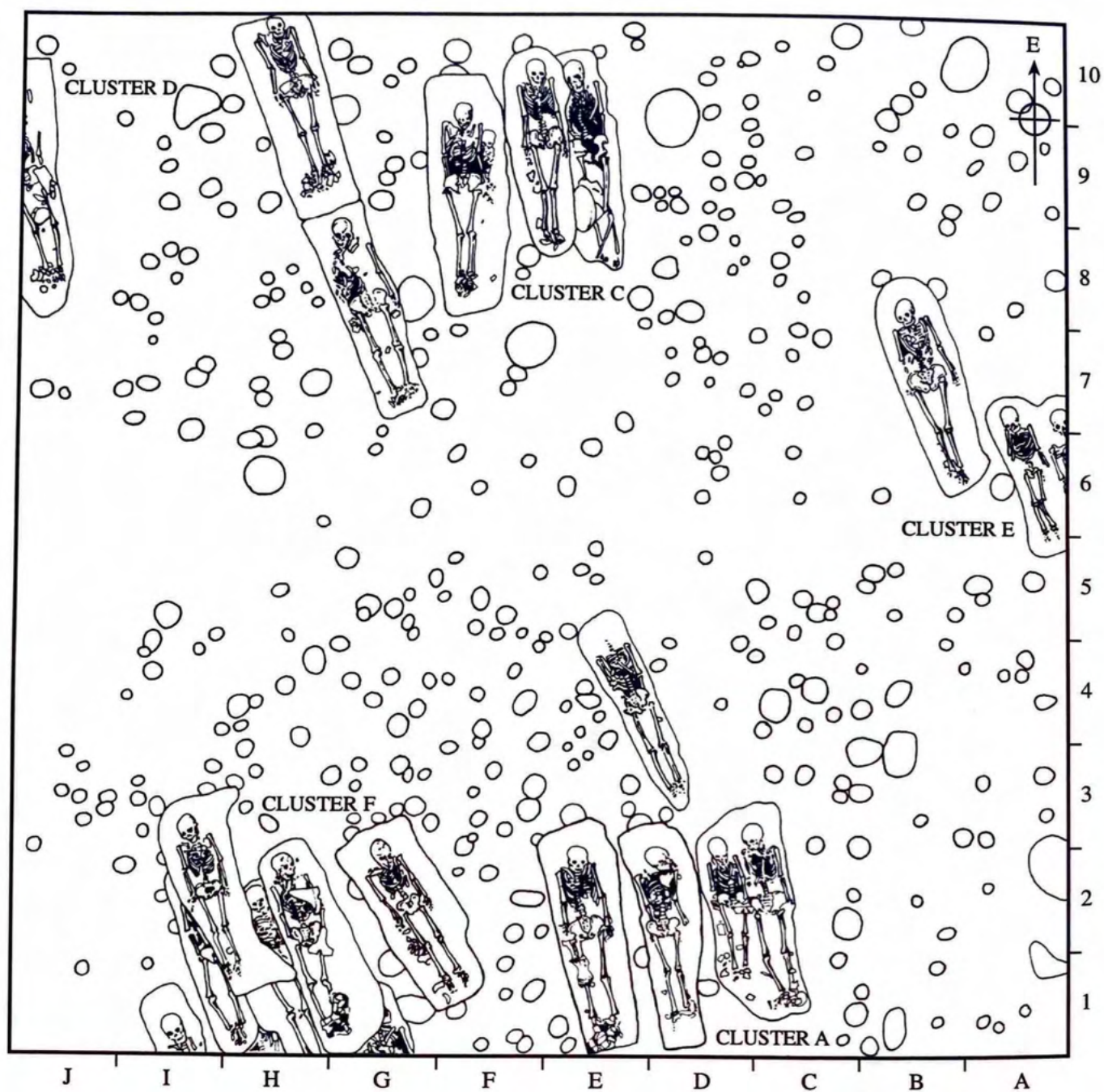


FIG. 6. The distribution of burials and features in MP4. Unfilled circles are postholes or areas of disturbance. Pits are hachured. Scale in metres. (Higham and Bannanurag 1990)

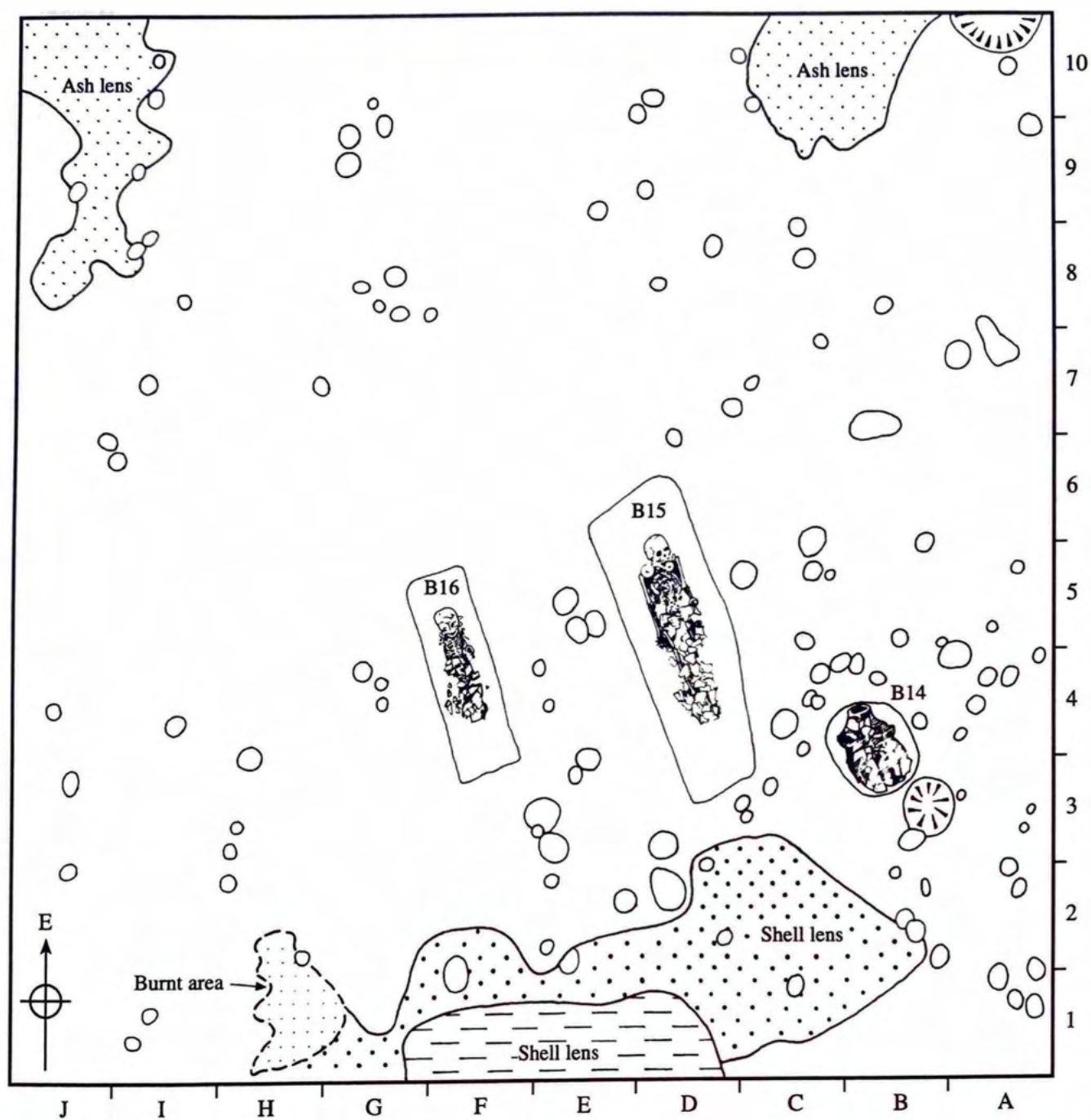


FIG. 7. The distribution of burials and features in MP5. Unfilled circles are postholes or areas of disturbance. Pits are hachured. Scale in metres. (Higham and Bannanurag 1990)

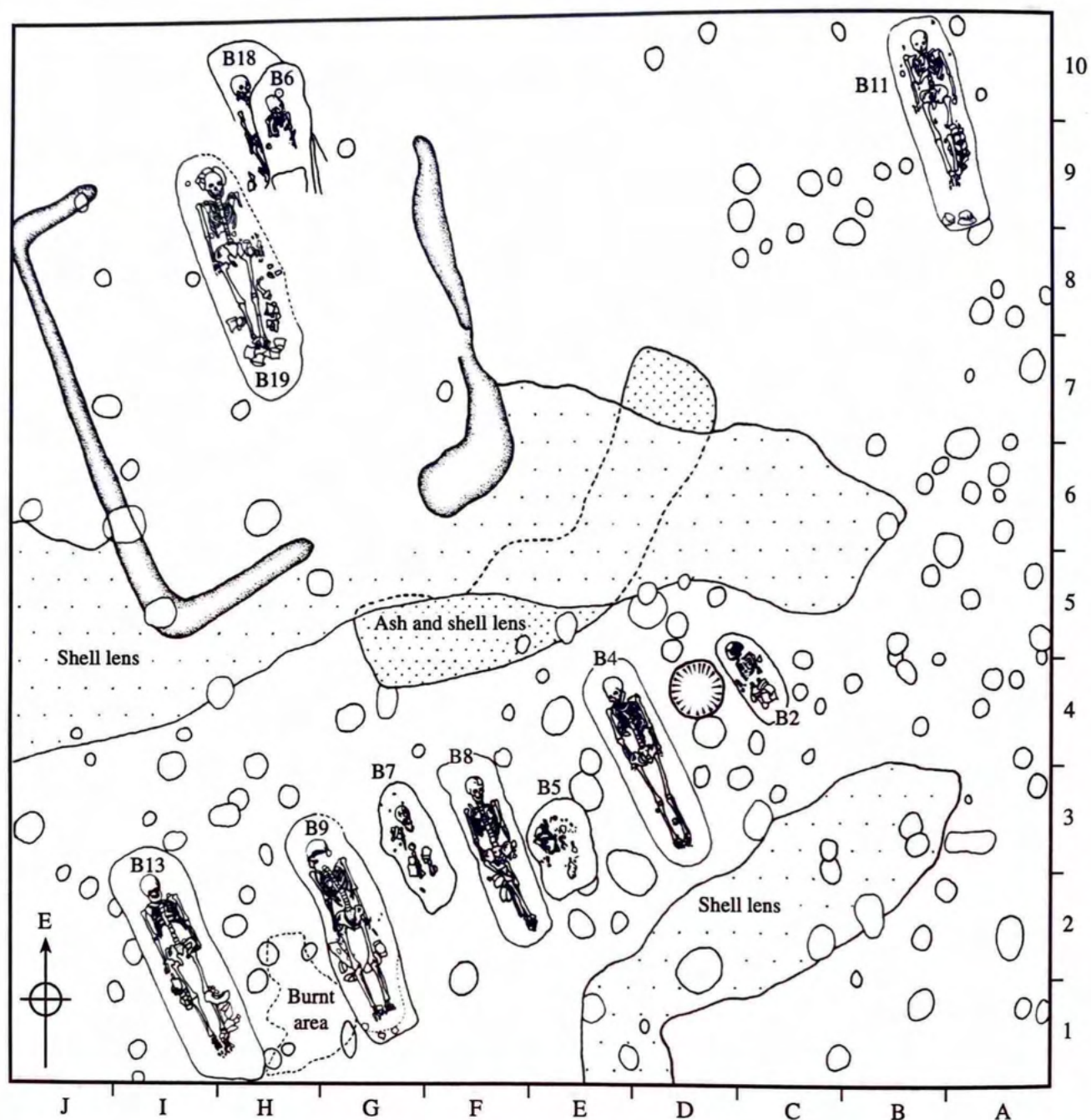


FIG. 8. The distribution of burials and features in MP6. The rectangular structure around B19 is the raised platform, and unfilled circles are postholes or areas of disturbance. Scale in metres. (Higham and Bannanurag 1990)

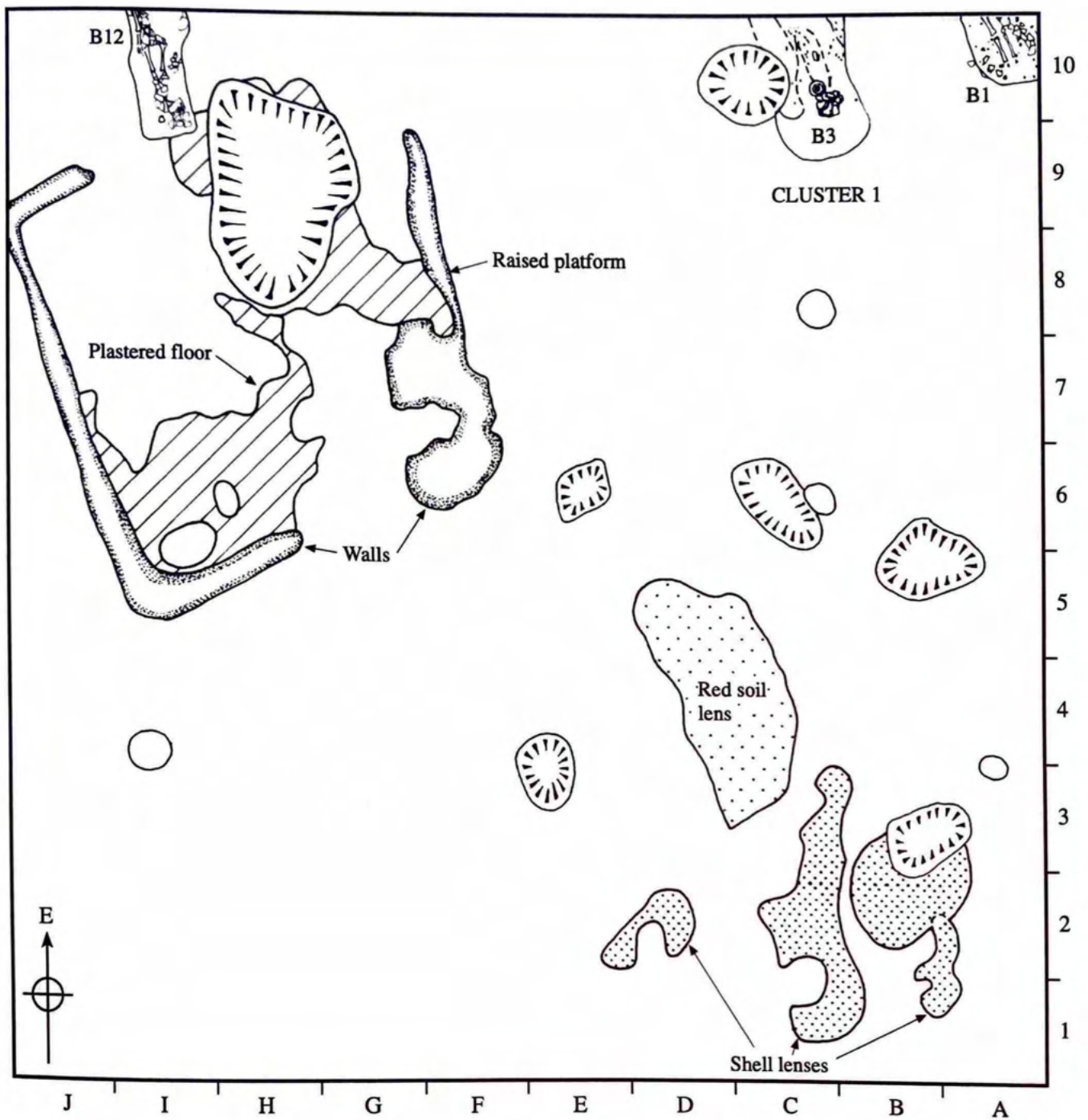


FIG. 9. The distribution of burials and features in MP7. Unfilled circles are postholes or areas of disturbance. Pits are hachured. Scale in metres. (Higham and Bannanurag 1990)

Use of the excavation area primarily as a cemetery continued during the final three mortuary phases, although during these phases the burials were arranged in rows or small groups. There was a total of 21 burials in 1.4 m of deposits during MP5-7. The surrounding features continued to include pits, postholes, midden and hearths, all of which may have reflected activities related to the mortuary ritual rather than occupation. These later phases were distinguished from the preceding period and from each other on the basis of cultural variation rather than significant stratigraphic breaks, although there were brief hiatuses represented by c. 10 cm of deposit between the phases. The lack of any evidence for a break in site use suggests occupation of the site by a sedentary group.

The six graves in MP1 were devoid of grave goods apart from a single individual with a few shell beads. In other respects, however, they established some of the mortuary practices which continued throughout the cemetery. These include the orientation of the graves to the east and covering of the body with ochre. The concentration of burials in clusters in MP2-4 suggests the deliberate interment of burials in discrete areas for the significant period represented by 3 m of deposits. Most of the burials were located within the clusters, with only a few infants found in isolated graves. Within the clusters some graves intercut others but there was no disturbance of the skeletons. This, together with the depth of the deposits in which the clustering continues, suggests that the concentration of burials was deliberate rather than the result of random distribution. In MP3 in particular, the distribution of a large shell midden with a depth of up to one metre suggests the presence of structures over the clusters (Fig. 5). Many of the graves in the clusters extend beyond the edges of the excavation square, however, and all clusters border on the baulk for at least part of the deposit. This does not negate the possibility that the clusters are indeed discrete and extend beyond the excavation square, but leaves open the questions of the actual extent of each cluster in time and space and the number of burials they contain.

Cluster C has the least contact with the edge of the square and, together with Cluster F, is represented almost continuously throughout MP2-4. Burials are present in all three phases in the area of Cluster A but in fewer numbers and with more breaks in the deposits than Clusters C and F. The areas in which the remaining clusters, B, D, E and G, are defined, contain fewer burials and the interments are sporadic or confined to some levels. A single individual in MP4 (B22) was in a grave isolated from the clusters but adjacent to the burials in MP5, described below.

The provision of grave goods becomes a feature of the mortuary ritual from MP2, although the amount and type of goods varies among individuals and over time. In her analysis of the mortuary remains, Bannanurag (1989) has compared the number and type of grave goods between clusters. The comparison was made by allocating arbitrary values to each type of grave good, with consideration of rarity, whether the material used was exotic, and the skill required in manufacture. The results of the multivariate analysis show that although there are differences between clusters at particular times, there is no consistently wealthy cluster. Rather, wealth as evidenced by grave goods appears to oscillate between clusters over time.

From MP5 there were significant differences from earlier interments. The burials were no longer superimposed in discrete clusters. Each phase was represented by shallower deposits with fewer burials than earlier phases, and several burials were elaborate and richly endowed with grave goods. The one adult amongst the three graves in MP5 (B15) was buried in an exceptionally large, deep, grave, with a wealth of grave goods, including pots and pottery manufacturing implements. The

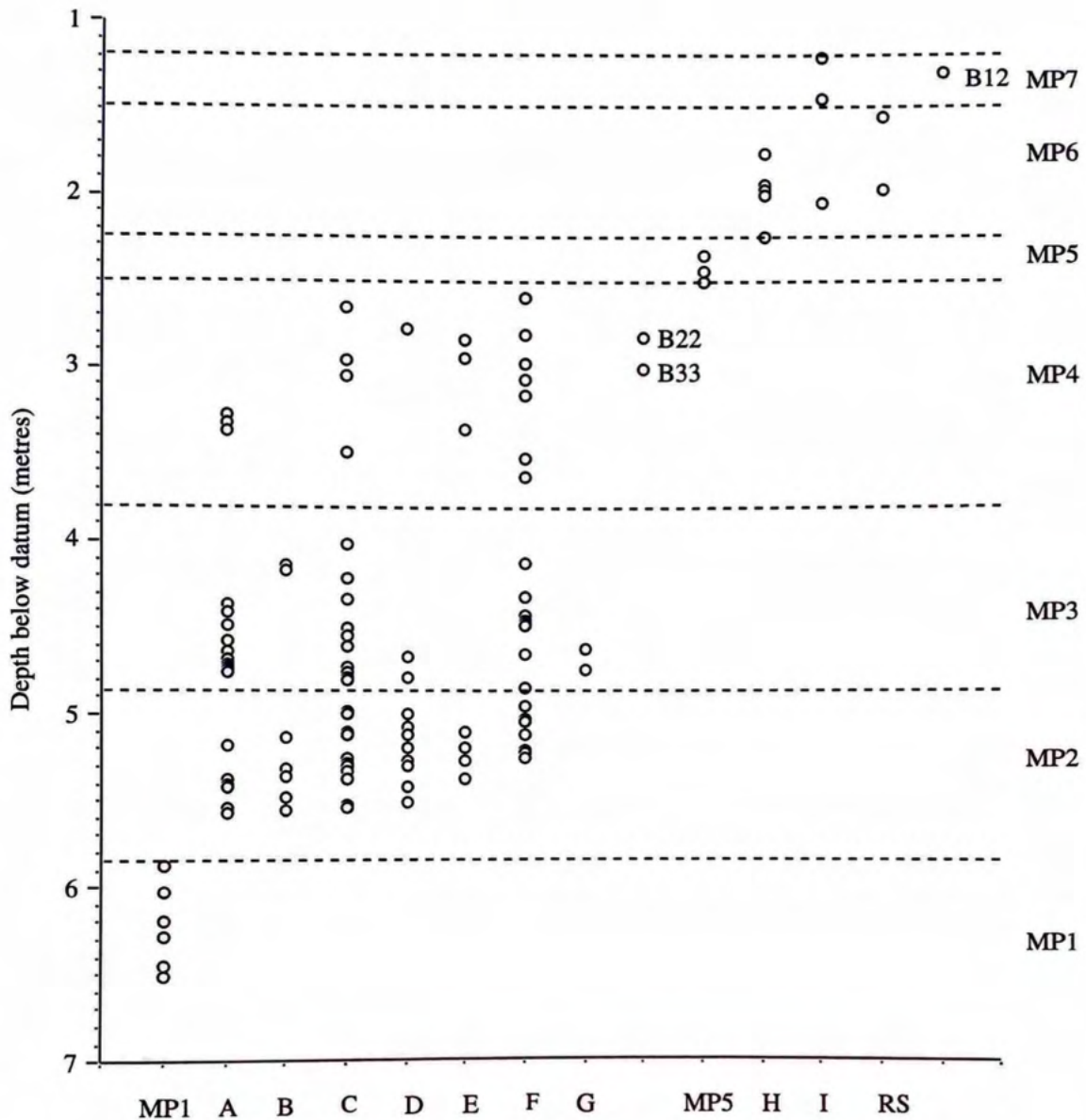


FIG. 10. The stratigraphic relationships of the burials, showing the distribution in mortuary phases (MP) and clusters (A-I). The individual burials (B) are those which were not allocated to clusters or belong to other groupings. RS denotes the burials beneath the raised structure

body was covered with a mound of clay cylinders (probably the raw material of pottery manufacture) and broken pots. The isolated burial in MP4 (B22) was adjacent and almost directly below B15. The three other burials in this phase were also very rich. An infant, B16, buried parallel and

adjacent to B15, was interred with the same ritual, including a miniature potter's anvil. Another infant (B14), also close to B15, was buried in two large, elaborate, pottery vessels. This was the only jar burial at the site. The grave of the fourth burial in the phase, a male (B43), was separate from the others and projected partly into the east baulk.

During MP6, the density of graves increased slightly. There was a row of graves in approximately the same area as the graves from MP5. These are designated Cluster H, although the characteristics of the earlier clusters (superposition and concentration of burials) were not present. There was also a group of three graves associated with an elaborate raised structure. The most recent of these, B6, disturbed an earlier burial and both were disturbed by a later pit.

The four MP7 burials are subadults. One grave with two individuals (B12.1, 12.2) was isolated above the MP6 mortuary structure and three were situated in the south east corner. The latter, together with one individual from MP6 (B11), were designated Cluster I. The graves contain a similar number and complement of grave goods as the earlier Cluster H.

Further analyses of grave goods in relation to various factors (Bannanurag 1989) have shown that there was no significant relationship between the amount and type of grave goods and age at death, either for adults or subadults, except in the burials of newborn infants, who were almost without exception buried with nothing, or with a covering of red ochre. Infants who survived birth by a few months were treated in the same manner as older individuals. In many cases, there is evidence that the wealth of subadult graves matches that of adjoining contemporary adults. Variation in wealth of grave goods on the basis of sex in adult graves was also found to be absent or insignificant in MP2-3. Some goods which appeared from MP3 were sex specific, with turtle carapaces and plastron objects being restricted to males, and pottery anvils to adult females and children. In MP5-6 most of the wealthiest graves were those of females.

THE SKELETONS OF THE PEOPLE OF KHOK PHANOM DI

The excavated area contained 154 skeletons. Of these, 153 were identified during the excavation as burials (numbered 1-154, with numbers 148 and 149 being seen only as ochre traces and infant bone fragments in section, and one burial containing two individuals). During laboratory analysis, a further burial was found to contain the remains of two individuals, both children. The skeletons in the double burials are identified by decimal suffixes to the original burial numbers.

Most of the skeletons are complete and the bone tissue is in very good condition. The considerable number of burials which partly extended into the baulks were removed at the end of the excavation. Some individuals, particularly during MP1 and MP6, were slightly disturbed by intercutting postholes and pits, but the proportion of children and adults with major bones completely or partly missing as a result of disturbance is less than 10 per cent, and there is almost no evidence for mixing of skeletons during prehistory. There is one adult individual (B22) without a skull. Although the reason for this cannot be unequivocally identified, the presence of all cervical vertebrae except the atlas suggests that it was removed sometime after burial, rather than decapitation being the cause of death. There was other minor disturbance of skeletons, particularly infants, during prehistory, possibly the result of natural phenomena such as movement of waterlogged soil during millennia

of successive rainy seasons. In the majority of cases the disturbed bones were identified when the skeletons were lifted. There are some bones, again particularly from infants but including some adult hand and foot bones, which were identified during the analysis of the faunal assemblage. A further minimum of six infant skeletons, mainly from the lower deposits of the site, have been excluded from the skeletal analysis because their provenance is uncertain. Burials 148-9 have also been excluded.

The preservation of the bone is particularly good, with only a few cases where the damage prevented reconstruction. Many of the skeletons, however, were heavily encrusted with a calcareous deposit which cemented the bones to each other and to the surrounding matrix. In the lower levels of the site, many of the bones were hardened to an extent that suggests that fossilization had commenced.

Cleaning, preparation, data recording and management

The bones were cleaned as much as possible using small implements such as scalpels, dental probes and brushes. The manual cleaning was supplemented where necessary to remove the concretions by soaking with a solution of 7 per cent acetic acid, 2 per cent calcium acetate for 10-15 minutes (Dennison pers. comm.). Small bones which could be immersed in the solution were soaked overnight. This softened the deposits which were then removed manually. The bone was then soaked in clean water for 20-30 minutes and dried. The method was not always successful, and the manual removal of the encrustation resulted in some damage to the subperiosteal surfaces. Where possible, broken bones were reconstructed using latex glue and masking tape. This work was carried out during 1986 at the University of Otago by workers employed under a New Zealand government-sponsored work scheme, by students employed on summer student job programmes, and by Thai students and their families.

All measurements for cranial and infracranial bones were made using standard osteometric techniques with sliding and spreading calipers, a goniometer and an osteometry board. All data have been recorded in a database using the 'Double Helix' application on a MacIntosh computer. This comprehensive database is held at the Department of Anatomy and Structural Biology, University of Otago Medical School.

PREVIOUS RESEARCH IN PREHISTORIC SOUTH EAST ASIAN HUMAN BIOLOGY

Existing reports of the skeletal remains of prehistoric South East Asians are not confined to those from Thailand, but include samples from other countries of mainland South East Asia. The area of relevance is defined primarily by the drainage systems of the Chao Phraya, Mekong and Red rivers, with an extension to the south on the Indo-Malaysian Peninsula. This geographical circumscription is a reflection not only of the spread of populations which may be considered to represent suitable comparative populations, but also of the availability and accessibility of skeletal studies. No reports have been located for the area to the west, within Myanmar, and recent reports from Vietnam and China have been published in local languages and are effectively inaccessible.

The availability of reports of any nature on prehistoric skeletal material from the core area of Thailand and the immediately adjacent countries is subject to a number of factors. These include the changes in approach to skeletal analysis since the beginning of the twentieth century when 'modern' archaeological research first started in South East Asia. For most of that time, the skeletal material recovered was almost universally viewed only as a means of establishing phylogenetic relationships from morphology. The emphasis was on complete skulls, suitable for measurement and classification, and consequently other material was only minimally reported and not well conserved. In addition there is the universal problem of postmortem damage or destruction of skeletons through either environmental conditions or human activities. This is particularly evident in some sites where acid soils have resulted in the complete destruction or such poor preservation of bone that burials have only been recognised by the presence of grave outlines or the disposition of grave goods, for example at Dong Son and Lang Ca, Vietnam (Higham 1989:194).

There is no consistent pattern of archaeological investigation, and consequently of skeletal material available for study, among the various modern states of South East Asia. The political history of each has had a significant effect on the local development of archaeology and, along with varying cultural ideas, continues to influence attitudes towards archaeology and expectations from research. The direction in which these indigenous attitudes and expectations have developed shows in the type of research being done and whether the international archaeological community is encouraged to contribute to and use the research. The latter may simply involve the publication of results in international journals, or allow active participation of nationals from other countries in excavations and/or analyses.

Bray and Glover (1987), have discussed the development of South East Asian archaeology. The concept of the study of prehistory as a source of information about humanity is a western one, and archaeology in South East Asia was initiated by westerners and carried out by them for most of its history. The earliest work, within the context of colonial society, was based on:

....the dominant mode of archaeological procedure of the early twentieth century: concentration on material form and typology for the recognition of culture groups and culture areas, and explanation of all change in the archaeological record as the result of diffusion of techniques or the migration of peoples from one culture to another. (Bray and Glover, 1987:111)

This philosophy was applied beyond the field of artefact typologies to skeletal analyses, resulting in the concentration on racial classification of skeletons as a means of identifying migration routes and population relationships. This was based primarily on cranial morphology. Crania showing features from one or more of the accepted categories were classified as 'mixed', based on a belief in the genetic control of morphology. Collections of skulls such as that from Lang-Cuom, North Vietnam, (Mansuy and Colani 1925) were variously classified and reclassified by Mansuy and Colani as Melanesian/Australoid/Indonesian, by Saurin (1939) as Melanesian/Negrito/Indonesian and by Coon (1962) as 'modified Australoid', 'more or less Mongoloid' or 'mixed Negrito'. Classifications such as these, and the interpretations of timing and routes of migrations and racial mixing based upon them, formed the basis of some theories of the origins of South East Asian peoples (e.g. Barth 1952, Coedès 1966, Coon 1966, Jacob 1967) which continue to influence thinking

until recently (Howells 1973, Bellwood 1978, 1985) despite the major changes in understanding resulting from the explosive growth of the discipline of molecular biology.

As Western attitudes towards archaeology changed, particularly with the impetus of the 'New Archeology' in the U.S.A. in the 1960s, attitudes towards skeletal material also changed and many studies widened to include skeletal biology. This included the study of pathology, demography, diet and evidence of physical activity. Western archaeologists see this as developing part of the wider picture of prehistoric humans, whereas indigenous archaeologists are more interested in chronicling the history of their individual countries as part of the growing national consciousness.

This brief overview of the history of archaeology in South East Asia, concentrating on those sites which have produced human skeletal material, illustrates the way in which history mirrors the political and social changes which have occurred in each of the countries, as well as showing the effect of changes in international research methods.

Vietnam, Laos and Cambodia

The earliest published work was initiated by the French in the area now covered by the modern states of Vietnam (formerly Tonkin, Annam and Cochin China), Laos and Cambodia. This area had been colonised in the latter half of the nineteenth century and was then known as French Indo-China. Most of the research was undertaken under the auspices of the *École Française d'Extrême Orient* and the *Service Géologique de l'Indochine*. These excavations, carried out from the beginning of the twentieth century until the withdrawal of the French in the 1950s, were directed by French archaeologists and geologists such as Mansuy, Colani, Patte and Fromaget, and were generally well reported for those times. Although some resulted in the recovery of skeletal material, this has been minimally reported and the way in which it has been discussed is largely limited to racial classification. Table 2 lists some of the material found, with brief details of the provenance, dating and publication.

Pietrusewsky (1974b) records that Huard and Saurin (1938) listed 205 individuals which comprised all known skeletal material recorded to that date, primarily in North Vietnam and Laos. After the end of the French colonisation the Vietnamese continued working in archaeology. Nguyen Duy (1967) has very briefly summarised the skulls found in Vietnam up until 1967, including some of those recovered after independence. Most of their skeletal reports have been published in Vietnamese (Pietrusewsky (1974b) lists some publications). A brief report on the two best preserved skulls amongst the 30 skeletons excavated from Quynh Van, Nghe An, North Vietnam, has been published in English by Nguyen Duy and Ng Quang Quyen (1966). This typically concentrates on the description of morphology and metrics with the aim of racial classification, but with brief mention of dental and skeletal pathologies. Other finds are noted by Ha Van Tan (1976, 1980) and Higham (1989) but no detailed information on the skeletal remains is accessible. Recent reports on skeletal material by western researchers is represented by a single report by Pietrusewsky (1988a), who visited Hanoi in 1984. He examined and recorded measurements and non-metric traits on eleven intact crania amongst the 104 individuals excavated from the Coastal 'Neolithic' site of Con Co Ngua, Thanh Hoa province of Vietnam, with the major objective of assessing the biological relationships of the new material from Vietnam with previously examined Neolithic and near contemporary crania from South East Asia and the Pacific through the application of multivariate statistics.

TABLE 2: *Excavations with prehistoric skeletal finds, Vietnam, Laos and Cambodia*

Year	Excavator	Site(s)	No. of individuals	Dating	Skeletal report
1906	Mansuy	Pho Binh Gia; Vietnam	3	Bacsonian	Verneau 1909
1924	Mansuy	Keophay , Khackiem, Hamrong, Donthuoc; Vietnam	4	Bacsonian/ Neolithic	Mansuy 1924*, 1925a, 1925b.
1923	Patte	Minh-Cam; Vietnam	1	Late Neolithic	Patte, 1925
1925	Mansuy and Colani	Lang-Cuom; Vietnam	18?	Early Neolithic	Mansuy and Colani 1925, Saurin 1939*
1927	Colani	Lang Cao; Vietnam	200 skulls	Hoabinhian	unpublished? (Colani 1927)
1932	Patte	Da But; Vietnam	12	Neolithic	Patte 1932, 1965
1938	Fromaget	Tam Pong, Tam Hang; Cambodia	1 12	Mesolithic Palaeolithic- Neolithic	Fromaget and Saurin 1936*

*Complete skulls studied by Pietrusewsky and included by him in analyses of biological relationships using multivariate statistical methods (e.g. 1974b, 1981, 1988a)

This work represents the modern method of searching for the origins and biological relationships of populations. He also examined 'several other prehistoric specimens, including pre-Hoabinhian crania' (1987:2) although these are not included in the reports.

Malaysia

Archaeology was also first initiated in Malaysia by Europeans, with excavations which recovered skeletons conducted by Sieveking in 1954 at Gua Cha (Sieveking 1954) and van Stein Callenfels at Gua Kepah in 1936 (van Stein Callenfels 1936). Gua Cha was dated to c. 8000 BC–AD 1 and the skeletal report was by Trevor and Brothwell (1962). Skeletal reports for Gua Kepah were by Mijsberg (1940) and Jacob (1967). Both these sites are on the Malaysian Peninsula. Gua Cha was recently re-excavated by Bellwood and Adi bin Haja Taha (1981 - skeletal report by Bulbeck 1981, 1982).

Thailand

The development of prehistoric archaeology in Thailand contrasts with that in other South East Asian states because the country was never colonised. The European influence evident in the neigh-

bouring countries from the early twentieth century was not significant in Thailand until the first of a series of joint Thai/European projects in the early 1960s, Danish inspired excavations at Ban Kao and Sai Yok, Central Thailand. Prior to that, in the 1930s, an excavation by Quaritch-Wales of an historical site at P'ong Tük, near Ban Kao, had exposed eleven individuals who were buried with copper and iron grave goods 1.5 feet below structures dating from the sixth century AD. These are reported briefly by Quaritch-Wales (1937, 1964), but without any skeletal analysis. The excavations at Ban Kao and Sai Yok were led by van Heekeren and Sørensen in 1960-62 (van Heekeren and Knuth 1967, Sørensen and Hatting 1967). The skeletal material from the site was studied and reported by Sangvichien *et al.* (1969), and was the first to be published in detail from Thailand.

Other Thai sites from which skeletal material has been recovered and reported in detail in English are all from the Khorat Plateau. The first was Non Nok Tha, excavated in 1966 and 1968 (Solheim *et al.* 1966, Bayard 1971). This was followed by the excavation of Ban Chiang in 1974 and 1975 (Gorman and Charoenwongsa 1976, White 1982), Ban Na Di in 1980 (Higham and Kijngam 1984) and Non Pa Kluay in 1984-85 (Wilen 1989). The dating of these sites has been the subject of disagreement. Figure 11 illustrates the most recent interpretations (Higham 1996). At Ban Kao, all but two were from the 'Neolithic'; at Non Nok Tha, 13 were from the early Phu Wiang Phase and the majority from the later Non Nok Tha phase; at Ban Na Di, all but eight were from MP1. However, at Ban Chiang, the burials are less easy to ascribe to a particular period. A total of 69 burials were recovered in the Early Period (EP) I-V. Those from EPI-II are probably Neolithic. The Bronze Age covers EPIII-V. Iron enters the sequence during the Middle Period (30 graves) and continued with the Late Period (16 graves).

The skeletal reports are summarised in table 3. It should be noted that this list is restricted to those published in English prior to 1992, excluding the numerous reports which have been published in Thai, by staff of the Fine Arts Department. These analyses suggest that the lifespan of prehistoric South East Asians was as long as that of prehistoric peoples generally. Few of them appear to have lived beyond 40-50 years, but the mean life expectancy at age 15-17 years ranges from 16 to 19 years, exactly the range of mean estimates calculated from a large, international, sample of prehistoric hunting and gathering and protoagricultural populations (Weiss 1973a:49). Infant and child mortality ranges from 15 per cent at Ban Kao (Sangvichien *et al.* 1969) to 42 per cent at Ban Na Di (Houghton and Wiriyaromp (1984) with most within the range of 30-50 per cent to be expected in such populations (Weiss 1973a).

The mean stature of males ranges from 166.0 cm to 174.0 cm and of females from 156.0 cm to 159.0 cm. This is bordering on the level described as 'tall' on an international scale (Wells 1970), although the calculations have often been based on formulae derived from populations which are temporally, geographically and genetically distinct from prehistoric South East Asians. The reports show that the prehistoric South East Asians suffered from dental problems involving both teeth and periodontal tissues. Joint degeneration and fractures are no more prevalent than could be expected in people living an active life and dying at a relatively early age. Otherwise, there is no skeletal evidence of any major pathology apart from the suggestion of widespread anaemia diagnosed in some groups on the basis of thickened cranial vaults. If the diagnosis of anaemia is correct, the aetiology of the condition is of particular interest in describing and explaining the health of the people.

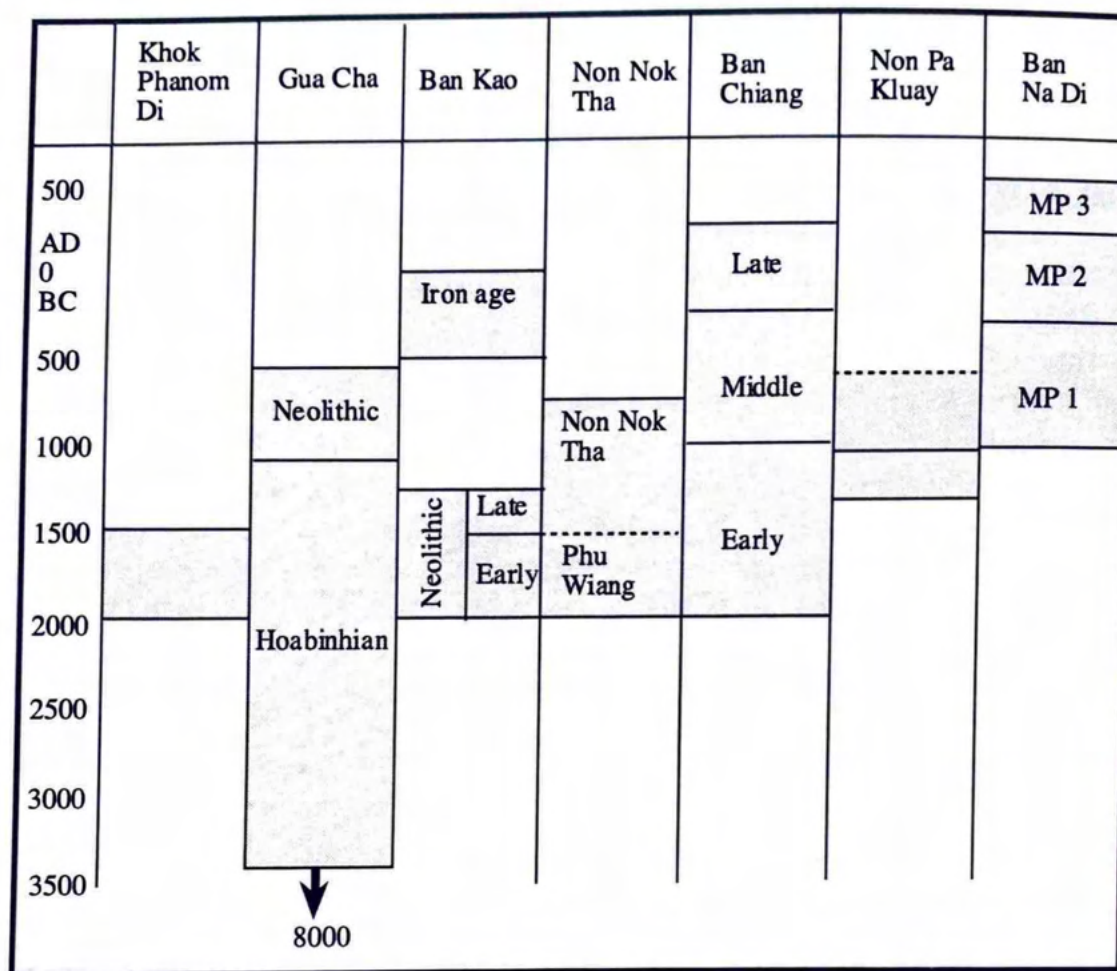


FIG. 11. The chronological relationships of the prehistoric South East Asian sites discussed in this chapter. Sources of these data are: Khok Phanom Di - Higham and Bannanurag 1990, Non Nok Tha - Ban Kao - Higham (1989:159), Non Pa Kluay - Wilen (1989), Ban Na Di - (Higham and Kijngam 1984). The chronology is controversial and the illustration is based on a recent interpretation by Higham (1996)

Four of the six populations are from the Khorat Plateau in North East Thailand, and the evidence from these is indicative of the skeletal variation which appears to occur even over a relatively restricted area. The people of Ban Na Di were tall and robust, and the people of nearby Ban Chiang relatively short. The adult crania at Non Pa Kluay have a high prevalence of porotic hyperostosis (Pietrusewsky 1988b). None of the individuals from Ban Na Di had cranial vault thicknesses beyond the normal range (Houghton and Wiriyaromp 1984). Interobserver variation in the analysis and interpretation is possible in producing these differences.

This rather bland picture of the health of these people is admittedly committing the sin of amalgamating data from populations from different environments who lived over a period of millennia, but it illustrates the situation which prompted Pisit's comment at the beginning of this chapter.

TABLE 3: *Excavations with prehistoric skeletal finds, Thailand*

Year	Excavated by	Site	No. of individuals	Skeletal report
1960-62	Sørensen <i>et al.</i>	Ban Kao Kanchanaburi	38	Sangvichien <i>et al.</i> 1969
1960-62	van Heekeren and Knuth	Sai Yok Kanchanaburi	1	Jakob 1969
1966	Solheim/Bayard	Non Nok Tha Khon Kaen	81-83	Pietrusewsky 1974a, 1974c
1968	Bayard	Non Nok Tha Khon Kaen	107-119	Unpublished
1974-75	Gorman/Charoenwongsa	Ban Chiang Udon Thani	111	Pietrusewsky 1982, 1984, 1997
1980	Higham/Kijngam	Ban Na Di Udon Thani	73	Houghton and Wiriyaromp 1984 Wiriyaromp 1984a, 1984b
1984	Wilen	Non Pa Kluay Khon Kaen	19	Pietrusewsky 1988b

In this volume, the census and demography of the sample from Khok Phanom Di, including the methods used for age and sex estimates, are discussed in chapter II. The physical appearance of the people is described in chapter III. Chapters IV–VI describe the evidence for health and disease and the significance of the findings is discussed in chapter VII. Chapter VIII discusses chronological changes within the site. Summary statistics are listed in tables within the text; individual data tables are provided at the end of each chapter.

II. CENSUS AND DEMOGRAPHY

CENSUS - AGE AND SEX

THE most basic and perhaps the most important information to be obtained from the skeletons is the census data, the age at death and sex of the individuals. These data are important not only in establishing the composition of the sample but as basic parameters for the analysis of many other aspects of human biology and for the interpretation of social or cultural characteristics of the population from mortuary practices.

The estimation of age and sex often appears to be approached as an exercise in the application of sets of standards, with the subjection of the results to intensive statistical analyses giving a spurious sense of accuracy. The estimates are subject to many potential errors and biases, partly because of the subjective nature of many methods, and partly because of the potential for significant variation of the population from available standards. Although the process of aging and the morphological evidence of sex are universal, they are highly variable in rate and expression as a consequence of both genetic and environmental factors, although the relative uniformity of environmental factors affecting small, homogeneous, prehistoric groups suggests that intrapopulation variation may be lower than in modern heterogeneous populations. The problem of the applicability of age and sex standards can only be minimised by using the indicators least variable among populations, although in many cases the condition of the skeletal material dictates the methods or indicators which can be used, rather than any consideration of suitability or accuracy. The interrelationship among the parameters also complicates the procedure, with sex influencing the aging process and age affecting the degree of expression of sex characters. As the latter is the least marked, the estimation of sex is usually undertaken first.

ESTIMATION OF SEX

As the indicators used in the estimation of sex are mediated by both genetic and environmental factors, the degree of sexual dimorphism is population specific and sex estimation ought to be based on population specific parameters. In the absence of suitable standards for modern South East Asians, the most appropriate available standards are used.

For adults and older adolescents, the principal indicators used are on the pelvis, as this reflects sex differences in function. Sexually dimorphic features principally relate to the size of the pelvic cavity, as the wider female inlet is reflected in a longer pubic bone and the wider outlet in a more open angle of the sciatic notch and sub pubic arch. Both metric and subjective methods are available for assessing these characteristics. Pitting adjacent to the joints of the pelvis in response to pregnancy and parturition (Houghton 1974, Ullrich 1975) is also clearly sex-specific and therefore a useful indicator.

The characteristics of the cranium, mandible, and remainder of the post-cranial skeleton reflect dimorphism in body size in the form of relative bone sizes, rugosity of muscle attachments and general robusticity. This dimorphism is partly the result of hormonal stimulation, as in the relatively greater width of the shoulders and greater muscle mass in males, but the male also has a longer pre-pubertal growth period and this results in a generally greater body size and differences in body proportions (Tanner 1962, Harrison *et al.* 1988). The use of these size/robusticity dependent characteristics in estimating sex relies on the assumption that dimorphism in a population is sufficient to differentiate between the sexes.

The cranium and mandible are the most useful indicators of size and robusticity. The use of multiple factors where possible reduces the likelihood of misclassifying individuals where sexual dimorphism in a population is not strong. As there is a well-documented strong positive relationship between joint dimensions and body size (Frisancho and Flegel 1983, Ruff 1988), the diameters of the major joints, particularly of the weight-bearing bones, are also useful. Tooth size has also been shown to be sexually dimorphic (Ditch and Rose 1972, Rösing 1983).

Metric methods of discrimination are available for all indicators, and morphological observations for the pelvis and skull. Opinion is divided as to the value of each, although Meindl *et al.* (1985a) found that metric methods were no more successful in resolving problem cases than observation in the pelvis, and were less successful in the skull when the mandible is included. The characteristics used for the pelvis were:

1. Subjective assessment of pubic arch angle.
2. Presence of pitting on dorsum of pubis.
3. Pubic indicators identified by Phenice (1969).
 - (a) form of ventral arc.
 - (b) form of ischio-pubic ramus.
 - (c) shape of sub-pubic cavity.
4. Subjective assessment of sciatic notch shape and angle.
5. Morphology of preauricular groove (after Houghton 1974).

The characteristics of the cranium and mandible were selected from those listed by Acsádi and Nemeskéri (1970), as follows:

1. Shape of anterior mandible.
2. Size of supra-orbital ridge and glabella.
3. Size of mastoid process.
4. Rugosity of muscle attachments on the occiput.
5. Shape of orbits.

Sex was first estimated independently from the pelvis and the cranium. Measurements of femoral head diameter (medio-lateral) were taken, and for those individuals where the pelvic and cranial indicators were equivocal or at variance, the femoral head measurement was compared with that of individuals where sex was unequivocal. All adult skeletons from Khok Phanom Di have pelves, with only seven having missing or badly preserved pubic bones and three with other significant damage to the bones. All except two have crania, including mandibles. Femoral head diameters could be measured on at least one femur in all except six individuals.

The estimated sex and indicators used for all adults are listed in table 13. Of the 68 individuals aged 15 years and over, 36 have been sexed as female, 30 as male and two provisionally as male. All were sexed primarily on the basis of the pelvis, with the estimation confirmed by cranial (including mandibular) morphology and/or femoral head diameter in 57 cases. The measurements of femoral head diameters are listed in table 14 and summarised in table 4.

TABLE 4: *Summary of femoral head diameters for both sexes (medio-lateral, mm - Martin and Saller (1957) measurement 19)*

	n	Mean diameter	s.d.	Range
Females n=29				
Right	25	40.9	2.3	36.5–44.7
Left	22	41.1	2.0	37.9–44.6
Males n=29				
Right	22	45.8	2.3	39.9–49.9
Left	27	45.5	2.4	40.6–49.5

Figure 12 illustrates the ranges and sexual dimorphism of femoral head diameters. With three exceptions, the femoral heads under 43 mm in diameter belong to females and those over 45 mm to males. The males with small femoral heads are burials 92 and 117, both young males with recently fused epiphyses, and B24, the smallest adult male in the group. Twenty-two of the females (76 per cent) have femoral head diameters of less than 43 mm and 17 of the males (59 per cent) have diameters of more than 45 mm.

There was some disparity among indicators in eleven individuals (Table 5). In most cases the individuals were either young males (aged 15–19 years) with smaller, less robust characteristics which probably reflect incomplete growth. Others include the shortest male and some of the taller females. The oldest person in the group, B122, is a female with a more robust cranium which is in accord with a process noted by Meindl *et al.* (1985b:81) whereby sex dependent morphology of the skull is significantly affected by age, with greater age producing an increasingly masculine morphology. Two individuals where sex is estimated provisionally as male are B8, the youngest 'adult' at 15 years whose pelvis was not fused, and B28, an individual showing signs of suffering

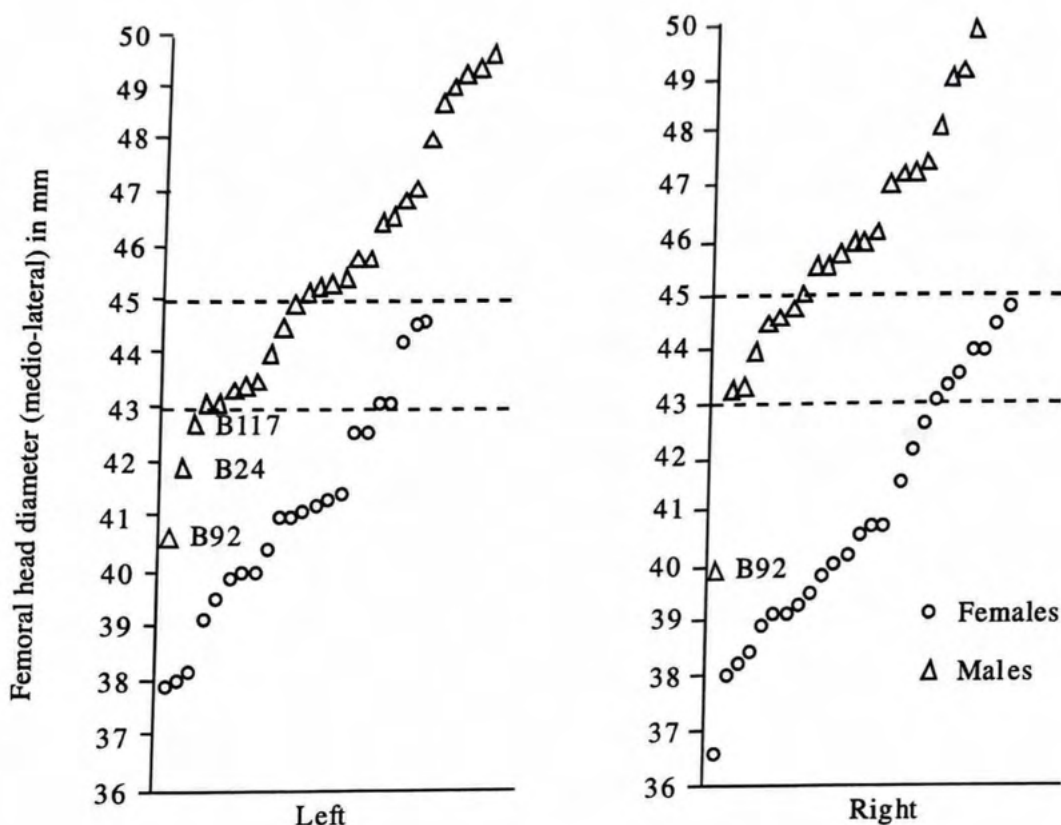


FIG. 12. Femoral head diameters, illustrating the degree of sexual dimorphism

from a severe, chronic disease which had caused osteoporosis of the entire skeleton and could have had an effect on the pelvic morphology in particular.

As most secondary sex characteristics in the skeleton do not develop until puberty, the estimation of sex in children is generally not possible. An exception to this is tooth size, which Garn *et al.* (1967) have shown to have a genetic basis. The formation of the permanent dentition during childhood provides a sexually dimorphic characteristic at an early age. This is a valid indicator where there are a number of adults sexed on the basis of other characters, and the degree of dimorphism is sufficient to allow the calculation of discriminant functions which can then be applied to the dentition of children. Tests have shown up to 97 per cent accuracy using this technique on adults (Ditch and Rose 1972, Rösing 1983). The canine teeth, in particular, show dimorphism as they have a relatively longer period of development in males but incisors, premolars and first and second molars can also be used with a high degree of accuracy (Garn *et al.* 1967, Moss and Moss-Salentijn 1977).

Accordingly, dental measurements for all teeth, both bucco-lingual and mesio-distal, were compared for adult males and females in an effort to establish whether the extent of dimorphism was sufficient to provide a means of establishing the sex of children and young adolescents with permanent teeth ($n=12$). The sample sizes for many measurements were too small to indicate whether or

TABLE 5: *Individuals with disparity among indicators of sex*

Burial	Age	Pelvis	Cranium	Femur head*	Comment
Females					
26	35	F	M	-	Tall
47	26	F	M	43.0	Tall
122	52	F	M	-	Oldest individual
142	31	F	M	-	-
Males					
8	15	M	F	-	Young
24	25	M	F	41.9	Short
28	21	M**	F	43.1	Pathological
44	18	M	F	43.1	Young
92	18	M	M	39.9	Young
117	16	M	F	42.7	Young
147	19	M	F	44.6	Young, tall

** probably male; F = female, M = male; * diameter in mm

not sexual dimorphism was present. For those measurements with larger sample sizes sexual dimorphism was not statistically significant, and accordingly no estimates of sex were made for children. Tooth size was also not a useful indicator of sex in adults.

ESTIMATES OF AGE

Individual variability in the aging process means that any estimation of age will represent biological, not chronological, age, as there is no 'normal' state for a given chronological age. The evidence used in estimation of age in subadults and early adulthood is developmental, reflecting the skeletal and dental maturity of the individual. Despite some intra- and interpopulation variation, estimation of biological age from younger skeletons is more likely to approximate chronological age than in older skeletons. In middle and late adulthood the evidence is primarily from degenerative changes, and the potential for variation in the rate and extent of changes is very much higher, both because of the greater effect of environmental factors on these indicators and the longer period of influence.

Methods of age estimation available include those using gross morphological observation, such as dental eruption, epiphyseal fusion, pubic symphyseal changes, dental wear and skeletal degeneration, radiography (dental calcification, trabecular involution and cortical thinning) and histology (osteonal counts). Lovejoy *et al.* (1985a), advocate seriation to reduce inter-observer error in within-group estimations. As with the estimation of sex, the use of multiple factors is best to minimise the effects of any aberrant indicators. Acsádi and Nemeskéri favoured this approach in 1970, and it has now been widely approved, (Buikstra and Konigsberg 1985, Lovejoy *et al.* 1985a, Angel

et al. 1986, Greene *et al.* 1986, Bedford *et al.* 1993) although different methods and indicators from those suggested by Acsádi and Nemeskéri are now used.

Infants and children

Eveleth and Tanner (1976:198) identify three indices of physical maturation: dental, skeletal and sexual. They note that these indices measure aspects of development which are universal in that they all ultimately reach the same end-point in all normal individuals. In the case of stature, however, where the final point is highly variable, the proportion of development represented at any given time is unknown until development is completed. Therefore the most suitable indicators of subadult age are those which measure maturation rather than size.

Dental maturity is the most suitable as it is least affected by environmental factors, with up to 90 per cent genetic control (Garn *et al.* 1965). The formation of the dentition takes place early in life, and unlike bone, does not thereafter alter its form, other than through deterioration. Calcification is more reliable than eruption as it is less subject to environmental influences such as delayed or premature loss of deciduous teeth, crowding, or oral pathology, but it does require radiographs for recording. This indicator can be used from birth until adolescence, when the apex of the roots of the second molars close. Skeletal maturity, ossification and fusion of epiphyses, can also be used. However, the recovery of small epiphyses or bones such as the carpals, used in methods for aging the living, is unlikely in archaeological sites as the small bones easily disintegrate or are overlooked. They are also difficult to identify out of context if they are recovered. Epiphyseal fusion is widely used in older children, although the standards available are based on populations limited in ethnic origin, age or sex. In the absence of dentition in small children, a more practical indicator of age is body size as evidenced by diaphyseal length. Most standards available are based on modern x-ray data (Maresh 1955, Gindhart 1973, Hoffman 1979, Stewart 1979, Angel *et al.* 1986) and appear to reflect the secular trend to earlier maturity when compared with standards based on prehistoric skeletal groups. Those available for prehistoric groups, with age estimation based on dental development, are for Amerindian and Eskimo (Johnston 1962, Merchant and Ubelaker 1977, Sundick 1978) and Slavs (Stloukal and Hanáková 1978, cited in Ferembach *et al.* 1980). Until about three years of age the rate of modern and prehistoric growth is relatively close, but at ages beyond this there is considerable variance among the diaphyseal lengths published, with evidence of accelerated growth in the modern standards. However, growth can still be used for older children where there is a sufficient number aged from the dentition to form a population-specific base sample of diaphyseal lengths.

For infants, dental calcification and eruption, skeletal maturity (fusion of fontanelles, mandible and vertebral arches) and diaphyseal length were recorded. Ages were first estimated for individuals with teeth present, based on radiographic atlases (Symington and Rankin 1908, van der Linden and Duterloo 1976). Those with no teeth were aged using standards for diaphyseal lengths based on the remaining individuals. Five infants with minimal material were aged by direct comparison of sizes of bones such as vertebrae with those of other individuals already aged on other criteria. Reference was made to standards for diaphyseal length in prehistoric American Indians (Johnston 1962, Merchant and Ubelaker 1977, Sundick 1978) where there was no comparable individual. Eleven of the twelve children aged 5 to 14 years were aged using dental calcification based on the radiographic

atlases supplemented by reference to the calcification charts of Moorrees *et al.* (1963). The remaining individual was aged using comparative diaphyseal length. Information on skeletal maturation was used only to confirm ages based on other indicators, as the majority of individuals were sufficiently complete to have more specific data available. Diaphyseal lengths are listed in table 15.

Adolescents and adults

Ages for the group from c. 15 years until mid-late twenties can be estimated using developmental factors, and these individuals then form the basis for extrapolation of degenerative indicators into older age groups. By the age of 15 years, dental calcification is virtually complete, apart from the third molar which is highly variable. However, the final stage of skeletal maturation, the fusion of almost all epiphyses, occurs during this time of life. The epiphyses subject to the most physical stress fuse first and by the early twenties all limb epiphyses are united. There are late fusing epiphyses such as the medial clavicle and the iliac crest which are useful for older individuals up to the late twenties.

Many of the methods advocated for aging mature adults are population specific and relative rather than absolute. The age estimation of prehistoric groups requires the establishment of standards which should ideally be based on younger individuals within the same group who have been aged using developmental indicators, such as dental calcification and epiphyseal fusion. The recognition of possible ranges of variation in maturational stages is important in applying standards, as is the appreciation of the compounding error which can develop when using these population specific relative methods based on an 'absolute' aged sample. This potential compounding error can increase with distance of the extrapolation from the base sample, as the distance between biological and chronological age widens with increasing influence of environmental factors. Weiss (1973b) advocated overcoming the problem by establishing a maximum 'expected' age, assigning this to the oldest individual and aging the rest relative to that age. The question remains, however, as to a reasonable upper age limit. Weiss advocates 75–80 years but, as he also notes, very few skeletal studies include individuals with age estimates over 45–50 years. Both Weiss, and Howell (1982), question the ability to survive of groups in which all individuals died at an early age, but a population with individuals assessed as surviving 30 years beyond that reported for other prehistoric groups would imply an extraordinary good level of health.

Beyond the ages covered by developmental features, the most widely used indicator involves changes in the pubic symphysis. Revisions of the method developed by Todd (1921), Brooks (1955), McKern and Stewart (1957) and Gilbert and McKern (1973) favour the Todd/Brooks scheme, with further modifications (Meindl *et al.* 1985b, Katz and Suchey 1986, Suchey *et al.* 1986, cited in Angel *et al.* 1986). The Meindl *et al.* scheme is for both sexes, but Katz and Suchey and Suchey *et al.* suggest that the indicator is valid only for males in the age range 17–30. Symphysis changes in males beyond this age are too variable to be useful and in females, hormonal effects, particularly in pregnancy, can alter the symphysis to such an extent as to produce variations which do not follow anything other than a very general pattern.

The alternative population-specific relative aging methods for older individuals based on evidence from younger individuals in the same group rely on the progression of degenerative changes

in the dentition and bones. A method using the attrition of dentition was first systematically devised by Miles (1963a, 1963b, 1978) and has been successfully tested on living populations by Kieser *et al.* (1983). Lovejoy *et al.* (1985a) used a modified version and judged the method as being the most reliable of those available. They advocate using it within multifactorial aging systems. The method involves extrapolating from the differences which appear in wear in the molars of young adults as a consequence of timing of eruption and length of wear. This requires a sufficiently large group on which to base the wear rate and the assumptions that the base line group is a representative sample of the population and that all members of the group have a similar diet (Nowell 1978). Other assumptions relate to relative wear rates of different teeth, and the eruption times of molars and the intervals between them.

Degeneration of bone with increasing age takes place in two ways, through atrophy and hypertrophy (Angel *et al.* 1986). Atrophy is evident in the reduction of trabecular bone and thinning of the cortex through endosteal resorption. These can be assessed from radiographs, with care taken that postmortem damage is identified and excluded. The bones most frequently used are the proximal femur and proximal humerus (Acsádi and Nemeskéri 1970), although Walker and Lovejoy (1985) found clavicular changes to have a higher correlation with age. Changes in cortical bone structure can also be measured histologically using the method originally devised by Kerley (1965) and modified by Ahlquist and Damsten (1969), Singh and Gundberg (1970), Kerley and Ubelaker (1978), and Thompson (1979). This involves counting osteons and circumferential lamellar bone on thin sections and assessing age using regression analysis. Stout (1988) notes various physiological factors which influence the rate of remodelling such as levels of physical activity, metabolic disturbances and hormones.

Hypertrophy is evident in the lipping of joint margins and is most common on the margins of vertebral bodies. Both forms of degeneration can occur at any age as a result of pathology, but in older individuals, progresses with general physical aging. The rate of degeneration is population specific, as genetics, nutrition, and patterns of physical activity all influence the progression of the bony remodelling. The homogeneity of prehistoric groups again improves the within-group comparability, although its use as a primary aging indicator will reduce its usefulness as an indicator of sex or status differences in physical activity patterns.

For all individuals aged 15 and over (n=68) the following were recorded:

1. Stage of fusion of the epiphyses, initially for all epiphyses using both the McKern and Stewart (1957) and Webb and Suchey (1985) methods. For final age estimation, the scores of the nine key epiphyses identified by McKern (1970) and the iliac crest and sternal clavicle were used.
2. Calcification of third molars, using the data from Moorrees *et al.* (1963).
3. Dental wear, using the eight point Molnar (1971) scale.
4. Stage of metamorphosis of the pubic symphysis, using both the Todd (1921) scheme with modifications by Brooks (1955) and the McKern and Stewart (1957) and Gilbert and McKern (1973) schemes.
5. Dental pathology. Details of the scoring methods are given in chapter VI.

6. Joint degeneration. Details of the scoring methods are given in chapter V.

The grades on the Molnar scale were found to be too wide for the initial stages of dental wear in younger individuals, and were modified by adding intermediate grades. The dental wear score on these individuals was then compared with that of older individuals and an attempt made to seriate the entire group on the basis of dental wear. At this stage the pubic symphysis scores were also compared. All factors taken into consideration were weighted in the age estimation in the following order:

1. Individuals aged 15–c. 28 years:

- (a) Epiphyseal fusion.
- (b) Pubic symphysis score.
- (c) Dental wear.

2. Individuals aged over c. 28 years:

- (a) Dental wear.
- (b) Pubic symphysis score (males only).
- (c) Joint degeneration.
- (d) Dental pathology.

3. For the older individuals, a maximum age of 50 years was assumed.

An attempt was made to make thin sections from tibial cores as suggested by Thompson (1979) but in many cases the bone was too hard for the cores to be drilled, or the periosteal bone split off the rest of the cortex during drilling. Of those cores which were successfully drilled, several were ground using Frost's method (Frost 1958) but this was also unsuccessful as the bone was either too brittle and disintegrated before it became thin enough or the subperiosteal bone broke off during grinding.

During recording of the pubic symphysis scores, the auricular surface of the ilium was examined following the method of Lovejoy *et al.* (1985b) but the age-related changes which they identified were not found. Consideration was also given to the changes of the sternal end of the 4th rib identified by İşcan *et al.* (1984a, 1984b, 1985, 1989) but the problem of identifying the correct rib became immediately apparent and this method was abandoned.

The number of adolescents and young adults forming the base group aged on developmental indicators totalled 23 (13 females and 10 males). These individuals were aged 15–26 years. They were aged on skeletal maturity and dental calcification in both sexes, and the pubic symphysis in the males. The individuals aged 15–19 comprised three females and five males. The McKern and Stewart (1957) data on epiphyseal fusion in males was adjusted downwards by approximately two years to reflect the earlier maturation in females. All long bone epiphyses were fused in females by the age of 18 years and in males by the age of 20 years. Beyond this age, the iliac crest and

clavicular epiphyses were fused by the mid twenties in both sexes. The use of modern standards for fusion sequence and rate precludes any detailed comment on the comparative sequence and rate in this population.

With the older adults, it became clear that the ages suggested by dental wear did not necessarily compare with that of other indicators, with some extreme differences. Visual seriation of all mandibular dentitions highlighted the extent to which dental pathology influenced wear patterns, particularly in older females. In those individuals where the dental pathology was widespread and no indicators of age other than dental wear were available, dental pathology was taken into account. Because of the variation in distribution and severity of pathology, this was necessarily a subjective exercise. Joint degeneration was then compared, and was found to progress with the increase in ages in almost all cases. This was taken into consideration only in those older individuals where the dentition was too damaged to provide a score for wear. Clavicle and femur radiograph samples were seriated, independently of each other and of the ages estimated on other parameters. However, a comparison of the ranking on the basis of both series of radiographs showed very little correlation with estimated age.

Table 6 lists the ranges of mandibular molar tooth wear for both sexes in the base group and table 7 lists the mean adult wear grades for molars in both sexes in five year age groups. Table 8 lists the ranges of pubic symphysis scores for the males in the base group. Joint degeneration in the base group was absent or minimal apart from individuals with specific pathology.

TABLE 6: *Mandibular molar wear (Molnar 1971 scale) in the base group of young adults independently aged using developmental criteria*

	Age group	n individuals	1st molar	2nd molar	3rd molar
Females					
	15-19	3	2-3	1-2	0-1
	20-24	5	2-3	1-3	0-2
	25-29	5	3-4	2-4	0-2
Males					
	15-19	5	2-3	1-2	0-1
	20-24	2	2-3	2	0-1
	25-29	2	3	3	2

The estimates of age and the indicators used are listed in table 13. There are 74 infants (aged 0-4 years), 12 children (aged 5-14 years) and 68 adults (aged 15 years and older). Ages were recorded in single years as part of the seriation process. This has the benefit of removing a tendency to group individuals at the beginning of each age group, which can happen if wider intervals are used (Lovejoy *et al.* 1985a). This age therefore represents age relative to others in the group but does not imply a level of accuracy to single years, except in infants. In older individuals it is the midpoint in

TABLE 7: *Mean adult molar wear (Molnar 1971 scale) for both sexes in five-year age groups*

Age group years	n	1	2	3	1	2	3
Females		Maxillary			Mandibular		
15-19	3	2.83	2.17	2.00	2.83	2.00	1.67
20-24	5	3.00	2.30	1.10	2.90	2.20	1.11
25-29	7	4.31	2.55	2.00	4.00	2.92	1.71
30-34	4	3.40	2.60	2.00	3.63	2.50	2.00
35-39	7	4.78	3.30	2.00	4.91	3.80	2.33
40-44	4	3.00	2.00	2.00	4.67	2.67	-
45-49	4	8.00	3.00	3.17	4.67	4.67	3.00
Males							
15-19	5	2.63	2.11	2.00	2.90	2.00	1.50
20-24	1	3.00	2.00	2.00	3.00	2.00	2.00
25-29	8	3.73	2.50	2.00	3.47	2.73	2.00
30-34	10	4.87	3.56	2.60	4.69	3.83	2.53
35-39	4	6.57	5.71	4.00	6.33	6.29	4.33
40-44	1	7.00	-	4.00	6.50	4.50	6.00
45-49	1	8.00	7.00	-	-	8.00	8.00

TABLE 8: *Pubic symphysis scores in the base group of young adult males. 'Todd' scores are based on the Todd (1921) scheme with modifications by Brooks (1955) and 'McKern/ Stewart' on McKern and Stewart (1957)*

Age group	n	Todd	McKern/Stewart
15-19	5	0-2	0-4
20-24	3	2-5	4-7
25-29	2	3-5	6-10

a range of approximately two years in individuals aged 5-20 years, five years in individuals aged 21-30 years and ten years in older individuals.

The ages estimated on the basis of the parameters originally selected were adopted, as the requirement of multiple age indicators was satisfied despite the contradictions and failures of the additional methods attempted.

TABLE 9: *Life table*

x	n	dx	lx	qx	Lx	Tx	Ex	Cx
All individuals (n=154)								
0-4	74	48.1	100.0	0.481	76.0	1590.9	15.91	23.9
5-9	7	4.5	51.9	0.088	49.7	1211.0	23.31	15.6
10-14	5	3.2	47.4	0.068	45.8	962.7	20.31	14.4
15-19	8	5.2	44.2	0.118	41.6	733.8	16.62	13.1
20-24	8	5.2	39.0	0.133	36.4	526.0	13.50	11.5
25-29	15	9.8	33.8	0.288	28.9	344.2	10.19	9.0
30-34	14	9.1	24.0	0.378	19.5	199.7	8.31	6.0
35-39	11	7.1	14.9	0.478	11.4	102.3	6.85	3.6
40-44	5	3.3	7.8	0.417	6.2	45.5	5.83	2.0
45-49	6	3.9	4.5	0.857	2.6	14.6	3.21	0.8
50+	1	0.6	0.6	1.000	0.3	1.6	2.50	0.1
Female adults (n=36)								
15-19	3	8.3	100.0	0.083	95.8	1861.1	18.61	
20-24	5	13.9	91.7	0.152	84.7	1381.9	15.08	
25-29	7	19.5	77.8	0.250	68.1	958.3	12.32	
30-34	4	11.1	58.3	0.190	52.8	618.1	10.60	
35-39	7	19.5	47.2	0.412	37.5	354.2	7.50	
40-44	4	11.1	27.8	0.400	22.2	166.7	6.00	
45-49	5	13.9	16.7	0.833	9.7	55.6	3.33	
50+	1	2.8	2.8	1.000	1.4	6.9	2.50	
Male adults (n=32)								
15-19	5	15.6	100.0	0.156	92.2	1437.5	14.38	
20-24	3	9.4	84.4	0.111	79.7	976.6	11.57	
25-29	8	25.0	75.0	0.333	62.5	578.1	7.71	
30-34	10	31.3	50.0	0.625	34.4	265.6	5.31	
35-39	4	12.5	18.8	0.667	12.5	93.8	5.00	
40-44	1	3.1	6.3	0.500	4.7	31.3	5.00	
45-49	1	3.1	3.1	1.000	1.6	7.8	2.50	

x = age interval - span five years. n = number of individuals dying at age x. dx = number of individuals dying at age x expressed as a proportion of a cohort of 100 individuals. lx = Survivorship, or the number of individuals living at age x, obtained by subtracting the number of deaths from the number alive at the beginning of each age interval. qx = mortality rate, or the number of those dying expressed as a proportion of the living ($qx = ndx/lx$). Lx = number of individuals alive between x and x+1 ($Lx = ((lx + (lx+1))/2)$ multiplied by the span of each age interval). Tx = The number of years that can be lived from the beginning of the age interval until all have died ($Lx = sum + remaining Lx$). Ex = Life expectancy, or the average number of years of life remaining at beginning of age interval ($Ex = Tx/lx$). Cx = Percentage of population who are age x in an age distribution (from Howell 1982:264)

DEMOGRAPHY - MORTALITY

The adaptation of standard demographic techniques for use in palaeodemography has required the assumption that a number of characteristics can be attributed to the population or sample being studied. There has been a long-standing debate in the literature on the value of palaeodemography based on skeletal material, because of these assumptions. Many of the problems raised have been solved, or recognised as deficiencies which must be accepted, and discussion has focussed on the fundamental problem of the validity of aging methods (e.g. Bocquet-Appel and Masset 1982, Van Gerven and Armelagos 1983, Bocquet-Appel and Masset 1985, Buikstra and Konigsberg 1985, Greene *et al.* 1986), stimulating much of the recent research into aging methods. However, some authors (e.g. Angel 1969, 1971, Cook 1984) express reservations about the use of life tables in analyses of prehistoric groups. The problem with the use of life tables on small samples has been succinctly stated by Smith (1960):

The precise methods used by actuaries in the construction of life tables are too involved to be of general interest or significance. They suffer from many of the defects that arise when highly refined mathematical techniques are applied to observations or measurements which themselves are grossly lacking in precision. (Smith 1960:364, cited in Timiras 1972:589)

The main assumptions which must be made in using basic census data from a small skeletal sample as a population for demographic analysis are:

1. That the data are from a complete biological population or are representative of the population. Most skeletal populations appear to have an under-representation of infants with very low infant mortality rates in comparison with those shown in historical data, in ethnographically recorded demography, or in some skeletal groups. This may reflect the action of taphonomic processes, differential age-related mortuary practices or sampling error. Wide deviations from a 1:1 sex ratio, or unusual fluctuations in numbers of individuals in various age groups, may also reflect these factors.
2. That the population is 'stable' in that there is no migration and the size and age structure of the population is constant. Stochastic fluctuations can cause the size of small populations to deviate significantly from predictions based on stable population theory (Moore *et al.* 1975), but the time depth involved in skeletal populations covering several generations results in the smoothing out of these fluctuations (Sattenspiel and Harpending 1983).
3. That the estimates of age at death and sex are accurate. The background to this has already been covered in some detail.

Accepting these assumptions clearly requires suspension of belief in a number of key characteristics about the population. With this caveat, for the record, the demography life tables have been prepared for the entire group and separately for males and females (Table 9). The age structure of the group is also depicted in the form of a histogram (Fig. 13).

Mean ages at death are as follows:

1. All individuals: 14.37 years.
2. Females 15 years of age and over: 32.17 years.
3. Males 15 years of age and over: 28.56 years.
4. All adults 15 years of age and over: 30.47 years.

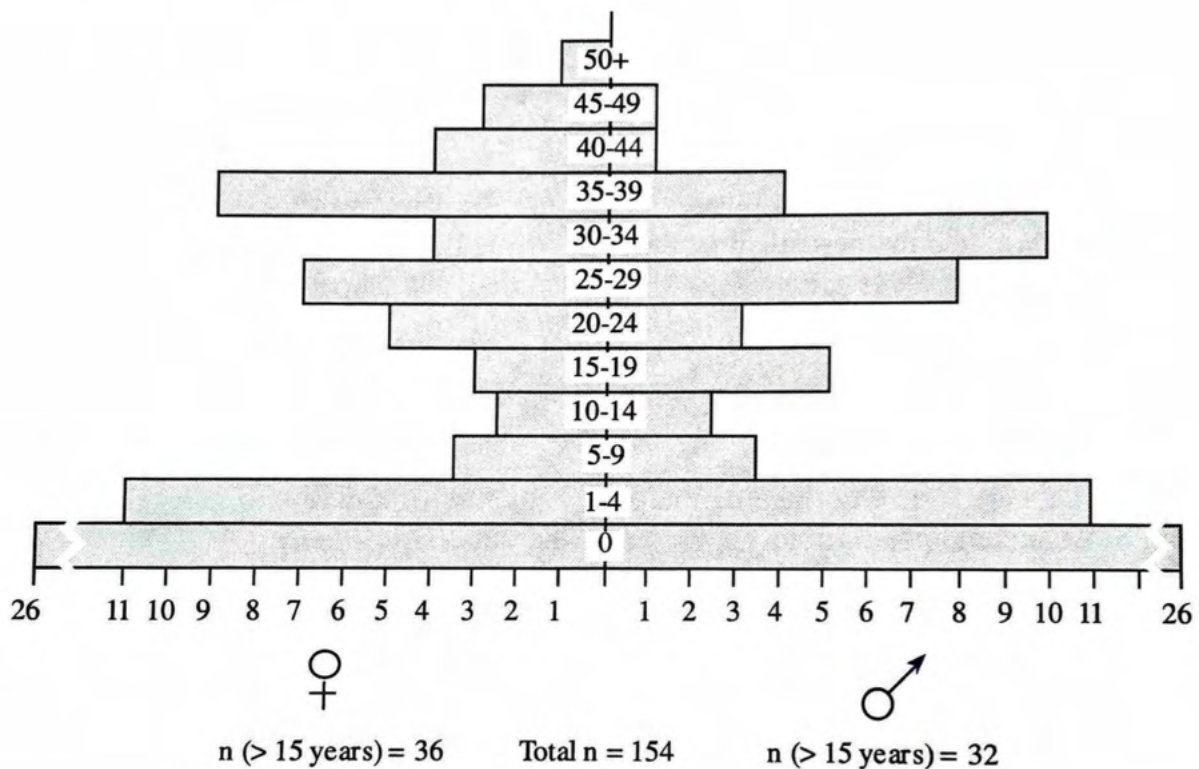


FIG. 13. Age structure, by sex, of the sample from Khok Phanom Di. Individuals less than 15 years of age have been equally divided between males and females

DISCUSSION OF CENSUS AND MORTALITY

The estimation of sex and age at death of the Khok Phanom Di sample has been in some ways easier and in others more complex than with other skeletal samples because of the very good preservation of the skeletons. The possibility of using multiple indicators improves the chances of arriving at the correct answer and creates confidence in the results, particularly for sex estimation and for the relative, if not the absolute, ages at death. However, the greater number of indicators available for

TABLE 10: *Number of burials and proportions of adults and children in each mortuary phase*

	< 5 years		Children		< 15 years		Adults > 14 years				Total
	n	%	n	%	n	%	Females n	Males n	All adults n	%	Burials n
MP1	3	50	0	0	3	50	1	2	3	50	6
MP2	35	63	2	3	37	66	11	8	19	34	56
MP3	23	55	0	0	23	55	9	10	19	45	42
MP1-3	61	59	2	2	63	61	21	20	41	39	104
MP4	5	17	5	17	10	34	10	9	19	66	29
MP5	2	50	0	0	2	50	1	1	2	50	4
MP6	4	33	2	17	6	50	4	2	6	50	12
MP7	2	40	3	60	5	100	0	0	0	0	5
MP4-7	13	26	10	20	23	46	15	12	27	54	50
Total	74	48	12	8	86	56	36	32	68	44	154
(MP5-7	8	38	5	24	13	62	5	3	8	38	21)

each individual also emphasises both the possibility of conflicting interpretations, and the extent of variability both between and within the sexes and age groups.

Age structure, sex ratio, and representativeness of the sample

Evidence from the earlier excavations at Khok Phanom Di suggest that the cemetery extended far across the mound, so that the skeletons from the 1985 excavation probably comprise only a small proportion of the cemetery population. The question of whether the sample is representative of the entire cemetery population can only be assessed from the adult sex ratio, which should be close to unity, and a comparison with the age structure found in other prehistoric groups and with ethnographic data (Waldron 1994).

The sex ratio of adults is close to unity at 1.12:1 females:males (females = 53 per cent, males = 47 per cent of the group). A one-sample t-test shows this difference from the expected ratio of 1:1 not to be significant. The histogram and life table for the complete group show a high level of infant deaths, low levels of mortality amongst children aged 5–15, an increase in deaths from age 15 to late thirties and a gradual decrease in the numbers who survived into the late forties. A single individual, the oldest in the sample, survived into the early fifties. When the mortality rate from the life table is plotted (Fig. 14) this shows the 'U' shaped curve characteristic of mortality in most human populations (Acsádi and Nemeskéri 1970, Waldron 1994).

The proportion of infants dying at birth or within the first year of life, at 41 per cent of the entire group, is within the range recorded in ethnographic data collected from preindustrial populations (20–60 per cent; Weiss 1973a) and above that recorded from a variety of skeletal populations where

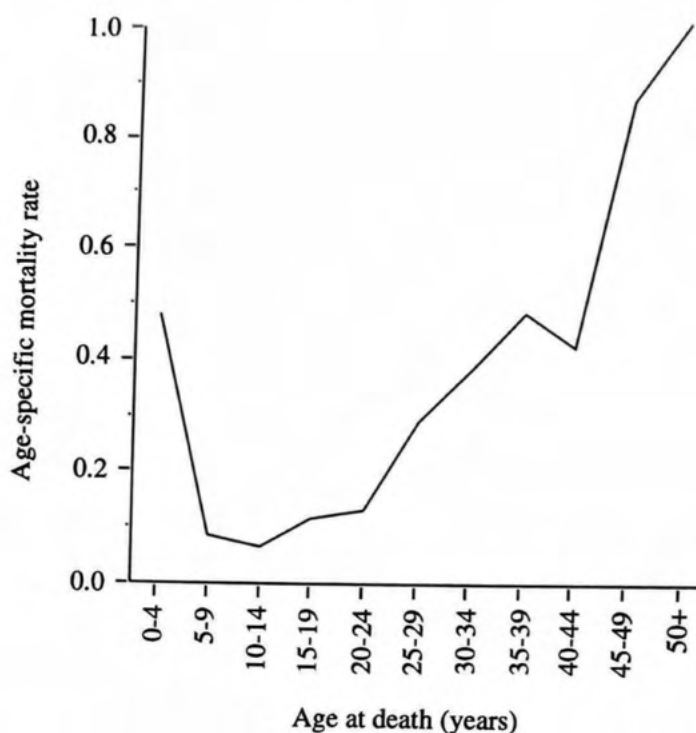


FIG. 14. Age-specific mortality rate (q_x) at Khok Phanom Di

entire cemeteries were excavated (13–30 per cent; Acsádi and Nemeskéri 1970). In the latter, however, the possibilities of lack of recovery of infant skeletons either because of poor preservation of small bones or because of the exclusion of infants from the cemetery cannot be discounted. The relatively low numbers of deaths of children aged 5–15 years results in a survivorship at age 15 (l_{15}) of 0.44, which is within the range recorded from ethnographic data (0.3–0.7; Weiss 1973a) and at the lower limit of the range recorded from skeletal populations (0.4–0.6; Acsádi and Nemeskéri 1970). Waldron (1994:23) suggests that a representative sample should include ‘..a substantial number of juveniles, perhaps up to 30 per cent.’, although he gives no basis for this assessment. Assuming his ‘juvenile’ age range is up to 14 years, the proportion at Khok Phanom Di, at 56 per cent (table 10) is almost twice this rate. The mortality rates of infants and children are further discussed in chapter IV.

The age structure of the adults and hence the representativeness of the sample is difficult to assess without recourse to the life tables, despite their acknowledged limitations. The life expectancy calculated in the life tables (E_x) is compared with data provided by Weiss (1973a). Weiss provides data for life expectancies at age 15 (E_{15}) for a variety of skeletal series with a range of 12 to 28 years (graduated data). He notes that populations with E_{15} of less than 15 years are suspected of underrepresentation of older individuals, although the method of aging will affect non-graduated

data. The life expectancy at age 15 in the Khok Phanom Di sample of 16.62, although at the lower level, is within this range.

In summary, the demographic characteristics of the sample as a whole are within the ranges which could be expected from such a population and the sample may be representative of the age and sex composition of the entire cemetery. Nevertheless, the following analysis should be read with the caution that without a complete excavation of the cemetery, acceptance of the sample as representative of the population as a whole is provisional.

Chronological differences in demography

The age composition of the cemetery sample changes over time. The age and sex composition of burials for each mortuary phase is listed in table 10. Subtotals are given for early (MP1-3) and late (MP4-7) burials. This approximates a subdivision into groups pre-dating and post-dating the major environmental change.

The caution regarding the representativeness of the sample applies particularly to the small sub-samples from each mortuary phase. The age composition of the burials varies markedly over time. In MP1-3, there are very high levels of infant mortality and virtually no older children in all three phases. The sample is almost two-thirds infants, one third adults, with female and male adults equally represented. In MP4-7 the sample is almost half infants and children, half adults, but the pattern is not consistent over mortuary phases. In MP4 there is a low level of infant mortality and an equal proportion of older children. Infant mortality is again high and child mortality minimal in MP5-6. In MP7 there are no adult burials and older children form most of the sample. Figure 15 illustrates the age composition of the mortuary phases.

Despite the apparent representativeness of the sample as whole, the variation in infant and child mortality over time raises the possibility that the samples from some mortuary phases are not representative. Preservation of bone was as good during the later mortuary phases as in the early phases so the low numbers of infant skeletons cannot be assumed to represent differential preservation. A possible explanation is simply that the spatial distribution of burials was responsible for the exclusion of older infants and children in MP1-3 and neonates in MP4-7 from the excavation square. This cannot be tested without further excavation at the site but the spatial continuity and mixed age composition of the clusters argues against this explanation.

Demographic factors which may have been responsible for creating an apparent change in mortality include the possibility that the population was not stable. The emigration, or burial elsewhere in the cemetery, of parents of infants who were buried during MP2 and 3 would result in a relatively high proportion of infants in the sample. This imbalance would be exacerbated if the emigration included surviving but weak children, whose removal could account for the apparent lack of deaths in older children during MP3. An alternative explanation for the variation in the proportion of infants is discussed further in chapter VIII, in the light of the results of the study of health.

The adult sex ratio, which is close to 1:1 in all phases except in the small samples from MP5-6, is more positive evidence that the samples are representative of adult deaths.

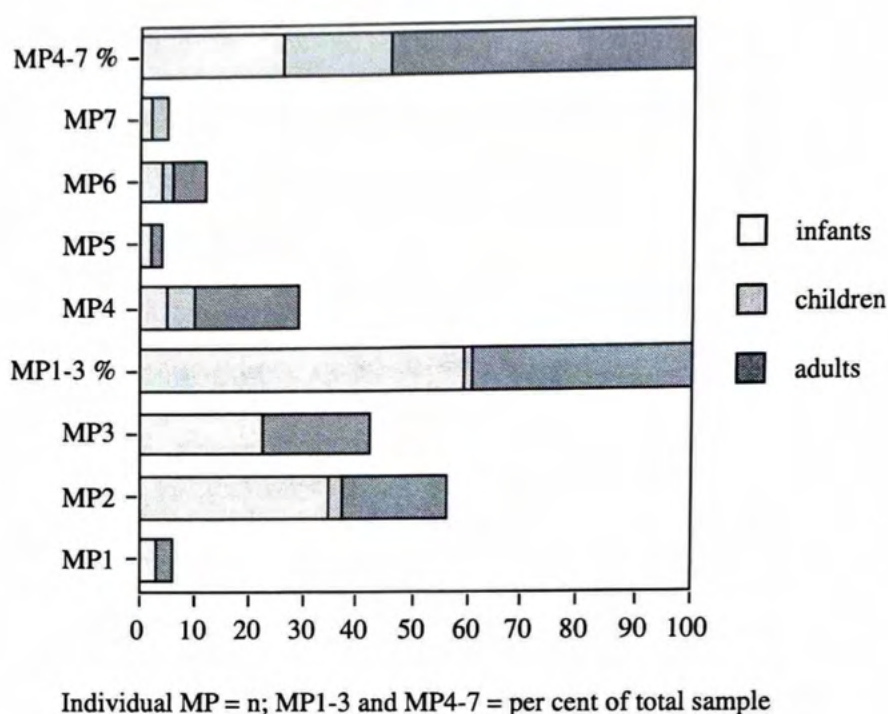


FIG. 15. Age structure of the burials from each mortuary phase. The bars for individual mortuary phases represent numbers of burials. The bars for MP1-3 and MP4-7 summarise the proportion of infants, children, and adults in the early and late phases

Validity of the range of estimates of adult ages

Adult mortality is highest between the ages of 25–40, with few surviving beyond this age and the oldest aged *c.* 50 years. This is a pattern often described in skeletal populations but questioned by Weiss (1973b) and Howell (1982) as has previously been noted. Howell has examined the biological viability of such populations using the data from the Libben site. This is an Amerindian site with a large cemetery ($n=1327$) which was completely excavated (Lovejoy *et al.* 1977), enabling Howell to make a detailed study of the demographic structure of the living population represented by the burials. Howell compares the Libben survivorship (*lx*) levels with those represented by the 'West' model life tables published by Coale and Demeny (1966), based on modern western populations and those of a series of national populations from the last century with high rates of infant mortality (Preston *et al.* 1972). The Libben population shows a very different mortality pattern from these populations, both in infants and adults. Comparison of Khok Phanom Di with the same data (Fig. 16) shows that survivorship in infants, children and young adults closely follows the model tables, but from ages 25–30 deviates sharply from the pattern to follow that of the Libben population closely.

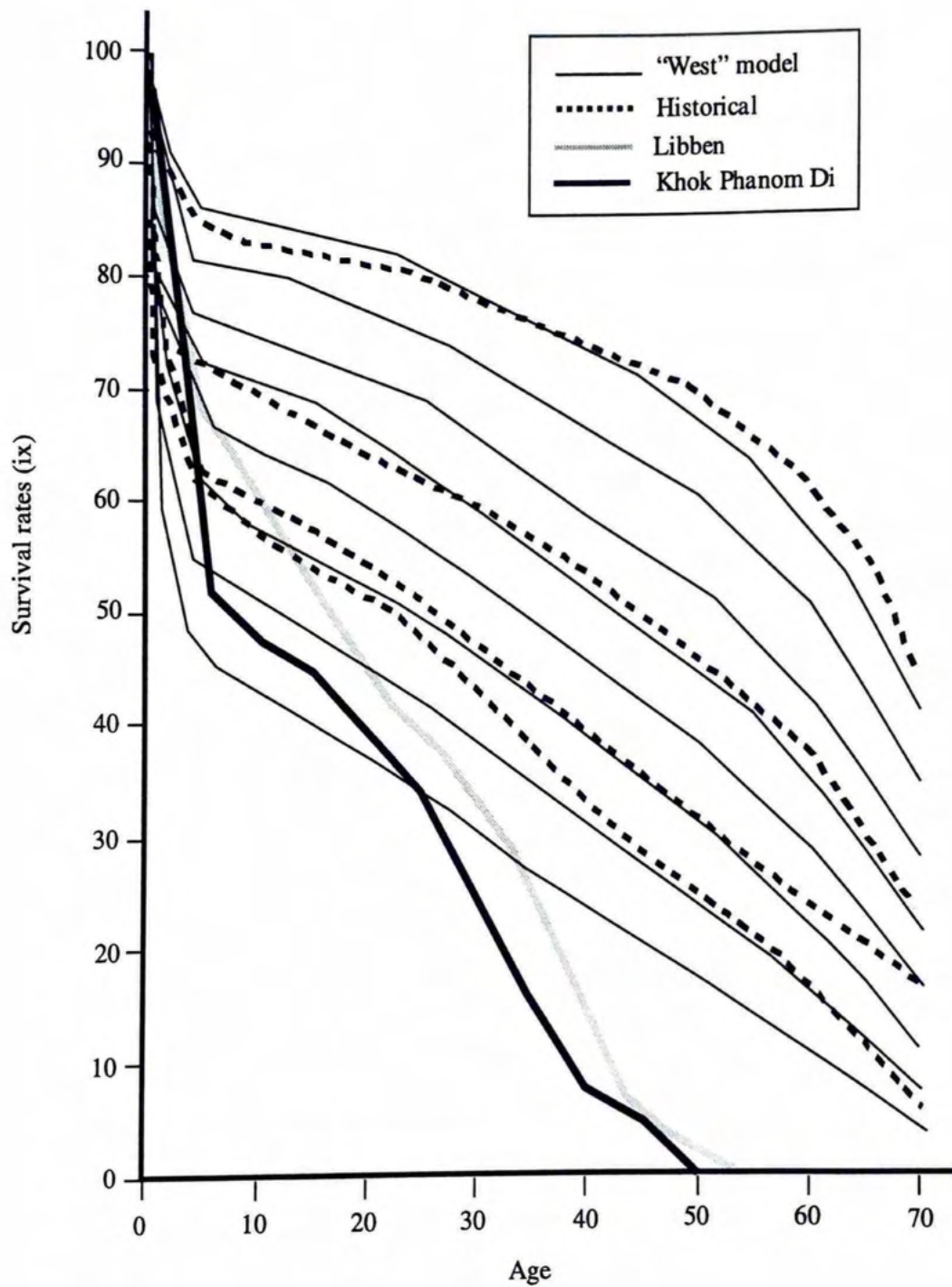


FIG. 16. Survival rates, which show the percent surviving from birth to each age, for Khok Phanom Di compared with the Libben population (Lovejoy *et al.* 1977). 'West' model life tables are based on modern western countries (Coale and Demeny 1966) and historical data on populations with high infant mortality rates (Preston *et al.* 1972). Adapted from Howell (1982:265)

Howell then translates the Libben information into the living population represented by the skeletons. The living population represented by the Khok Phanom Di sample, although similar in structure to that of the Libben population in that all adults are dead by *c.* 50 years, has an even smaller proportion of adults to subadults than Libben (Fig. 17).

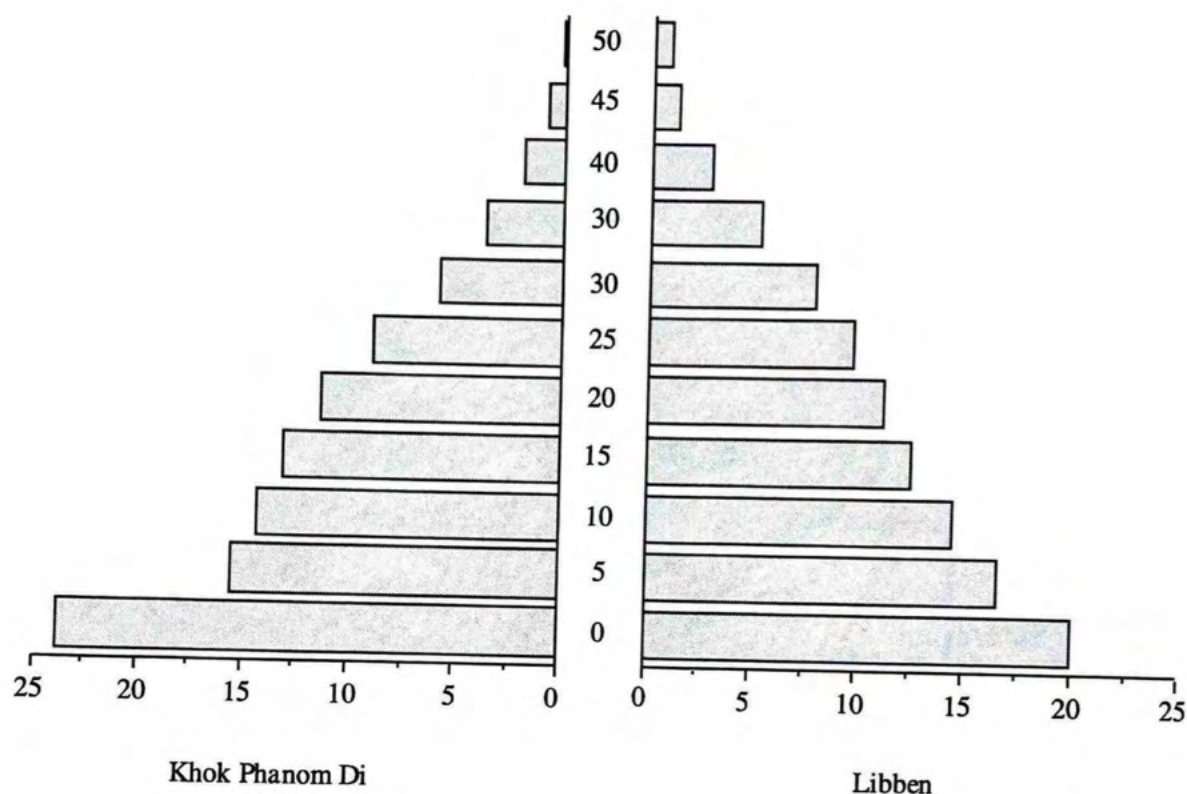


FIG. 17. Age structure of living population in 5-year age groups (Cx) at Khok Phanom Di and Libben (Libben after Howell 1982)

It is not possible to translate the Khok Phanom Di information into actual figures because the size of the complete skeletal population is not known. Environmental conditions at the two sites show some similarities, with both apparently having access to rich, plentiful and varied food supplies. Both sites could have experienced seasonal shortages, with severe winters at Libben and the monsoon and dry seasons at Khok Phanom Di. From her study, Howell draws the following conclusions:

The living population that produced a cemetery with the age distribution of that shown by Lovejoy *et al.*, at Libben would necessarily be a population in which adults had an extremely heavy work load and in which the kinship relations differed from any group known by extremely high rates of orphanage and only two generations alive at the same time. (Howell 1982:268)

This situation is a reflection of the high mortality rates in young adults and, as Howell notes, the possible explanation is either that the skeletal mortality levels are genuine and life was hard, or that the life table includes sources of error or bias. The difficulty of estimation of age is the most likely explanation if the latter is the case. Projection of a theoretical age distribution for Khok Phanom Di, with a more consistent mortality rate in the age groups over 25 years and consequent aging of the oldest individuals at c. 60 years, still produces survivorship rates at variance with the model populations. The model populations, together with some historical groups, show the oldest adults dying at age 70–80. However, these age groups are represented by such small numbers, it is possible that none of the few elderly people in the cemetery are included in a small sample the size of that excavated. When the structure of the living population represented by the cemetery is considered in comparison with Howell's analysis of the living population at Libben, a similar conclusion is reached. This shows a population with a high proportion of subadults, resulting in adults having a difficult life in caring for the young and providing for the entire group. The problem of aging the oldest individuals, with consequent underaging, may be the explanation for this apparent population structure, but recalculation of the life table using a theoretical age structure with the high numbers of deaths in midlife spread more evenly over subsequent decades still does not produce an age structure similar to the model populations used by Howell for comparison.

The age structure represented is similar to that estimated for many prehistoric groups, so despite any question of the viability of the population, there is insufficient evidence for defining a different structure.

DEMOGRAPHY - FERTILITY

The method of identifying female fecundity involves alterations to the pelvis as a consequence of pregnancy. All female pelves were examined for evidence of pitting at the preauricular sulcus of the ilium and at the dorsal pubis. Figure 18 illustrates examples of the pitting at each site.

The degree of pitting for both the dorsal pubis and the preauricular groove for all females is listed in table 16 and summarised in table 11. The identification is 'nulliparous' where there was no pitting on either site or 'parous', where there was pitting on one or both sites. Of the 36 females aged 17 years and over at death, all were sufficiently complete to be assessed for pitting on at least one side of the preauricular groove, and 31 were sufficiently complete to be assessed for pitting on at least one side of the dorsal pubis. All except B142, which was assessed only at the preauricular groove, and B140 which was aged 17 years at death, showed some degree of pitting at either or both sites. Pitting was more common at the preauricular groove than at the dorsal pubic bone. Of the 31 individuals able to be assessed at both sites, only four had the same score at both. Six of the eight individuals with a higher degree of pitting at the dorsal pubis than at the preauricular groove were aged over 35 years at death. In all those individuals with the greatest degree of pitting (grades 3 and 4) at either site, the pitting at the second site was minimal. In those cases where both left and right bones were present for either the preauricular groove or the dorsal pubis, neither side showed consistently greater pitting than the other, and in no case did the scores vary by more than one grade between the sides.

TABLE 11: *Prevalence of pregnancy pitting on the pelves of adult females*

	N	n	%		N	n	%
Preauricular groove	36	31	83	Dorsal pubis	31	21	68
Both sites	31	17	55	Total with pitting at either site	36	34	94

As most studies on pelvic pitting discuss only one of the sites, Ullrich (1975) provides the most useful study for comparison. He found a similar pattern to that at Khok Phanom Di, in that a larger proportion of individuals had pitting at both sites, or only at the preauricular groove, than only at the dorsal pubis. This is possibly a reflection of the transmission of the weight of the upper body through the sacro-iliac joint and therefore the greater stress on these ligaments during pregnancy (Houghton 1974).

When inferences as to fecundity in the females are drawn from this evidence, all individuals over the age of 17 at death had the possibility of being fecund, with B142 the only individual with no extant evidence of parity. The patterns of pitting of the preauricular groove and the dorsal pubis in the Khok Phanom Di females do not suggest any correlation of the degree of pitting with fertility. The ages at death of the individuals with the greater degree of pitting at the preauricular groove argue against a positive correlation. Both those with grade 4 (the most extensive pitting), are aged under 25. Burial 25, aged 21 years at death, has grade 3 pitting and of the ten individuals aged 40 or older, only one has pitting higher than grade 1. The degree of dorsal pubic pitting appears to be more variable within the group, and with fewer recordings possible and more individuals having no pitting, there is no discernible pattern. The high prevalence of older females with a greater degree of pitting at the dorsal pubis compared with the preauricular groove raises the possibilities that the lower scores at the preauricular groove reflect remodelling of the bone at this site with age (e.g. burials 27, 61), and that the higher scores at the dorsal pubis reflect arthritic degeneration at the symphysis and a consequent apparent increase in the depth of the pitting through lipping of the borders of the symphysis (e.g. B112). Therefore no assumptions will be drawn on individual fertility at Khok Phanom Di from the pitting, other than the evidence for almost universal fecundity. Cox and Scott (1992) also found no significant relationship between the pitting and known parity status in their series from the Spitalfields excavation.

DEMOGRAPHY - INTERSITE COMPARISON

The largest four of the previously excavated South East Asian prehistoric sites with demographic data available are used as a basis for a brief comparison with the Khok Phanom Di sample (Table 12). As different age groupings have been used in these reports in calculating demographic statistics, the raw data have been used to recalculate summary data with standardised age groups to improve the comparability of the results. The age groups used are those adopted by Pietrusewsky (1974c), as this is the only source of detailed mortality data from the 1968 season at Non Nok Tha.

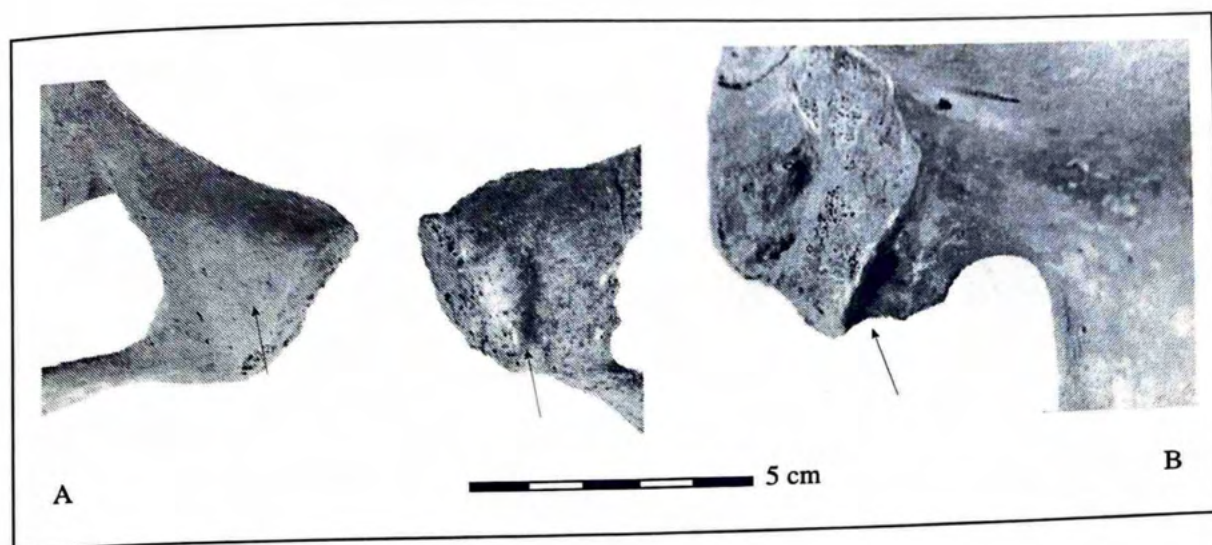


FIG. 18. A: Examples of dorsal pubic bones from females with pregnancy pitting of grade one on the left and grade four on the right. B: An example of pitting of grade four on the preauricular groove of a female pelvis

The use of broad age groups (15 years) for the adults will reduce the chance of results reflecting interobserver variation in aging of individuals rather than actual variations in adult longevity.

TABLE 12: *Demographic structure of comparative prehistoric samples*

	Infants	Children	Adults > 16 years			Total	Total
	0-1 years	2-16 years	Females	Males	Unknown		All ages
Khok Phanom Di	70	18	36	30	-	66	154
Ban Kao	1	4	10	13	6	29	34
Non Nok Tha	4	43	39	50	3	92	139
Ban Chiang	10	26	43	32	-	75	111
Ban Na Di	16	8	15	18	-	33	57

The sources of the data are as follows:- Ban Kao: calculated from the individual descriptions in Sangvichien *et al.* (1969). Non Nok Tha: samples from both excavation seasons as reported by Pietrusewsky (1974c). Ban Chiang: from Pietrusewsky (1984). Ban Na Di: recalculated from Houghton and Wiriyaromp (1984)

The sample sizes and age composition are also presented graphically in figure 19. Ban Kao is the smallest sample, and has very few infants and children. Non Nok Tha and Ban Chiang are similar sized samples, with fewer infants but more children and adults than Khok Phanom Di. Ban Na Di is a smaller group, but has a similar age composition to Khok Phanom Di.

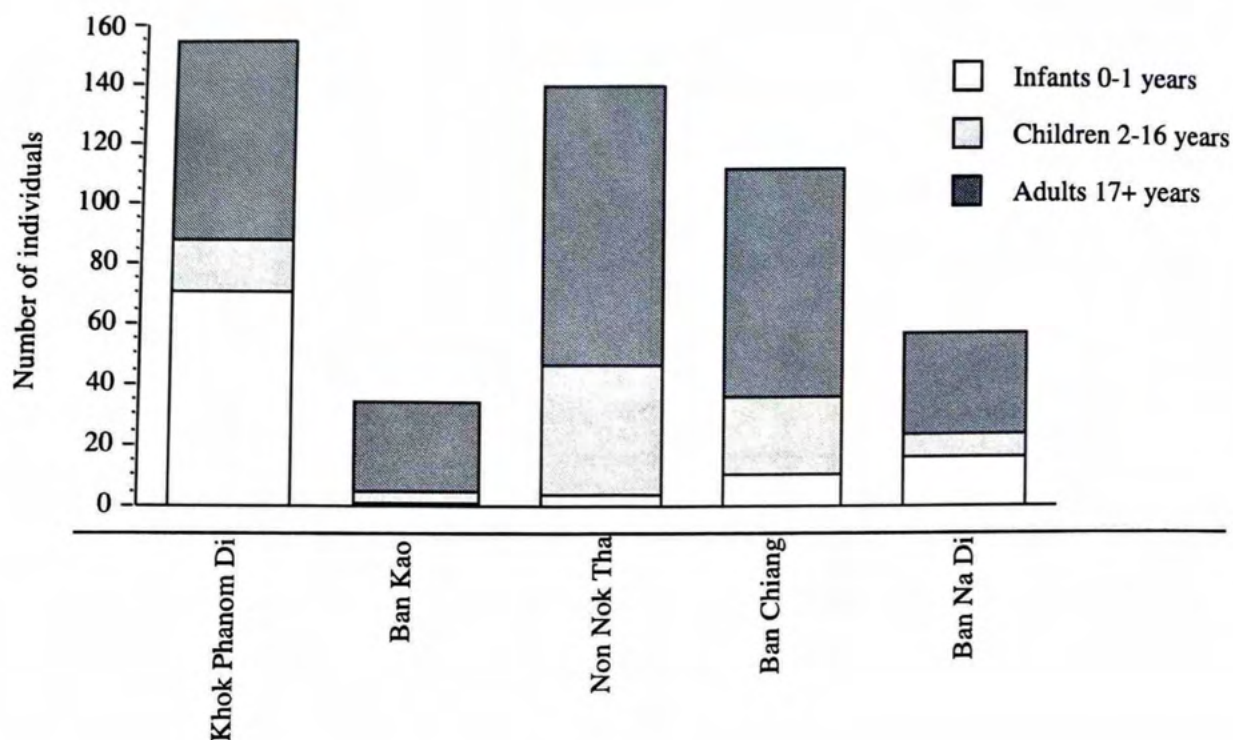


FIG. 19. Size and age composition of comparative prehistoric South East Asian skeletal samples (sources of data as for table 12)

The mortality data are plotted in figure 20. These reflect the differences in the composition of the samples, particularly in the proportions of infants and young children.

The question of the representativeness of the samples from the other sites is briefly considered here. Pietrusewsky (1974b, 1974c, 1984) questions the samples at Non Nok Tha and Ban Chiang, citing such factors as the necessary exclusion of poorly preserved adult skeletons from the demographic analysis (16 at Ban Chiang, 49 at Non Nok Tha), and the variations in age structure and sex ratios in subgroups representing different time periods.

Only at Khok Phanom Di and Ban Na Di is the mortality of infants within the ranges found in other skeletal and pre-industrial populations, as previously discussed. At Non Nok Tha and Ban Chiang, the high numbers of children aged 2–6 years suggests that preservation of the bone of infants was not a problem, and that neonates and infants dying within the first year of life may have been buried elsewhere or disposed of in some other manner. Pietrusewsky (1984) records that at Ban Chiang the numbers of infants varies over time, with the highest proportion recovered from an early phase during which infants were buried in jars. The virtual absence of young children at Ban

Kao may reflect the same selective burial practices, but it is also possible that the conditions were not favourable for preservation or recovery of the smaller skeletons.

The adult burials include a preponderance of males in three sites (Ban Kao, Ban Na Di and Non Nok Tha) in a ratio of 1.2/1.3:1. At Ban Chiang the ratio is reversed, with more females, as at Khok Phanom Di. At all sites, the longest surviving individuals died before the mid-fifties. Life expectancies at age 17 are lowest in the Ban Kao sample at 13 years and range up to 19 years at Ban Chiang, but as variations in treatment of samples from different time periods, aging methods and age groupings have some influence on this statistic as on all other measures of adult longevity, the results are discussed in more detail in chapter IV.

The question of the representativeness of the sample of adults is difficult to answer because of the potential inter-observer variation in aging criteria, but the data suggest that the samples are not markedly imbalanced. In the case of sex estimation, the 12 per cent imbalance in the sex ratio in favour of males, evident in the largest four of the five Thai sites, is exactly the proportion noted in other skeletal reports by Weiss in his study of systematic bias in skeletal sexing (1972). Although the questions of the effect of sample selection and segregation of burial areas on representation of the sexes remain unresolved, this predominance of males is suggestive of consistent observer bias. The subadult samples are unlikely to be representative for Ban Kao, Ban Chiang and Non Nok Tha, but Ban Na Di is possibly closer to the true age composition of the total cemetery population.

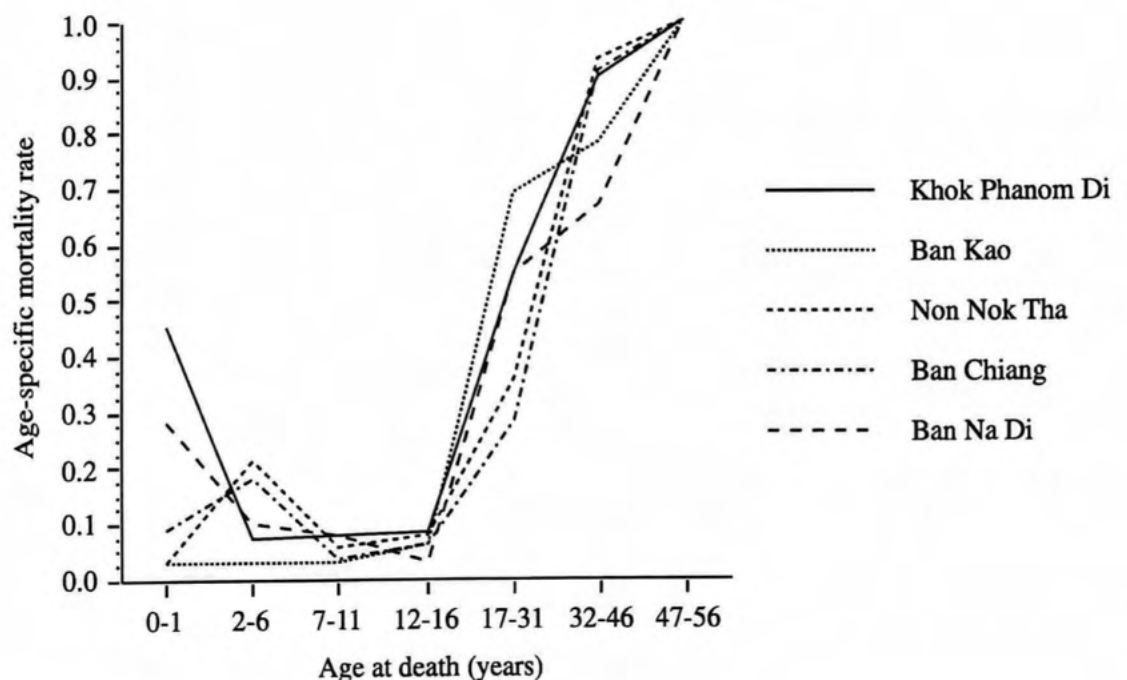


FIG. 20. Age-specific mortality rates of comparative prehistoric South East Asian skeletal samples (sources of data as for table 12)

TABLE 13: *Estimates of age and sex*

Burial	Age	Indicators of age	Sex	Indicators of sex
1	9.00	dental calcification		
2	0.75	dental calcification		
3	0.30	size		
4	26.00	skeletal maturation	female	pelvis, cranium, femur
5.1	0.00	diaphyseal length		
5.2	0.00	diaphyseal length		
6	9.00	dental calcification		
7	1.75	dental calcification, diaphyseal length		
8	15.00	skeletal maturation, dental calcification	male	pelvis
9	30.00	dental wear, pubic symphysis	male	pelvis, cranium
10	1.25	size, skeletal maturation		
11	12.00	dental calcification		
12.1	12.00	dental calcification		
12.2	5.00	dental calcification		
13	35.00	dental pathology, joint degeneration	female	pelvis, cranium
14	1.25	dental calcification, diaphyseal length		
15	35.00	dental wear, dental pathology	female	pelvis, cranium, femur
16	1.25	dental calcification, diaphyseal length		
17	12.00	dental calcification		
18	42.00	dental wear, dental pathology	female	pelvis, cranium, femur
19	25.00	dental wear	female	pelvis, cranium, femur
20	12.00	dental calcification		
21	8.00	dental calcification		
22	21.00	skeletal maturation, pubic symphysis	male	pelvis, femur
23	30.00	pubic symphysis, joint degeneration	male	pelvis, cranium, femur
24	25.00	dental wear, pubic symphysis	male	pelvis
25	19.00	skeletal maturation	female	pelvis, cranium, femur
26	35.00	dental wear, dental pathology	female	pelvis
27	41.00	dental wear, dental pathology	female	pelvis, femur
28	21.00	skeletal maturation, pubic symphysis	male?	pelvis
29	27.00	dental wear, pubic symphysis	male	pelvis, cranium, femur
30	34.00	dental wear, pubic symphysis	male	pelvis, cranium, femur
31	25.00	skeletal maturation, dental wear	female	pelvis, cranium, femur
32	11.00	dental calcification		
33	3.00	dental calcification, diaphyseal length		
34	0.00	dental calcification, diaphyseal length		
35	21.00	skeletal maturation	female	pelvis, cranium
36	21.00	skeletal maturation	female	pelvis, cranium
37	9.00	dental calcification		
38	33.00	dental wear, pubic symphysis	male	pelvis, cranium, femur
39	25.00	skeletal maturation	female	pelvis, cranium, femur
40	37.00	dental wear, dental pathology	female	pelvis, cranium, femur
41	0.75	dental calcification, diaphyseal length		
42	32.00	pubic symphysis, joint degeneration	male	pelvis, cranium, femur

Table 13: *Census: estimates of age and sex (cont.)*

Burial	Age	Indicators of age	Sex	Indicators of sex
43	30.00	dental wear, pubic symphysis	male	pelvis, cranium
44	18.00	skeletal maturation, dental calcification	male	pelvis, cranium
45	45.00	dental wear, dental pathology	female	pelvis, cranium, femur
46	0.30	dental calcification, diaphyseal length		
47	22.00	skeletal maturation	female	pelvis, cranium, femur
48	0.00	dental calcification, diaphyseal length		
49	0.00	dental calcification, diaphyseal length		
50	0.00	dental calcification, diaphyseal length		
51	0.00	diaphyseal length		
52	0.00	dental calcification, diaphyseal length		
53	0.00	size of vertebrae and ribs		
54	0.00	dental calcification, diaphyseal length		
55	0.00	diaphyseal length		
56	45.00	dental wear, dental pathology	female	pelvis, cranium, femur
57	26.00	dental wear, pubic symphysis	male	pelvis, cranium, femur
58	35.00	dental wear, dental pathology	female	pelvis, cranium, femur
59	0.00	diaphyseal length		
60	19.00	skeletal maturation	female	pelvis, cranium, femur
61	48.00	dental pathology, joint degeneration	female	pelvis, cranium, femur
62	0.50	dental calcification, diaphyseal length		
63	1.75	dental calcification, diaphyseal length		
64	21.00	skeletal maturation, dental calcification	female	pelvis, cranium, femur
65	0.00	dental calcification, diaphyseal length		
66	0.00	dental calcification, diaphyseal length		
67	35.00	dental wear, pubic symphysis	male	pelvis, cranium, femur
68	0.00	diaphyseal length		
69	0.00	dental calcification, diaphyseal length		
70	0.00	dental calcification, diaphyseal length		
71	0.00	diaphyseal length		
72	32.00	dental wear, pubic symphysis	male	pelvis, cranium, femur
73	25.00	skeletal maturation	female	pelvis, cranium, femur
74	39.00	dental wear, pubic symphysis	male	pelvis, cranium, femur
75	0.00	dental calcification, diaphyseal length		
76	34.00	dental wear, pubic symphysis	male	pelvis, cranium, femur
77	26.00	skeletal maturation	female	pelvis, cranium, femur
78	0.00	dental calcification, diaphyseal length		
79	47.00	dental pathology, joint degeneration	female	pelvis, cranium, femur
80	0.00	dental calcification, diaphyseal length		
81	0.00	diaphyseal length		
82	0.50	diaphyseal length		
83	30.00	dental wear	female	pelvis, cranium, femur
84	0.00	dental calcification, diaphyseal length		

Table 13: *Census: estimates of age and sex (cont.)*

Burial	Age	Indicators of age	Sex	Indicators of sex
85	0.00	dental calcification, diaphyseal length		
86	25.00	skeletal maturation, pubic symphysis	male	pelvis, cranium, femur
87	24.00	skeletal maturation	female	pelvis, cranium, femur
88	0.75	dental calcification, diaphyseal length		
89	0.50	dental calcification, diaphyseal length		
90	27.00	dental wear, pubic symphysis	male	pelvis, cranium, femur
91	45.00	dental wear, pubic symphysis	male	pelvis, cranium, femur
92	18.00	skeletal maturation, dental calcification	male	pelvis, cranium
93	42.00	dental wear, pubic symphysis	male	pelvis, cranium, femur
94	31.00	dental wear	female	pelvis, cranium, femur
95	0.00	size reported at excavation		
96	4.00	dental calcification, diaphyseal length		
97	0.50	diaphyseal length		
98	0.00	diaphyseal length		
99	0.60	dental calcification, diaphyseal length		
100	32.00	dental wear, pubic symphysis	male	pelvis, cranium, femur
101	1.50	dental calcification, diaphyseal length		
102	35.00	dental wear, dental pathology	female	pelvis, cranium, femur
103	26.00	dental wear, pubic symphysis	male	pelvis, cranium, femur
104	0.00	diaphyseal length		
105	0.30	dental calcification, diaphyseal length		
106	0.50	diaphyseal length		
107	40.00	dental pathology, joint degeneration	female	pelvis, cranium, femur
108	0.00	dental calcification, diaphyseal length		
109	31.00	dental wear	female	pelvis, cranium, femur
110	36.00	dental wear	female	pelvis, cranium, femur
111	0.00	diaphyseal length		
112	40.00	dental wear, dental pathology	female	pelvis, cranium, femur
113	46.00	dental pathology, joint degeneration	female	pelvis, cranium
114	0.00	dental calcification, diaphyseal length		
115	38.00	dental wear, pubic symphysis	male	pelvis, cranium
116	0.00	dental calcification, diaphyseal length		
117	16.00	skeletal maturation, dental calcification	male	pelvis
118	0.00	dental calcification, diaphyseal length		
119	0.00	dental calcification, diaphyseal length		
120	21.00	skeletal maturation, pubic symphysis	male	pelvis, cranium, femur
121	1.25	dental calcification, diaphyseal length		
122	52.00	dental pathology, joint degeneration	female	pelvis, femur
123	2.00	dental calcification, diaphyseal length		
124	0.00	dental calcification, diaphyseal length		
125	0.00	dental calcification, diaphyseal length		
126	0.00	dental calcification, diaphyseal length		

Table 13: *Census: estimates of age and sex (cont.)*

Burial	Age	Indicators of age	Sex	Indicators of sex
127	0.00	dental calcification, diaphyseal length		
128	0.00	dental calcification, diaphyseal length		
129	27.00	dental wear, pubic symphysis	male	pelvis, cranium, femur
130	0.50	dental calcification, diaphyseal length		
131	25.00	skeletal maturation, pubic symphysis	male	pelvis, cranium, femur
132	34.00	pubic symphysis, joint degeneration	male	pelvis, cranium, femur
133	0.40	dental calcification, diaphyseal length		
134	0.50	dental calcification, diaphyseal length		
135	0.30	dental calcification, size of cranium		
136	0.00	dental calcification, diaphyseal length		
137	5.00	dental eruption, diaphyseal length		
138	0.00	dental calcification, diaphyseal length		
139	0.00	diaphyseal length		
140	17.00	skeletal maturation, dental calcification, pubic symphysis	female	pelvis, cranium, femur
141	0.00	dental calcification, diaphyseal length		
142	31.00	dental wear	female	pelvis
143	0.00	dental calcification, diaphyseal length		
144	0.00	dental calcification, diaphyseal length		
145	8.00	dental calcification		
146	0.00	dental calcification, diaphyseal length		
147	19.00	skeletal maturation, dental calcification, pubic symphysis	male	pelvis, femur
148		not included		
149		not included		
150	2.50	dental calcification, diaphyseal length		
151	0.00	dental calcification, diaphyseal length		
152	37.00	dental wear, pubic symphysis	male	pelvis, cranium, femur
153	0.00	dental calcification, diaphyseal length		
154	25.00	dental wear	female	pelvis, cranium, femur

Table 15: *Maximum diaphyseal lengths of infants, children and adolescents (cont.)*

Burial	Age	Humerus		Radius		Ulna		Femur		Tibia	
		L	R	L	R	L	R	L	R	L	R
84	0.00	63.0	63.0					76.0	76.0	66.0	66.0
85	0.00		60.0								
98	0.00	66.0							77.0		
104	0.00	63.0	64.0					73.0	74.0		
108	0.00								75.0		65.0
111	0.00		64.0			59.0					
114	0.00	62.0	62.0	55.0	55.0		60.0	74.0		66.0	66.0
116	0.00							79.0	79.0		69.0
118	0.00		65.0	54.0	54.0		61.0	74.0	74.0	68.0	68.0
119	0.00		66.0	51.0		59.0	59.0	76.0	76.0	66.0	66.0
124	0.00		65.0		53.0	62.0		76.0	76.0	62.0	62.0
125	0.00	65.0	65.0		52.0		59.0	77.0	77.0	69.0	
126	0.00	64.0	64.0				61.0		75.0		64.0
127	0.00	67.0	67.0		53.0		61.0	75.0	75.0	67.0	67.0
128	0.00	68.0	68.0			64.0	64.0	78.0	78.0	68.0	68.0
136	0.00	65.0	65.0	52.0	52.0	60.0	59.0	74.0	74.0	65.0	65.0
138	0.00	60.0	60.0		50.0	60.0		73.0	73.0	62.0	62.0
139	0.00		60.0			59.0				62.0	
141	0.00		63.0					74.0	74.0	63.0	64.0
143	0.00	64.0	64.0	51.0	51.0	58.0	58.0	75.0	75.0	65.0	65.0
144	0.00	64.0	63.0	51.0	51.0	58.0	58.0	74.0	74.0	64.0	64.0
146	0.00		64.0		50.0		58.0	72.0	71.0	63.0	63.0
151	0.00	65.0	65.0	55.0	55.0	63.0	63.0		76.0	69.0	69.0
153	0.00		66.0						76.0		
46	0.30	74.0	74.0					86.0	88.0		73.5
105	0.30				67.0			88.0	87.0	78.0	78.0
135	0.30										
133	0.40	81.0	81.0		62.0	70.0	70.0	101.0	101.0	86.0	86.0
62	0.50	95.0	95.0	78.0		87.0	87.0		115.5	113.0	
82	0.50		82.0			74.0			99.0	86.0	86.0
89	0.50	87.0	86.0	75.0				101.0			
97	0.50							107.0			
106	0.50		84.0				74.0				69.0
130	0.50	91.0									
134	0.50	79.5	79.5		64.0	72.0	72.0		98.0		
99	0.60		86.0								
2	0.75	85.0									
41	0.75		94.0	74.0	73.0	82.0	82.0	114.0	112.0		95.0
88	0.75	101.0	93.0	80.0	80.0	89.0	90.0		125.0	109.0	
10	1.25										
14	1.25	91.0									102.6
16	1.25	99.5	98.5		89.0		91.5		122.7	103.8	
121	1.25		101.0				89.0				

EXCAVATION OF KHOK PHANOM DI

Table 15: *Maximum diaphyseal lengths of infants, children and adolescents (cont.)*

Burial	Age	Humerus		Radius		Ulna		Femur		Tibia	
		L	R	L	R	L	R	L	R	L	R
101	1.50		144.0	88.0		96.0		143.0	143.0	120.0	
7	1.75							125.0		118.0	
63	1.75							133.0	133.0		115.0
123	2.00	117.0	116.0	90.0	90.0	92.0	93.0	150.0	150.0	126.0	126.0
150	2.50						96.0		145.0		
33	3.00	129.0						174.0			
96	4.00										
12	5.00	154.0									149.0
137	5.00		148.0	117.0		132.0	134.0		209.0		179.0
21	8.00	185.0	188.0	150.0				268.0	264.0		233.0
145	8.00	193.0	193.0	155.0	157.0	170.0	171.0	270.0	270.0	232.0	232.0
1	9.00		186.0								220.0
6	9.00		207.0	168.0		188.0					
37	9.00	214.0	213.0	169.0	169.0	187.0	189.0	296.0	293.0		
32	11.00		205.0	166.0	164.0		183.0	292.0	292.0		
11	12.00	211.0		165.0	167.0	181.0		301.0		250.0	245.0
12	12.00				143.0			261.0			250.0
17	12.00		205.0	166.0	169.0	183.0	189.0		300.0		263.0
20	12.00				180.0	200.0		327.0		277.0	273.0
8	15.00	263.0	263.0		204.0		221.0	352.0			300.0
117	16.00						253.0				

TABLE 16: *Presence and degree of "pregnancy" pitting at the dorsal pubic bone (DPB) and the preauricular groove (PG) of the ilium of all adult females*

Burial	Age	PG		DPB		Burial	Age	PG		DPB		Burial	Age	PG		DPB	
		L	R	L	R			L	R	L	R			L	R	L	R
4	26	0	1	3	4	13	35	-	1+	-	-	15	35	1	1	2	2
18	42	2	2	0	2	19	25	2	2	-	-	25	19	3	3	-	0
26	35	0	0	1	-	27	41	1	1	-	-	31	25	4	3	0	0
35	21	1	0	0	0	36	21	2	1	1	0	39	25	1	1	-	0
40	37	2	3	-	0	45	45	1	1	0	0	47	22	1	0	2	-
56	45	4	4	0	0	58	35	1	2	2	1	60	19	1	1	0	-
61	48	0	1	2	2	64	21	3	4	0	1	73	25	2	2	1	-
77	26	3	3	1	1	79	47	1	1	4	4	83	30	1	0	0	0
87	24	1	2	2	2	94	31	1	1	-	-	102	35	2	1	2	2
107	40	0	0	1	2	109	31	-	0	3	-	110	36	1	1	1	1
112	40	1	1	2	3	113	46	-	1	1	1	122	52	0	0	-	1
140	17	-	0	0	0	142	31	0	0	-	-	154	25	2	-	1	1

III. PHYSICAL APPEARANCE

THIS chapter represents an attempt to breathe life into the Khok Phanom Di people by describing their physical appearance as far as it can be ascertained from their dry bones. Craniofacial and dental characteristics, body size and proportions are discussed. Definitions of measurements, individual data, summary statistics and indices are given in tables 29–47.

CRANIOFACIAL APPEARANCE

Craniofacial characteristics are described from subjective visual assessment and using craniometric data based on 'traditional' measurements.

Cranial vaults

Examples of adult skulls are shown in figures 24–30. A typical child skull is included in figure 30 and shown in figure 31. Figure 32 shows the best-preserved infant skull. On the basis of the cranial index (length/breadth) the mean shape in both sexes (index 80.0) would be classified at the boundary of medium - round (brachycranic) skulls (Bass 1971, Brothwell 1981). When the third dimension, height, is taken into account, the tendency to roundness in the sample is confirmed. Using the mean length-breadth/basion height index (Stewart 1965:363, as cited in Bass 1971:65) the vaults are close to the high-very high range at 88.5 in both sexes. Data are provided and summarised in tables 29–31.

Compared with the world-wide range of male *Homo sapiens* samples reported by Howells (1989) and with the range of Asian samples listed by Hanihara (1993), the skulls are of average length, but broad and high in absolute terms. The means suggest the male crania are wider and taller than most samples of prehistoric and modern South East Asian populations (Olivier *et al.* 1966, Pietrusewsky 1981, 1997) (Table 17). They are very much wider but of similar length to the skulls from the North Eastern Thai site of Ban Chiang and are shorter and wider than the small Neolithic samples from Laos and Vietnam. The means suggest that all the prehistoric skulls are longer than the modern South East Asian skulls. One individual from Khok Phanom Di, B45, has a very short skull (150 mm; Fig. 21). Breadth is average but the short length results in a cranial index of 88.0, making the skull hyperbrachycranic. The possibility that the skull had been artificially deformed has been considered, but as the occipital bone is flattened while the frontal bone is normal, deliberate moulding of the skull is not likely. This may be an extreme expression of the skull shape in this population.

Faces

As with the cranial vaults, these are large, with very pronounced cheek bones (maxillary and zygomatic (malar) bones). The width of the face is indicated by the bimaxillary and bizygomatic measurements, which are at the upper extreme of breadths in *Homo sapiens* in general (Howells

TABLE 17: *Cranial measurements; comparative male South East Asian examples from prehistoric and modern populations*

Population	n	Max. cranial length		Max. cranial breadth		Max. cranial height	
		mean	s.d.	mean	s.d.	mean	s.d.
Khok Phanom Di	7	180.0	7.6	145.6	4.7	144.8*	3.9
Ban Chiang ³	12	180.2	5.7	139.1	6.9	-	-
Neolithic Laos ²	5	182.4	3.5	138.6	4.1	-	-
Neolithic Vietnam ²	7	188.6	6.2	133.4	6.0	-	-
Modern							
Thai ¹	33	168.6	7.5	141.4	5.1	135.9	4.9
Khmer ¹	32	173.7	8.4	140.4	5.9	136.8	6.0
Cambodia ²	11	173.0	6.2	142.8	5.0	-	-
Laotian ¹	16	167.9	7.8	144.5	4.8	132.7	3.5
Laos ²	29	170.4	6.9	140.9	5.5	-	-
Vietnamese ¹	67	174.5	6.8	139.1	5.5	136.8	5.1
Vietnam ²	30	178.0	6.1	139.0	6.4	-	-
Burma ²	16	176.4	6.6	142.0	5.7	-	-

¹ Olivier *et al.* 1966 NB s.d. = $\pm$, ² Pietrusewsky 1981, ³ Pietrusewsky 1997. * n=5

1989) and for modern South East Asian populations (Olivier *et al.* 1966; Table 18). This facial width is not confined to the lateral structures of the face but is also evident in the width of the nose. The upper face is long but the nose is of average height when compared with Hanihara's Asian samples (1993). In the third dimension, the upper face is comparatively flat in both sexes, with a flat nasal bridge. The dentition is protrusive, with maxillary alveolar prognathism indicated by basion-prosthion distances in the upper ranges of lengths in Howells (1989) samples and longer than the modern South East Asians (Olivier *et al.* 1966). The males tend to have slightly less protruding midfaces than the females.

Mandibles

The mandibles are also large (Figs. 33–34, tables 33–34). They are particularly broad where they articulate with the base of the skull at the temporomandibular joint (indicated by bicondylar width) compared with a range of modern South East Asian populations (Table 19). This is as would be expected given the broad cranium and midface, although at the angle of the jaw (bigonial width) the mandibles are relatively less broad. The body of the mandible is long, and although not high at the chin (symphyseal height), it is midway along the body (m_1/m_2 height), indicating that the jaw was substantial. The gonial angle is steep.



FIG. 21. The skull of burial 45, a female aged 45 years. This individual has a very short skull (150 mm). Breadth is average but the short length results in a cranial index of 88.0, making the skull hyperbrachycranic. The possibility that the skull had been artificially deformed has been considered, but as the occipital bone is flattened but not the frontal bone, deliberate moulding of the skull is not likely

TABLE 18: *Facial measurements; comparative male South East Asian examples from prehistoric and modern populations*

Population	n	Bizygomatic breadth		Bimaxillary breadth		Upper facial height		Basion-prosthion length	
		mean	s.d.	mean	s.d.	mean	s.d.	mean	s.d.
Khok Phanom Di	5	146.2	4.3	109.3	5.9	73.4	1.2	101.3	1.0
Ban Chiang ²	12	-	-	104.8	4.3	71.5	3.7	-	-
Modern ¹									
Thai	33	129.7	5.1	98.9	5.5	67.2	4.5	93.7	5.2
Khmer	32	132.5	4.9	100.8	5.0	67.6	3.2	97.4	5.0
Laotian	16	131.1	4.5	97.7	3.1	67.8	4.4	89.2	4.7
Vietnamese	64	133.5	4.7	100.3	5.2	67.4	4.2	93.5	5.7

¹ Olivier *et al.* 1966 (NB s.d. = $\pm$), ² Pietrusewsky 1997

TABLE 19: *Mandibular measurements; comparative male South East Asian examples from modern populations*

Population	n	Length		Bicondylar Breadth		Bigonial Breadth		Gonial Angle		Symphyseal Height		Body Height	
		mean	s.d.	mean	s.d.	mean	s.d.	mean	s.d.	mean	s.d.	mean	s.d.
Khok Phanom Di	22	108.2	4.0	130.4	6.9	101.9	6.8	114.7°	5.2	34.1	2.6	32.0	2.4
Modern ¹													
Thai	33	103.4	5.1	117.6	6.5	97.4	7.0	117.1°	6.4	36.2	3.5	29.3	3.7
Khmer	15	103.9	6.2	119.5	7.3	97.1	6.0	114.9°	7.5	34.0	3.8	28.3	3.7
Laotian	17	103.3	3.5	118.9	4.4	97.6	3.8	121.5°	4.9	36.3	3.1	28.4	2.9
Vietnamese	64	102.0	4.9	120.2	6.0	97.7	5.7	117.7°	7.0	34.1	3.4	28.2	2.8

¹ Olivier *et al.* 1966 NB s.d. = $\pm$

DENTITION

As has already been mentioned, the maxillary dentition in these people tended to protrude because of alveolar prognathism. Together with the relatively short mandibles, this produced an overbite, with the upper teeth protruding over the lower teeth.

Tooth size

Individual dental measurements, definitions, and summary statistics are listed in tables 36–39. The overall size of the dentition, using a ‘summary tooth-size’ calculated according to Brace and Hinton (1981:551) is 1254.3. This is almost the same as the ‘Thai Neolithic’ (1252 - unknown source) figure given in Brace and Hinton (1981), and larger than the ‘Thai bronze age’ (1224 - assumed to be Non Nok Tha) and ‘Thai’ (1233 - assumed to be recent Thai) summary tooth sizes in the same publication. The size of the dentition is in the middle of the range of the large number of Asian and Oceanic populations listed by Brace and Hinton (range 1157–1371).

The summary tooth size obscures the relative sizes of maxillary and mandibular, anterior and posterior teeth. Compared with the north eastern Thai site of Non Nok Tha and with modern Thai (Brace 1978), the mean tooth areas of individual teeth of the Khok Phanom Di people (both sexes combined; Table 20) are all larger, with the exception of the maxillary premolars and the third molars (Tables 36–39).

TABLE 20: *Comparative tooth sizes (area mm²), both sexes combined*

	Khok Phanom Di			Non Nok Tha ¹			Modern Thai ¹		
	n	mean	s.d.	n	mean	s.d.	n	Mean	s.d.
Maxilla									
I1	29	65.7	7.4	34	61.8	5.9	19	61.7	6.1
I2	27	48.1	5.4	31	44.9	7.6	37	46.6	5.6
C	51	64.7	6.9	38	63.3	8.0	31	64.8	7.5
P1	68	68.4	7.7	44	68.1	7.7	34	70.1	7.9
P2	64	62.7	7.7	48	63.4	7.2	34	63.4	6.1
M1	68	125.9	11.5	52	120.8	10.5	30	119.0	10.6
M2	80	112.2	13.7	44	106.1	12.0	31	109.8	12.3
M3	53	100.8	14.9	32	99.2	21.0	24	100.8	12.0
Mandible									
I1	39	34.4	3.6	23	32.0	3.3	29	31.8	3.3
I2	50	39.2	4.8	33	38.4	4.8	32	38.7	4.3
C	61	55.7	6.0	41	53.7	6.8	41	53.0	5.9
P1	84	59.4	6.1	42	57.4	7.8	44	57.8	7.2
P2	90	62.2	6.8	45	59.7	7.0	41	60.6	6.9
M1	76	127.5	10.9	49	126.1	11.0	36	123.6	10.9
M2	88	113.6	10.9	48	112.6	10.9	38	113.0	11.5
M3	60	113.8	20.6	43	116.4	12.1	12	114.2	13.5

¹ Brace 1978

Tooth ablation/evulsion

In the initial recording of the dentition, it became clear that there was a symmetrical pattern of antemortem loss of incisors in adults. The symmetry of the missing teeth and the repetition of the patterns among individuals led to the conclusion that the teeth must have been deliberately removed. The ritual ablation or evulsion of anterior teeth has been recorded in many societies, both prehistoric and modern, although the reasons for the practice are not always clear and could range from a form of recognition of attainment of adulthood to membership of a social group or even simply as personal beautification. The patterns of ablated teeth at Khok Phanom Di varied. Both maxillary and mandibular teeth were involved, although the practice was almost entirely restricted to the incisors. The detail of the patterns, which varied over time and with age and sex, is given in Tayles (1996) and illustrated and discussed in chapter VIII.

Industrial or dietary anterior tooth wear

Some individuals in MP2-3 had wear on anterior teeth which involved deep erosion of the dentine to a degree markedly greater than the wear on their posterior teeth (Fig. 22A). Others had notches or grooves on the occlusal surfaces (Fig. 22B). This affected 12/16 females and 5/13 males buried during this period. The frequency difference between the sexes was statistically significant (χ^2 3.95, $p < 0.05$). In some of these individuals, the wear could be explained by the pattern of occlusion, as the opposing teeth were either missing or offset. Where the opposing teeth were lost post-mortem, an assessment of this cause of the unusual wear could not be made. The two wear characteristics often occurred together and may have resulted in the formation of caries in the anterior teeth. The wear was unilateral in all cases, suggesting that it had not contributed to, or did not reflect, the practice of tooth ablation. It is possible that the deeply-excavated dentine and some of the notches reflect an unusual pattern of tooth use, either industrial or dietary, and that this may have been a secondary cause of anterior tooth loss. The use of teeth as tools is well documented, both archaeologically (eg Merbs 1968) and ethnographically, but in case of Khok Phanom Di it is difficult to make such an assumption on the basis of the gross evidence. Microwear analysis may clarify the basis of the notch formation.

POST CRANIAL SKELETON

A selection of adult long bones, illustrating the range of variation in size and shape, is illustrated in figure 35. Data on long bone dimensions are listed and summarised in tables 40–47. Long bones from a typical child skeleton are illustrated in figure 36 and a complete infant skeleton in figure 37. Stature and limb proportions are calculated using long bone lengths (Table 40). Beyond these measurements, body size and shape is difficult to define. Most recent attempts to describe body mass (weight) have been stimulated by the race to identify evolutionary relationships among species ancestral to *H. sapiens*. The relationship of bone size to body weight/mass is discussed by various authors in the search for estimates of body mass as a contributor to understanding of hominid evolution (eg McHenry 1991, 1992, Hartwig-Scherer 1993). In addition to these easily defined (although not necessarily easily measured) concepts of stature and mass, 'robusticity' is another description

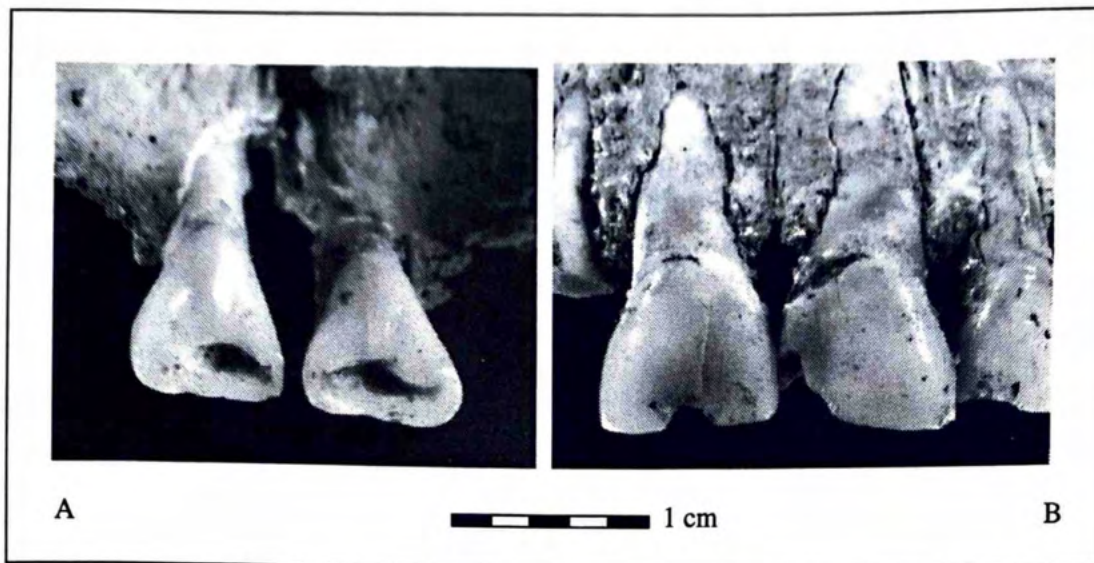


FIG. 22. A: Lingual view of maxillary central incisors with wear resulting in deeply excavated dentine. Burial 60, female, aged *c.* 19 years. The mandibular incisors were all present in this individual. B: Labial view of maxillary incisors with notches worn in the occlusal surfaces of the teeth. Burial 58, female, aged *c.* 35 years

of body size and shape often used in descriptions of skeletal population. However, it is not always clear what is meant by this term, whether it is referring to overall body shape, to muscular development, or to cortical thickness (St Hoyme and İşcan 1989). The standard presentation of 'robusticity' is as an index of long bone length to one or more shaft circumference measure but it is not clear what, if anything, the index indicates. Is a short, stocky person therefore more 'robust' than an average size stocky person? Are they heavier or more muscular? Is this the same thing? Is it a reflection of health, lifestyle or is it genetic? (or all of these?). The relationship between indices calculated from bones and body composition and physical appearance is not a simple one. As this is not a treatise on morphometrics or biomechanics, this problem of establishing the accuracy of descriptions of body habitus from the hard tissues is not going to be considered at length. Postcranial measurements presented are long bone lengths, from which the relatively straightforward stature and limb proportions are calculated, together with joint diameters, midshaft diameters and circumference of long bones. One estimate of body weight (mass) has been made, using femoral shaft diameters (Table 43).

Stature

The most straightforward relationship between the human skeleton and the physical appearance of an individual is that of the length of the long bones, particularly of the lower limb, and height. The skull, vertebral column and pelvis clearly also contribute but the relationship between the long bones of the legs and stature has long been recognised and provides the most practical

method of stature estimation. Numerous regression equations calculated from cadavers have been published but the most widely recognised calculation equations are those provided by Trotter and Gleser (1952, 1958, Trotter 1970), using data available from the dead from World War II and the Korean War, and the Terry Collection of autopsy specimens. These equations were developed from large groups of White and Black American males, smaller groups of females and a group of 'Mongoloid' males which included Japanese, Filipinos, Hawaiians and American Indians. Equations potentially relevant to South East Asian populations are also available for northern Chinese males (Stevenson 1929), New Zealand Polynesians (Houghton *et al.* 1975), a small modern autopsy sample from Bangkok which includes an unspecified proportion of Chinese individuals in addition to Thai (Sangvichien *et al.* 1985, n.d.), and an autopsy sample from Java which includes some Chinese and other peoples (Bergmann and The 1955).

To establish the most appropriate equations for use on the Khok Phanom Di individuals, statures were calculated using the individual equations for each of the long limb bones from the populations listed in table 21. The differences between the estimates for each individual for the lower limb (femur and tibia), the upper limb (humerus and radius) and among all four long bones were compared and the equations with the least differences were adopted.

TABLE 21: *Samples which form the basis of the stature equations used in the comparative analysis*

Ethnic group	Source	Sample size	
		Males	Females
White American	Trotter (1970)	3782	63
Black American	Trotter (1970)	825	177
Mongoloid	Trotter (1970)	68	-
North Chinese	Stevenson (1929)	48	-
Polynesian	Houghton <i>et al.</i> (1975)	450	54
Thai/Chinese	Sangvichien <i>et al.</i> (1985, n.d.)	50	27
Javanese	Bergmann and The (1955)	40	25

Long bone measurements were taken using the method described and used by Trotter (1970). This method was also used by Houghton *et al.* (1975), and Stevenson appears to have followed the same procedure. Sangvichien *et al.* (1985) used the medial condyle of the tibia rather than the lateral condyle, and Bergmann and The (1955) used physiological rather than maximum length on the femur and radius. The mean stature estimates and the mean individual differences are listed in table 22.

This shows that, for the lower limb, the Thai and Javanese equations give similar results, with the Javanese equations slightly better for the males. When the exercise is extended to include the upper limb, the Thai equations give less variable results than the Javanese, although for males the result from Polynesian equations is surprisingly less variable. Taking all four limb bones together,

TABLE 22: *Comparison of stature estimates using various equations (Sources of equations as for table 21)*

	Mean Femur	Mean Tibia	Difference	Mean Humerus	Mean Radius	Difference	Mean All bones	Difference
Females	n=25			n=31			n=20	
Wh. American	154.93	161.92	6.99	154.05	162.87	1.56	158.80	9.86
Bl. American	152.84	157.47	4.63	152.74	157.13	5.50	155.33	6.06
Polynesian	159.69	163.81	4.12	159.14	161.37	2.29	161.22	5.20
Javanese	158.76	158.81	1.88	156.52	160.60	4.14	159.00	5.08
Thai	154.63	154.47	1.87	152.73	153.40	1.48	154.07	3.68
Males	n=25			n=26			n=21	
Wh. American	163.18	169.68	6.50	164.91	173.73	8.82	197.97	10.68
Bl. American	160.58	165.16	4.58	162.08	167.26	5.19	163.83	7.03
Polynesian	168.74	172.42	3.68	169.56	172.57	3.04	170.92	4.79
Mongoloid	164.51	167.81	3.37	165.38	170.71	5.33	167.19	6.59
Nth Chinese	165.95	168.58	3.04	167.78	173.70	5.92	169.25	9.14
Javanese	163.74	164.76	1.61	164.50	170.67	6.17	165.99	7.56
Thai	162.06	162.57	1.85	162.64	165.83	3.42	163.35	4.86

the Thai equations clearly give the least variable results for females, but for the males the Polynesian and Thai variations are very similar. This result indicates that the proportions of the segments of the lower limb are similar in the Thai and Javanese, and that the proportions of the segments of the upper limb are similar in the Thai and Polynesian. The taller statures calculated using the Polynesian equations indicate that the ratio of the limb to axial length is different in the Thai and Polynesian groups.

Considered overall, the Thai equations represent the best for use on the Khok Phanom Di material, as they give the least variation for females and are least variable for males when each limb is considered separately, but both are taken into account. The Thai equations are listed in table 23.

Following Trotter's (1970) recommendations, the equation with the least standard error is used where possible, rather than averaging results for all equations. The mean of left and right bones is used. For the males, the equation with the least standard error is that for the femur and tibia combined. For consistency this combination was also used for the females, although the standard error for the femur alone is slightly less. Where measurements for the two bones were not available, the femur or tibia alone was used, or the humerus and radius combined where there were neither of the lower limb bones.

Stature estimates were made for all individuals with fused epiphyses, which includes all adults except two males aged 15 and 16 years (burials 8, 117; Table 40). Statures for all except three fe-

TABLE 23: *Equations used in calculating stature. Equations 1- 3 from Sangvichien et al., 1985; 4-6 from Sangvichien et al., n.d.*

Females		
1	Femur	$(2.5815 \text{ Fe Max.}) + 49.2412 \pm 3.0007$
2	Tibia	$(2.9716 \text{ Ti Max}) + 51.6015 \pm 3.4687$
3	Femur/Tibia	$(1.4074 (\text{Fe Max} + \text{Ti Max})) + 48.4454 \pm 3.1019$
4	Humerus	$(2.7501 \text{ Hu Max}) + 74.0862$
5	Radius	$(2.6743 \text{ Ra Max}) + 92.5011$
6	Humerus/Radius	$(1.6192 (\text{Hu Max} + \text{Ra Max})) + 70.4181$
Males		
1	Femur	$(1.7289 \text{ Fe Max}) + 88.1320 \pm 5.3885$
2	Tibia	$(2.7638 \text{ Ti Max}) + 62.6946 \pm 4.5232$
3	Femur/Tibia	$(1.4210 (\text{Fe Max} + \text{Ti Max})) + 49.6080 \pm 4.2087$
4	Humerus	$(3.0197 \text{ Hu Max}) + 70.0304$
5	Radius	$(3.3658 \text{ Ra Max}) + 81.4874$
6	Humerus/Radius	$(1.8816 (\text{Hu Max} + \text{Ra Max})) + 59.2273$

males and one male were calculated from lower limb bones, with the majority ($49/66 = 74$ per cent) based on the most suitable combination of bones, the femur and tibia.

Female statures range from 141.1 cm to 163.2 cm, with a mean of 154.3 cm (s.d. 4.5 cm), although the individual with a stature of 141.1 cm (B56) is 6.5 cm shorter than the next shortest woman. If this individual is excluded from the calculations, the mean stature is 154.7 cm (s.d. 3.9 cm). Males range in stature from 153.8 cm to 171.9 cm, with a mean stature of 162.3 cm (s.d. 5.2 cm). The mean statures are within the 'average' range for human populations generally (Wells 1970, Harrison *et al.* 1988, Molnar 1992) although at the extremes about one-third of the Khok Phanom Di males could be classified as 'short' compared with only 10 per cent of the females. None would be classified as 'tall'.

These data can be compared with prehistoric and modern South East Asian populations. The prehistoric comparative data represent mean statures for all available groups (Table 24). To remove variation attributable to the use of different equations in various reports, the estimates have been recalculated from the long bone measurements using the modern Thai equations of Sangvichien *et al.* (1985, n.d.)

The data are also shown graphically in figure 23. The females from Khok Phanom Di are similar in stature to the samples from the North East Thai site of Non Nok Tha and slightly shorter than those from Ban Na Di, although the differences are not statistically significant. The mean stature of the females from Ban Kao is significantly different but the small size of the sample and the effect of two tall outliers negates any conclusions being drawn from this statistic.

TABLE 24: *Summary of the mean estimated statures for prehistoric Thai populations. All estimates have been recalculated using the equations from Sangvichien et al. 1985, n.d.. Measurements in cm*

Females	n	Mean	s.d.	Range	Significance cf Khok Phanom Di
Khok Phanom Di*	36	154.3	4.5	141.1-163.2	
Non Nok Tha	19	154.4	3.9	148.1-161.3	ns
Ban Kao	7	159.0	6.2	148.5-166.7	p=<.05
Ban Na Di	11	156.6	5.1	149.9-164.8	ns
Non Pa Kluay	4	156.1	4.7	151.1-160.4	ns
Males					
Khok Phanom Di	30	162.3	5.2	153.8-171.9	
Non Nok Tha	10	166.2	3.5	161.1-172.4	p=<.05
Ban Kao	10	167.7	5.8	157.2-175.4	p=<.01
Ban Na Di	15	167.9	5.7	158.6-176.1	p=<.01
Non Pa Kluay	3	164.2	4.4	160.5-169.1	ns

*excluding burial 56, mean 154.68, s.d. 3.93, min 147.60

Sources of the long bone measurements on which these estimates are based:- Khok Phanom Di - this study; Non Nok Tha - Pietrusewsky 1974a (The material from the 1968 season is not included in this sample); Ban Kao - Sangvichien *et al.* 1969; Ban Na Di - Houghton and Wiriyaromp 1984; Non Pa Kluay - Pietrusewsky 1988b

The males from Khok Phanom Di are shorter than all other prehistoric Thai, with the differences statistically significant for three of the five sites. The tallest individuals, as with the females, are from Ban Na Di and Ban Kao.

Comparative data from modern South East Asians has been collected from a variety of sources, and are listed in table 25. The mean stature of the females from Khok Phanom Di is taller than the mean stature of any of the samples of modern South East Asians, including the autopsy sample on which the stature equations were based. The mean stature of the Khok Phanom Di males equates with the median of the modern South East Asian male statures.

Body proportions and symmetry

The consideration of proportions is limited to the relationships of the upper and lower limbs to one another and the relationships of proximal to distal components of each limb. Symmetry between left and right is also considered. Individual long bone lengths are listed in table 40 and summarised in table 41. The indices show that there is considerable variation in each index (Table 42). The mean brachial index (ratio of forearm (radius) to upper arm) at 80 is in the upper range compared with mean values for world-wide populations (range from 71–81, Martin and Saller 1957: 940, Krogman 1962:202-203, St Hoyme and İşcan 1989). The mean crural index (ratio of tibia to femur) at 85 is also in the upper range for world-wide populations (77-86, Martin and Saller 1957:970). Compar-

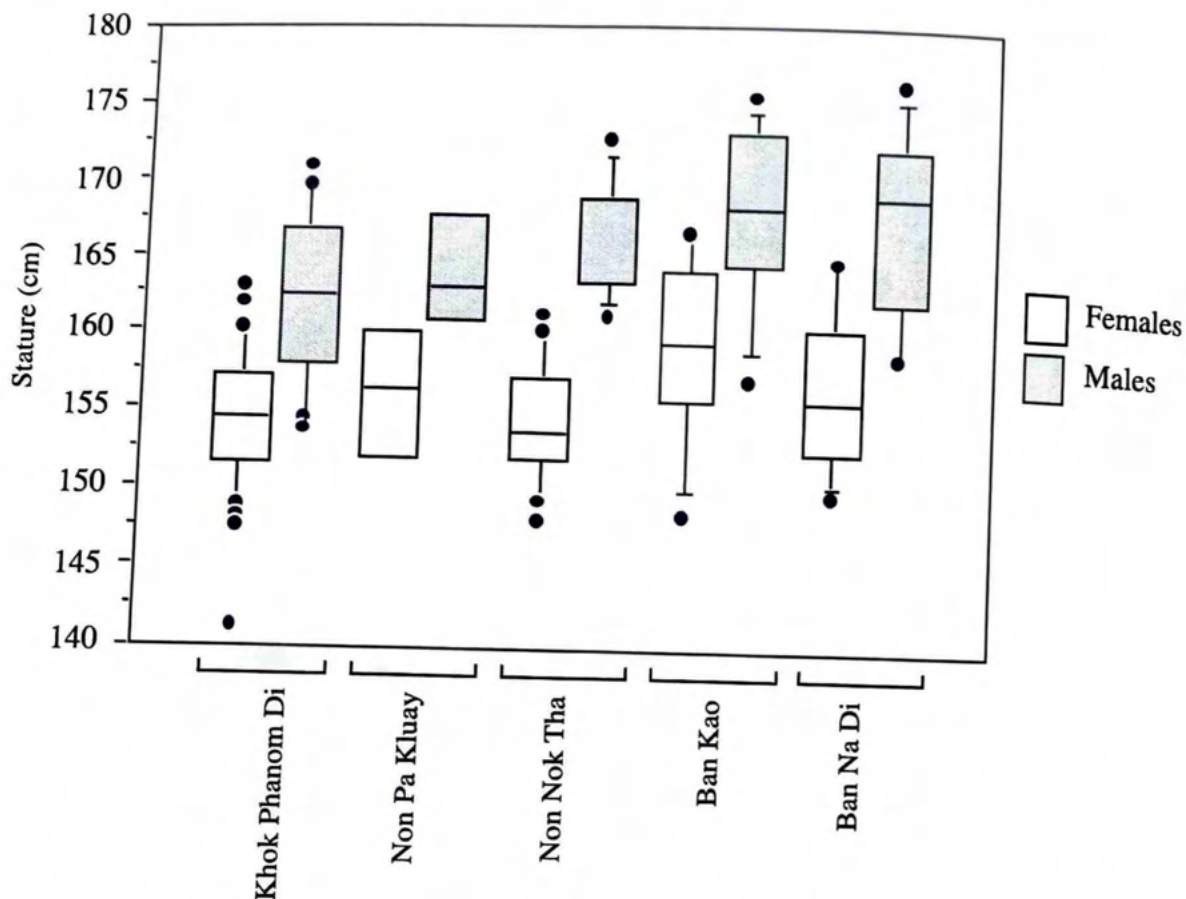


FIG. 23. Comparison of mean statures of adults at Khok Phanom Di with those from other prehistoric Thai sites. All were calculated using the equations of Sangvichien *et al.* (1985, n.d.). The sites are those listed in table 24

ing the mean lengths of the humerus, both sexes combined (296 mm) and the radius (238 mm) with the range of lengths for these long bones given by Martin and Saller (humerus 260–380 mm; radius 190–280) (1957: 1041,1051) shows that the high indices reflect relatively short humeri rather than long radii. A comparison of the mean femur and tibia lengths (femur 419 mm; tibia 353 mm) with the world-wide ranges given by Martin and Saller (femur 340–536; tibia 280–445, 1957:1079,1102) again suggests that the proximal segment of the limb is relatively short. These relatively high indices suggest that the population is adapted to a high temperature, in accord with Allen's Rule (Baker 1988) which indicates a positive correlation between the relative size of the most exposed portions of the body (i.e. the distal segments) and mean environmental temperature.

Most of the dimensions of the long bones are not asymmetric, although there are exceptions. In the upper limb the right humerus is significantly larger than the left in both sexes, although the dimensions involved in the asymmetry differ. In females the length, head diameter and shaft minimum diameter are significantly larger on the right. In males the shaft maximum diameter (and therefore

TABLE 25: *Mean statures of modern South East Asians*

Country	Sample source	Ethnicity	Date collected	n	Mean age (years)	Mean stature	Data Source
Females							
Cambodia	-	-	-	-	-	145.1	1
Cambodia	-	-	-	15	-	149.1	2
Thailand	Autopsy	Thai/					
	Bangkok	Chinese	1980-81	27	-	151.7	3
Thailand	Rural	-	1962	32	25-29	151.0	4
Burma	Students	-	1968	-	19-24	153.2	5
Thailand	Bangkok	-	1969-76	10914	15-65	154.0	6
Males							
Thailand	-	'Siamese'	-	-	-	159.9	7
Thailand	North	West Lao	1935	815	32.6	160.6	8
Thailand	Rural	-	1962	57	25-29	161.0	4
Thailand	South East						
	Rural	Thai	1935	186	-	162.1	8
Thailand	Central*	Mon	1935	274	-	162.9	8
Laos	-	-	1973	136	-	163.2	5
Thailand	Military	-	1964	2950	24.0	163.4	5
Thailand	Central*						
	Rural	Thai	1935	950	32.6	163.4	8
Thailand	Autopsy	Thai,					
	Bangkok	Chinese	1980-81	50	-	164.1	3
Thailand	Bangkok	-	1969-76	9606	15-65	165.0	6
Cambodia	-	-	1973	-	-	165.0	5
Thailand	Military	-	1948	137	22	171.0	9

* 'Central' covers the lowlands of the Chao Phraya Valley and includes the region of the Bang Pakong Valley.

1. Mondiere and Bonifacy, cited in Martin and Saller 1957:779; 2. Schreider 1975; 3. Andrews 1943; 4. Interdepartmental Committee for Nutrition and National Defense, Nutrition Survey, cited in Frisch and Revelle 1969; 5. Eveleth and Tanner 1976: Appendix tables 61-2: 366-71; 6. Bunnag and Piampiti 1979; 7. Annandale, in Martin and Saller 1957:779; 8. Sangvichien *et al.* 1985; 9. Suvarnakich 1950

circumference) is significantly larger on the right but the other dimensions are symmetrical. Also in males, the right radius and ulna are significantly longer than the left. In the lower limb the oblique length of the left femur is significantly longer than the right and the midshaft medio-lateral diameter is wider. The left tibia is significantly wider in the antero-posterior diameter but not in the medio-lateral. In females the oblique length of the left femur is also significantly longer but the right femur is significantly wider in the midshaft antero-posterior dimension. Other dimensions in the femur

and tibia do not differ. This pattern of larger bones in the dominant upper limb (the right) and the opposite lower limb are not unusual in human populations. The sex differences are discussed in the section on sexual dimorphism below.

Body weight

Of the methods available for assessing body weight (mass) from long bone measurements, that of Hartwig-Scherer (1993) using femoral circumference was selected as having a low prediction error and representing the most straightforward to apply. As body weight estimate is not a primary focus of the analysis of the Khok Phanom Di people, all available weight assessment methods have not been evaluated and the data are presented as one of a range of possibilities. The equation used is: $1.839 \cdot \text{LOG}_{10}(\text{circumference of femur}) + 1.172$ ($r=0.85$; $\text{PE \%} = 7$) (Hartwig-Scherer 1993:20). The mean of left and right femora was used where available.

The individual estimates are listed in table 43 and summarised in table 44. Female weights range from 39.7–59.5 kg, with a mean of 46.6 kg (s.d. 5.11 kg). Males range from 48.1–63.2 kg, with a mean of 54.7 kg (s.d. 4.49 kg). Both means are about the middle of the range for modern South East Asian populations (Table 26).

The ratio of weight to stature is 3.16 for both sexes combined (3.34 for females; 2.99 for males). In modern South East Asians (Table 27) the ratio in three female samples ranges from 3.21–3.30, suggesting that the Khok Phanom Di females were stockier than their modern counterparts. Modern male samples vary from 2.90–3.05. The Khok Phanom Di males were within this size range.

Sexual dimorphism

In the skull, the males are on average larger than the females in all dimensions. All differences are statistically significant, with the exceptions of the breadth of the nose and two measurements which reflect midfacial prognathism; basion-prosthion length and mandibular ramus breadth.

Differences in postcranial body size, shape and proportions between males and females can be described using all the measures discussed in the previous section. All major limb bone means are significantly larger in all dimensions in males than in females.

Dimorphism in mean stature is 7.9 cm, which is 4.9 per cent of the mean male stature. As expected, the difference is highly significant ($P < 0.001$). Assessments of the mean sexual dimorphism in stature in populations world-wide range around 9–13 cm (Eveleth 1975, Tanner 1978), or, expressed as a percentage, 4–11 per cent of female stature (Stini 1985), 6 per cent of male stature (c. 10 cm) (Wells 1970), or 5–7 per cent of male stature (Molnar 1992).

Table 28 summarises sexual dimorphism in stature for other prehistoric Thai sites and a selection of modern Thai samples. Sexual dimorphism in stature in the Khok Phanom Di people is lower than in most of the prehistoric Thai and the modern samples. At the two sites with similar dimorphism to Khok Phanom Di, Ban Kao and Non Pa Kluay, both sexes are taller than at Khok Phanom Di. The lower sexual dimorphism at Khok Phanom Di compared with the other three sites is a reflection of the relative shortness of the Khok Phanom Di males compared with the females, as discussed above. The degree of dimorphism in the Khok Phanom Di population is relatively low compared with modern Thai. Possible explanations for this are discussed in the following chapter.

TABLE 26: *Mean mass of modern South East Asians*

	Sample source	Ethnicity	Date collected	n	Age (years)	Mean mass	Source
Females							
Vietnam	-	'Tonkinese'	1944	33	-	42.7	1
Burma	Students	-	1968	-	19-24	47.7	2
Cambodia	-	-	1944	31	-	48.3	1
Thailand	Rural	Thai	1962	32	25-29	45.8	3
Thailand	-	'Siamese'	1944	51	-	47.6	1
Thailand	Bangkok	-	1956-57	-	21	46.6	4
Thailand	Chiang Mai	-	1956-57	-	17	45.0	4
Thailand	East Coast	-	1956-57	-	18	49.1	4
Thailand	West Coast	-	1956-57	-	18	46.3	4
Thailand	Bangkok	-	1969-76	10914	15-65	48.0	5
Males							
Vietnam	-	'Tonkinese'	-	100	-	57.2	6
Burma	Students	-	1968	221	28	55.8	2
Laos	-	-	1973	136	-	56.0	3
Cambodia	-	-	1973	-	-	56.8	2
Thailand	North	West Lao	1935	949	32.6	50.6	7
Thailand	-	'Siamese'	1944	51	-	53.1	1
Thailand	Rural	-	1962	57	25-29	53.2	3
Thailand	Rural central*	-	1935	950	32.6	53.5	7
Thailand	Military	-	1964	2950	24	56.0	3
Thailand	Military	-	1948	137	22	63.9	6
Thailand	Bangkok	-	1969-76	9606	15-65	55.0	5
Thailand	Rural Bangkok	-	1956-57	-	19	57.4	4
Thailand	Chiang Mai	-	1956-57	-	18	45.7	4
Thailand	East Coast	-	1956-57	-	18	49.1	4
Thailand	West Coast	-	1956-57	-	18	46.3	4

*Central covers the lowlands of the Chao Phraya Valley, including the valley of the Bang Pakong. 1. Von Eickstedt, In Martin and Saller, 1957: 779-802. 2. Interdepartmental Committee for Nutrition and National Defense, Nutrition Survey, In Frisch and Reville, 1969. 3. Eveleth and Tanner, 1976; appendix tables 61-2: 366-71. 4. Vachananda, Kittivijja and Snidvongs, 1959. 5. Bunnag and Piampiti, 1979. 6. Suvarnakich, 1950. 7. Andrews, 1943.

The patterns of asymmetry in both upper and lower limbs differ in males and females. In the upper limbs, the dimensions which are significantly larger in the right than the left in females reflect the underlying size of the bone (length, maximum head diameter, minimum shaft diameter) whereas in males the dimensions which are larger on the right than the left reflect the enlargements of the site

TABLE 27: *Mean mass:stature ratios of modern South East Asians*

	Mass	Stature	mass/stature ratio	Data source
Females				
Burma	47.7	153.2	3.21	Frisch and Revelle 1969
Thailand - rural	45.8	151.0	3.30	Eveleth and Tanner 1976
Thailand - Bangkok	48.0	154.0	3.21	Bunnag and Piampiti 1979
Males				
Laos	56.0	163.2	2.91	Eveleth and Tanner 1976
Cambodia	56.8	165.0	2.90	Eveleth and Tanner 1976
Thailand - north	50.6	160.6	3.17	Andrews 1943
Thailand - rural	53.2	161.0	3.03	Eveleth and Tanner 1976
Thailand - military	56.0	163.4	2.92	Eveleth and Tanner 1976
Thailand - rural Central	53.5	163.4	3.05	Andrews 1943
Thailand - Bangkok	55.0	165.0	3.00	Bunnag and Piampiti 1979

TABLE 28: *Sexual dimorphism in stature of prehistoric and modern South East Asians, expressed as a proportion of male stature*

	Females		Males		Difference	% of male stature
	n	cm	n	cm	cm	
Thai - prehistoric						
Khok Phanom Di	36	154.3	30	162.3	8.0	4.9
Non Pa Kluay	4	156.1	3	164.2	8.1	4.9
Ban Kao	7	159.0	10	167.7	8.7	5.2
Ban Na Di	11	156.6	15	167.9	11.3	6.7
Non Nok Tha	19	154.4	10	166.2	11.8	7.1
Thai - modern						
Bangkok (autopsy)	27	151.7	50	164.1	12.4	7.6
Bangkok	10914	154.0	9606	165.0	11.0	6.7
Rural	32	151.0	57	161.0	10.0	6.2

Sources of prehistoric data as for table 49. Sources of modern data: Bangkok (autopsy) - Sangvichien *et al.* 1985; Bangkok - Bunnag and Piampiti, 1979; Rural - Interdepartmental Committee for Nutrition and National Defense, Nutrition Survey, in Frisch and Revelle 1969

of deltoid muscle attachment (maximum shaft diameter incorporates the distal end of the deltoid crest). This may indicate a sexual division of labour although it also raises questions about the age

at which participation in community activities commenced. If females began helping their mothers at an early age, handedness would be reflected in the size of bones (together with other structures in the limb). However, the pattern in males of handedness being reflected in muscular development could indicate that their participation in adult activities commenced after the cessation of growth in body size.

The sex differences in the patterns of asymmetry in the lower limb also suggest sexual division of labour. The larger dimensions of the left femur in a medio-lateral direction and the tibia in an antero-posterior direction in males contrasts with the larger antero-posterior shaft dimensions on the right side in females. The larger dimensions reflect bending stresses in that direction; the differing patterns therefore indicate males and females were exposed to differing biomechanical stresses, presumably because of different activity patterns.

Proportions within the limbs do not significantly differ between the sexes for the lower limb, but in the upper limb, the brachial index is significantly higher in males than females, particularly in the right limb. This means that the males have relatively very short humeri. The intermembral index (upper limb/lower limb) is significantly higher in both left and right sides in the males than in the females, indicating relatively shorter lower limbs in men. Intrasex asymmetry in limb proportions is greater in males than in females with the brachial and intermembral indices differing significantly between left and right in males. These differences may again be related to the timing of introduction of individuals to the workforce and to sexual division of labour. Alternatively, sex differences in resilience to malnutrition or poor health may be the explanation. This possibility is discussed in the following chapter.

SUMMARY

Overall, the skulls of the people of Khok Phanom Di are both broad and high, meaning that their heads are large compared with other prehistoric South East Asians and with modern Thai. Their faces are correspondingly broad and long, with the upper face flat but the dentition protrusive and the chin substantial. Their teeth are large compared with more recent South East Asians.

There are sex differences in overall body size compared with other prehistoric and modern South East Asians. The males are of average height and weight, but the females relatively large. Sexual dimorphism in stature is correspondingly low. There is the possibility that sexual dimorphism in bone dimensions reflects different patterns of physical activity and possibly different expectations of young adults by the community. Variation over time in morphometrics is discussed in chapter VIII.



FIG. 24. Skull of burial 77, a female aged 26 years

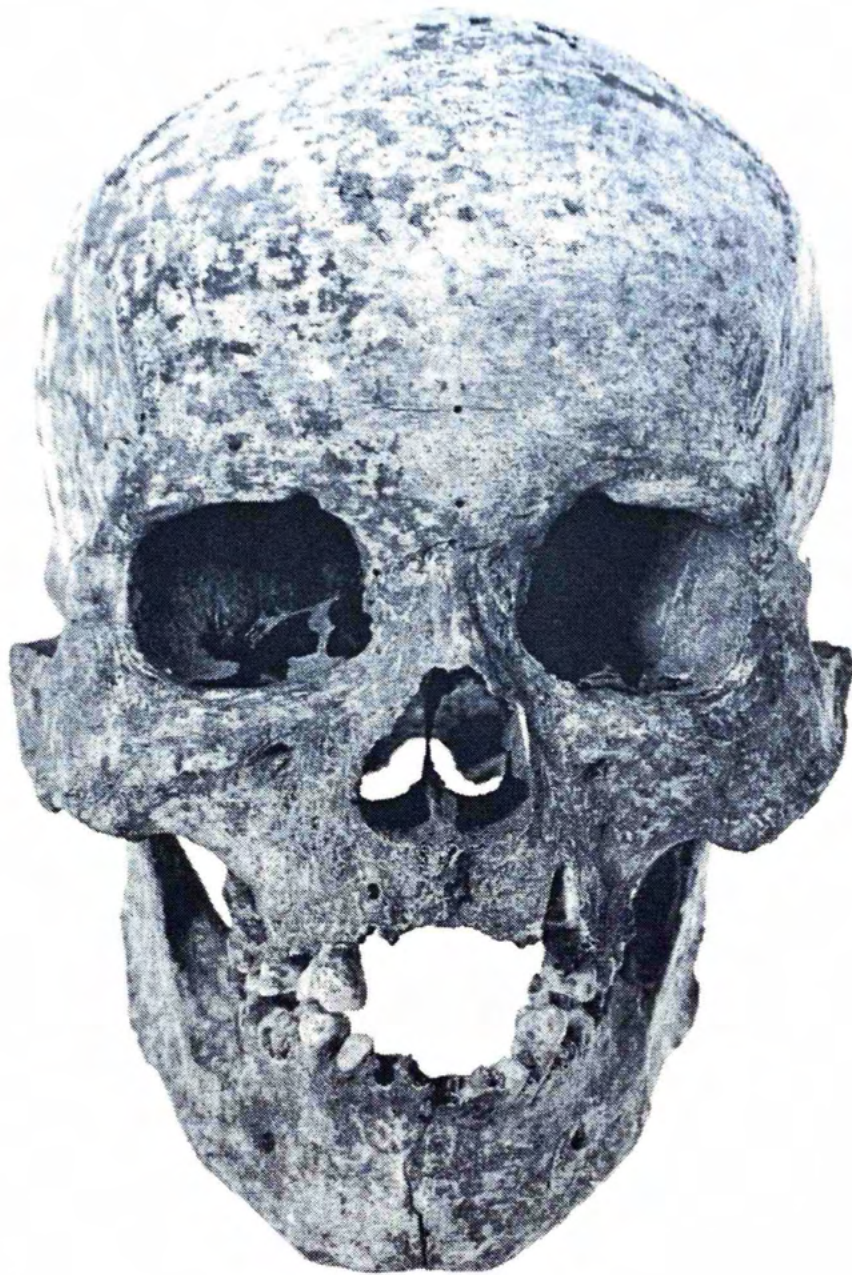


FIG. 25. Skull of burial 38, a male aged 33 years

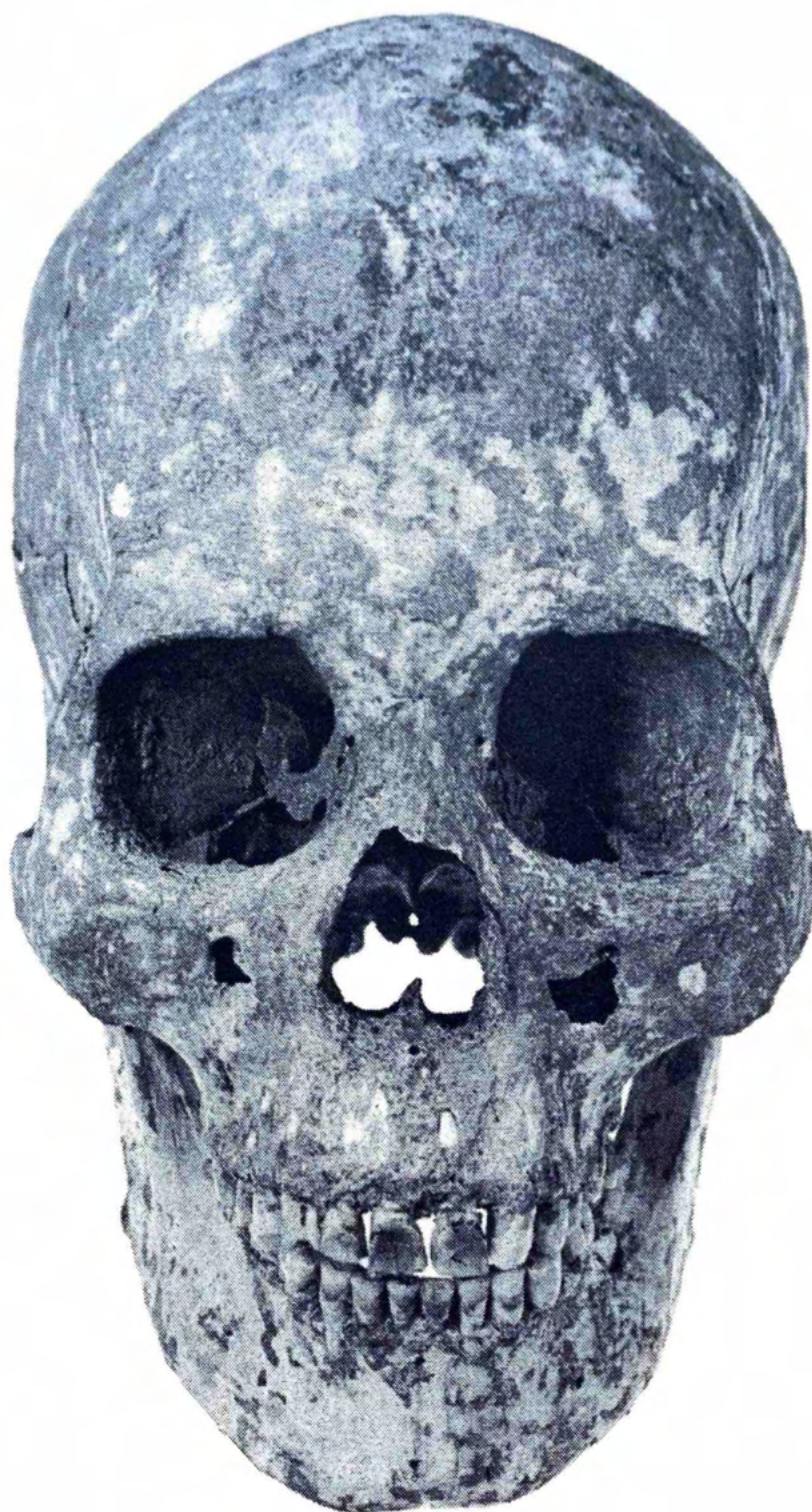


FIG. 26. Skull of burial 73, a female aged 25 years



FIG. 27. Skull of burial 72, a male aged 32 years

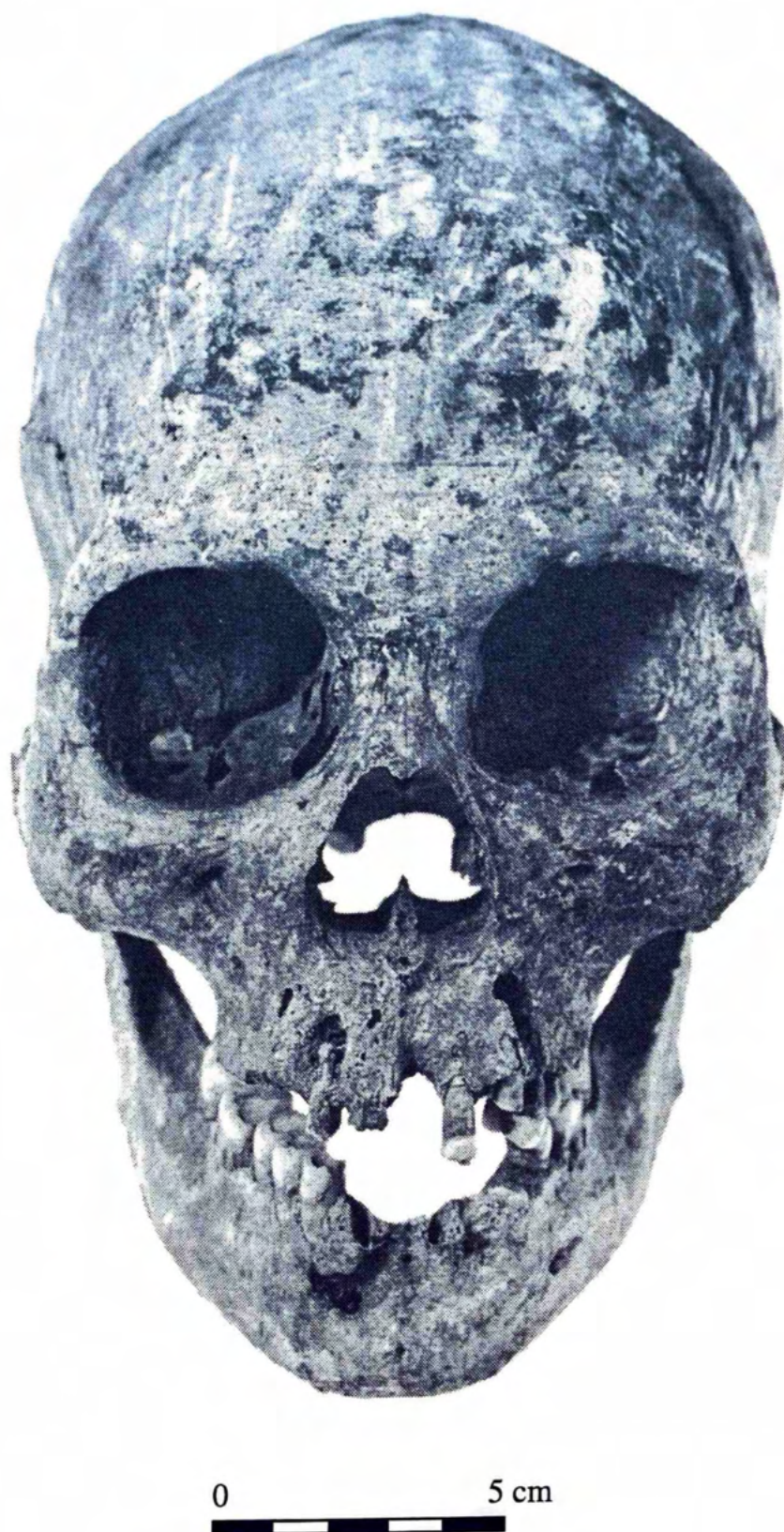
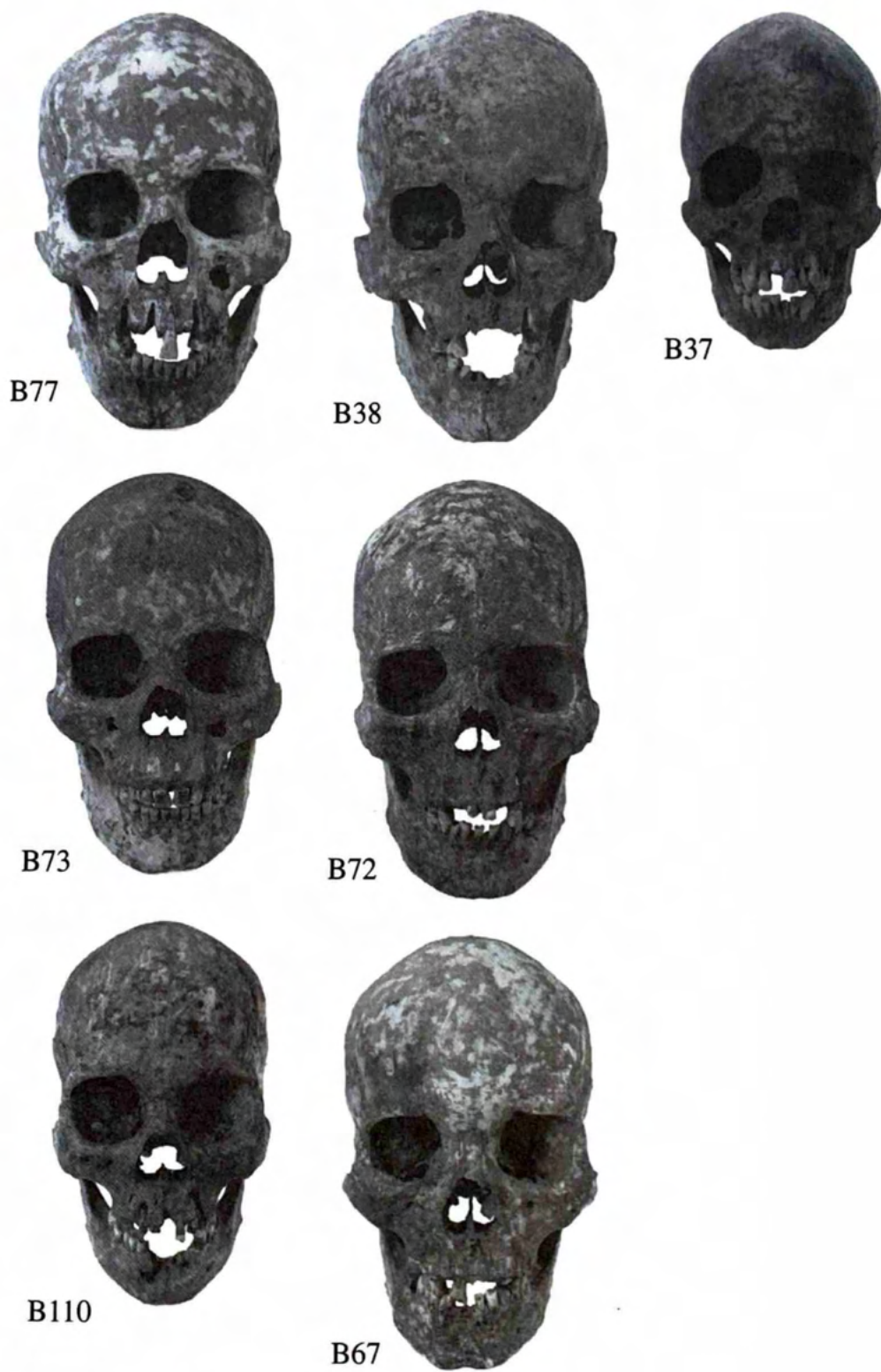


FIG. 28. Skull of burial 110, a female aged 36 years



0 5 cm

FIG. 29. Skull of burial 67, a male aged 35 years







0 10 cm



FIG. 30. Views of all aspects of skulls showing the range of sizes in both sexes and a typical child skull. The individuals are in the same position in each view. Top row: large female (burial 77), large male (burial 38), child (burial 37). Middle row: average female (burial 73), average male (burial 72). Bottom row: small female (burial 110), small male (burial 67). These skulls illustrate the cranial and facial characteristics discussed in the text



0 10 cm



0 10cm





FIG. 31. Skull of burial 37, a child aged 9 years



FIG. 32. Skull of burial 101, an infant aged 1.5 years. Although the facial skeleton is distorted, this is the best preserved of the infant skulls



FIG. 33. Posterior views of mandibles of a selection of adults, illustrating the range of morphology. The mandibles are large. They are particularly broad at the condyles but relatively less so at the gonial angle. The body is long, and although not high at the chin, it is midway along the body. The gonial angle is steep. A: burial 19, female aged 25 years. B: burial 61, female aged 48 years. C: burial 131, male aged 25 years. D: burial 72, male aged 32 years

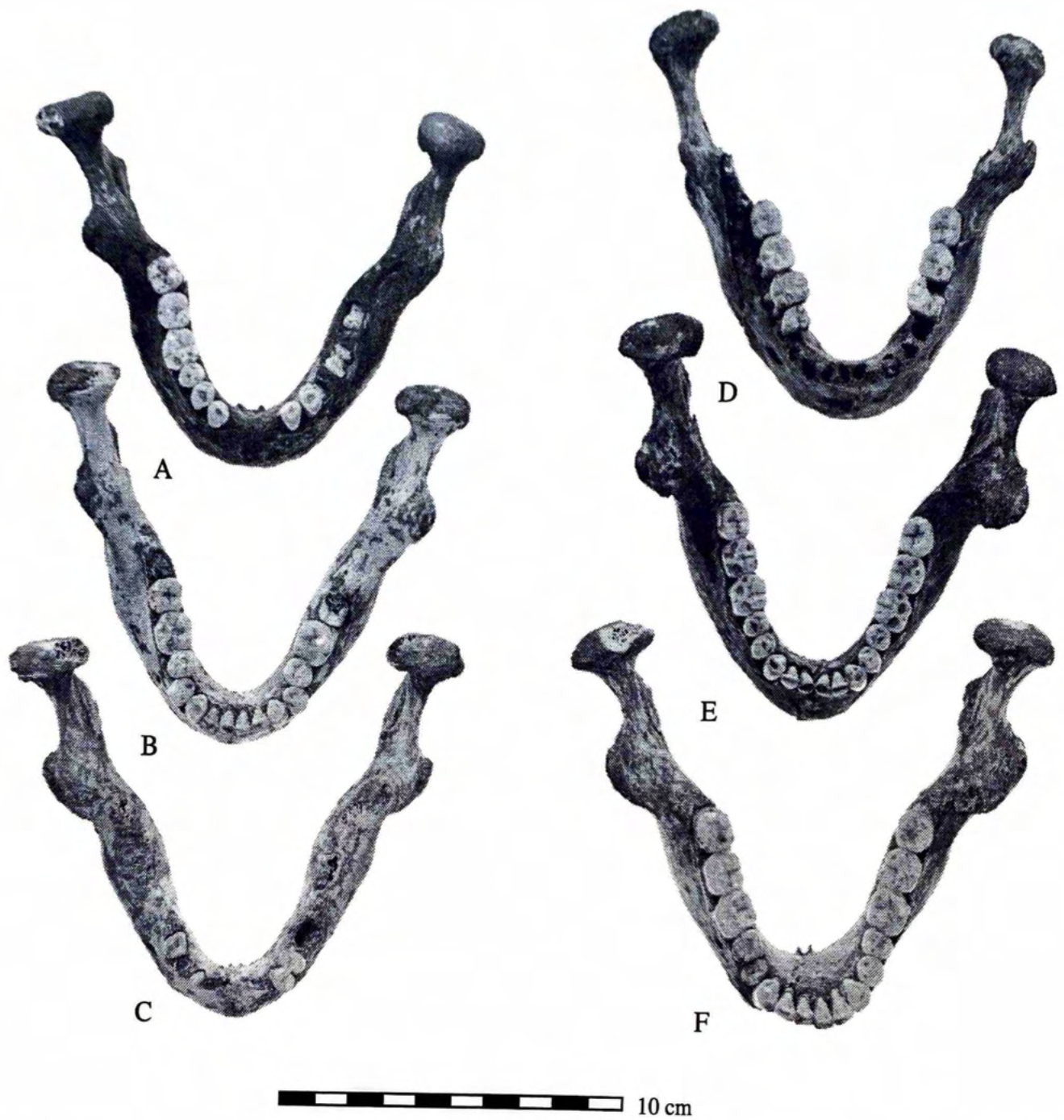


FIG. 34. Superior view of mandibles of a selection of adults. A: burial 19, female aged 25 years. B: burial 73, female aged 25 years. C: burial 61, female aged 48 years. D: burial 9, male aged 30 years. E: burial 131, male aged 25 years. F: burial 72, male aged 32 years



FIG. 35 Long bones from adults, illustrating the range of size and morphology. The smaller bones of each pair are from burial 39 (female) and the larger bones from burial 67 (male). All bones are from the left side, except the clavicles



FIG. 36. Long bones from burial 17 (12 years)



FIG. 37. Typical infant skeleton (burial 143, neonate)

Key for Table 29:

ML - Maximum length (from glabella to opisthocranium) (Martin no. 1)

MB - Maximum breadth (from euryon to euryon) (Martin no. 8)

MH - Maximum height (basion bregma height; from basion to bregma) (Martin no. 17)

MFB - Minimum frontal breadth (from fronto-temporale to fronto-temporale) (Martin no. 9)

BZB - Bizygomatic breadth (from zygion to zygion) (Martin no. 45)

BMB - Bimaxillary breadth (from zygomaxillare to zygomaxillare) (Martin no. 46)

IO - Interorbital breadth (maxillofrontale to maxillofrontale) (Martin no. 50)

UPH - Upper facial height (from nasion to prosthion) (Martin no. 48)

NH - Nasal height (from nasion to the mean of the lowest points of the right and left nasal margin) (Howells NLH)

NB - Nasal breadth (the greatest transverse diameter of the nasal cavity) (Martin no. 54)

PB - Palatal breadth (external) (Martin no. 61)

PL - Palatal length (internal from incisive to postnasal spine). This was measured as the distance between the incisive foramen and staphylion. This was used in preference to Martin no. 62 because of the alteration to the anterior maxillary alveolar in the large number of cases of missing incisors

BPL - Basion-prosthion length (from basion to prosthion) (Martin no. 40)

CBL - Cranial base length (from basion to nasion) (Martin no. 5)

TABLE 30: *Adult cranial and facial measurements: summary statistics (mm)*

	Females					Males				
	n	mean	s.d.	min.	max.	n	mean	s.d.	min.	max.
Maximum cranial length	15	169.0	7.6	150.0	182.0	7	180.3	4.8	172.0	185.0
Maximum cranial breadth	15	134.8	4.7	127.0	141.0	7	145.6	2.1	144.0	150.0
Maximum cranial height	12	134.1	5.6	125.0	141.0	5	144.8	3.9	139.0	149.0
Minimum frontal breadth	23	93.8	5.1	87.0	108.0	14	100.9	4.4	95.0	110.0
Bizygomatic width	15	130.6	5.3	123.0	141.0	5	146.2	4.3	142.0	151.0
Bimaxillary width	18	102.9	5.2	94.6	113.0	9	110.6	4.9	104.0	118.0
Inter orbital width	21	25.3	2.4	21.0	29.2	8	27.8	1.2	26.0	30.3
Upper facial height	14	67.4	3.4	62.4	73.0	4	73.4	1.2	72.5	75.0
Nasal height	18	49.5	2.3	46.2	54.0	8	53.1	2.8	46.7	55.4
Nasal breadth	18	27.0	1.6	23.8	30.8	10	28.0	1.4	26.2	30.5
Palatal breadth	15	65.1	3.7	60.0	72.9	11	68.9	2.4	63.0	71.8
Palatal length	13	42.7	1.9	38.8	45.5	9	46.4	1.0	45.0	48.0
Basion prosthion length	9	97.3	5.7	88.0	104.0	4	101.3	1.0	100.0	102.0
Cranial base length	12	95.8	4.7	89.0	102.0	5	101.6	4.7	96.0	108.0

TABLE 31: *Adult cranial and facial measurements: indices*

	Females			Males		
	n	mean	s.d.	n	mean	s.d.
Cranial (length/breadth)	14	79.9	3.3	7	80.2	2.9
Vault height (length/breadth/height)	12	88.5	3.0	5	88.4	2.7

TABLE 32: *Infant and child cranial and facial measurements (mm)*

Burial	Age	ML	MB	MH	MFB	BZB	BMB	IO	UFH	NH	NB	PB	PL	BPL	CBL
88.0	0.75				81.5										
101.0	1.5				85.7										
96.0	4.0				90.9			22.0	54.0	37.0					
21.0	8.0	170.0	132.0	126.0	88.0		83.0	19.5	51.9	37.9	22.0	58.1	33.1	86.0	83.0
1.0	9.0				86.6		82.5	20.8	58.5	40.2	22.6	54.6			
37.0	9.0	175.0	131.0	130.0	96.5	122.0	88.2	24.7	60.3	33.9	23.0	60.5	36.6	94.8	87.0
32.0	11.0				91.7			19.9	57.8		24.9				
11.0	12.0	170.0	132.5	127.0	89.9	130.5	96.7	23.0	46.0	24.7	24.7				90.0
17.0	12.0				94.0		92.4		63.0	44.7	22.1	64.8	40.9		

TABLE 33: *Adult mandibular measurements (mm)*

	ML	BicB	BigB	RB	SH	BH	BimB	BfB	RH	GA°
Females										
4	109.0	121.5	94.0	35.0		30.5	79.5	48.5	50.8	128
13	99.0	121.3	95.0	31.5			83.1	49.7	57.0	122
15				35.0					55.0	
18	105.0	117.5	83.4	32.3			77.7	47.2	55.0	122
19	93.0	128.3	93.9	35.1		26.7	86.0	50.0	65.0	109
25	101.0	117.7	88.5	37.7		32.0	81.2	49.8	65.0	118
26	107.0	125.3	96.0	30.4		29.9	83.8		65.0	123
27	111.0	131.8	97.5	33.0			91.1	51.9	61.1	119
31	101.0	127.5	86.9	35.0		30.7	82.8	49.0	65.0	127
35	106.0	129.5	91.5	38.8		31.9	88.0	49.0	62.0	117
36	106.0		96.5	36.4		35.0	87.0	52.9	69.0	113
39	96.0	120.5	93.0	35.2	31.5	31.2	83.1	48.0	59.0	119
40	106.0	124.3	92.5	33.4		28.8	80.5	49.8	63.0	113
45	100.0	123.5	95.3	33.3		32.5	80.5	46.7	58.0	119
47	98.0	121.6	96.5	28.8	27.5	26.1	83.5	47.4	52.5	123
56			94.3	33.0		32.8	84.0	47.8		
58	109.0	127.9	93.7	36.3	29.2	29.4	82.6	47.7	53.3	128
60	102.0	123.1	92.8	33.7	29.1	33.0	83.7	47.5	52.0	126
61	110.0	132.2	98.0	38.6			86.4	48.4	67.2	113
64	110.0	109.5	88.7	32.0	30.0	25.7	74.5	44.6	48.8	128
73	102.0		92.6	36.7	32.4	31.7	78.0	48.5	63.4	113
77	111.0	124.5	99.5	37.5	35.1	31.0	81.5	51.2	66.0	116
79	105.0	122.6	88.6	38.8			78.8	48.5	58.0	113
83	106.0	118.5	85.8	38.0	30.0	30.9	72.4	45.9	59.0	117
87	106.0	123.6	90.4	39.7	35.7	33.7	82.1	52.0	62.0	113
94	98.0	122.9	92.3	35.0	32.8	31.2	82.9	46.9	57.5	113
102	99.0	123.8	100.5	32.1	27.0		82.0	46.9	54.0	119
107	100.0	124.5	87.7	38.0	32.5		81.5	46.6	59.0	113
109				34.0	32.8	31.0			56.0	119
110	102.0	116.5	88.6	33.5		25.8	74.7	45.3	58.2	120
112	100.0			37.0	32.7	32.3			72.0	104
113			99.4	35.5				43.9	64.0	111

TABLE 33: *Adult mandibular measurements (mm) (cont.)*

	ML	BicB	BigB	RB	SH	BH	BimB	BfB	RH	GA°
Females										
122	107.0	122.9	89.8	33.8			81.2	45.5	58.0	120
140	100.0	121.6	86.4	38.5	31.8	30.3	77.1	43.6	62.7	110
142					31.6	29.4	83.0	47.5	62.0	119
Males										
9	99.0	114.2		31.5	30.8	29.3	77.0	43.7	62.6	121
24	111.0	130.6	106.1	33.4		30.3	89.2	50.0	62.3	118
29	105.0	127.7	106.0	33.4	31.1	32.3	82.7	49.8	63.0	119
38	105.0	135.7	100.3	35.2	38.0	31.3	91.9	49.1	56.0	119
42	106.0	128.7	112.6	34.5	35.0	31.3	90.0	51.9	69.6	119
43	112.0	128.8	104.3	38.2	37.2	34.2	84.0	50.1	66.0	114
44	110.0		100.2	37.3		33.4	88.9	50.6	66.9	
67	103.0		94.4	35.0		30.0	80.0	51.5	62.3	116
72	115.0	134.5	116.4	36.8	36.1	37.3	99.4	50.3	80.5	105
74	111.0	126.2	92.3	39.8	34.0	31.8	82.0	51.9	64.9	111
76	112.0	140.0	108.1	40.3	38.0	35.0	91.2	49.3	61.0	121
86	110.0	136.4	111.0	34.0	32.6	32.3	97.6	50.6	68.0	112
90	105.0	125.0	101.5	37.3	31.3	30.0	80.2	47.5	66.7	116
91	105.0	117.5	102.5	35.0	31.4	28.3	79.7	46.0	64.0	115
92	109.0	132.6	94.0	36.8	35.0	32.2	83.5	50.4	76.5	114
93	109.0	145.3	101.2				97.0	49.7	68.6	104
100	113.0	133.3	101.0	37.0	32.3	31.8	84.3	48.2	66.8	120
103	113.0	126.3	111.0	36.4	33.6	31.0	91.8	51.8	62.0	121
115	106.0	132.4	92.2	35.6	35.1	33.7	82.1	46.5	64.0	112
117	109.0	137.5	103.0	39.4		36.5	91.5	49.8	72.2	108
120				34.6		30.2				
129	102.0	128.0	97.5	37.0	35.7	34.0	85.7	47.8	61.2	115
131	108.0	131.3	94.0	33.3	34.3	29.3	84.0	50.5	63.3	116
132	106.0	124.7	96.3	38.8	31.1	29.9	83.7	50.1	63.7	105
147	113.0	132.7	98.2	45.0	38.6	34.7	87.5	48.5	64.4	116
152				34.1	31.0	29.4				

ML - Mandibular length (from the most posterior points on the condyles to the most anterior point of the chin. Martin no. 68.1).

BicB - Bicondylar breadth (between the lateral points of the condyles. Martin no. 65)

BigB - Bigonial breadth (from gonion to gonion. Martin no. 66).

RB - Ramus breadth (the minimum breadth of the ramus, measured at right angles to the posterior border of the ramus. Taken on the left when possible. Martin no. 71)

SH - Symphyseal height (the height at the anterior chin, from gnathion to infradentale. Martin no. 69).

BH - Body height (The height from the alveolar crest, between the first and the second lower molar, to the base of mandible. Taken on the left when possible. Martin no. 69.2).

RH - Ramus height (The height from the most superior point on the condyle to gonion. Taken on the left when possible. Martin no. 70).

BimB - Bimandibular foramen breadth. The distance between the mandibular foramina.

BfB - Bimental foramen breadth (The distance between the mental foramina. Martin no. 69.3).

GA - Gonial angle (The angle formed by inferior border of the body and the posterior border of the ramus. Taken on the left when possible. Martin no. 79)

TABLE 34: *Adult mandibular measurements: summary statistics*

	Females					Males				
	n	mean	s.d.	min.	max.	n	mean	s.d.	min.	max.
Length	30	103.5	4.8	93.0	111.0	24	108.2	4.0	99.0	115.0
Bicondylar breadth	27	123.1	4.8	109.5	132.2	22	130.4	6.9	114.2	145.3
Bigonial breadth	31	92.6	4.4	83.4	100.5	23	101.9	6.8	92.2	116.4
Ramus breadth	34	35.1	2.7	28.8	39.7	24	36.4	2.9	31.5	45.0
Symphyseal height	16	31.4	2.4	27.0	35.7	20	34.1	2.6	30.8	38.6
Body height	25	30.5	2.4	25.7	35.0	25	31.9	2.4	28.3	37.3
Bimandibular foramen breadth	31	81.8	4.0	72.4	91.1	24	86.9	6.0	77.0	99.4
Bimental breadth	31	48.0	2.3	43.6	52.9	24	49.4	2.0	43.7	51.9
Ramus height	34	59.9	5.5	48.8	72.0	24	65.7	5.2	56.0	80.5
Gonial angle	33	117.8	6.0	104.0	128.0	23	114.7	5.2	104.0	121.0

TABLE 35: *Infant and child mandibular measurements (mm)*

Burial	Age	ML	BicB	BigB	RB	SH	BH	BimB	BfB	RH	GA°
34	0.0	51.0			15.9	11.8				17.6	135
48	0.0	49.0			15.0	10.2				15.4	140
54	0.0	45.4				9.6				15.2	130
65	0.0	51.9			16.1	11.6				15.0	140
66	0.0	49.4			16.5	11.5				14.4	
69	0.0	50.0			14.7	10.6				17.0	140
75	0.0	52.6			16.2	9.7				16.9	
124	0.0	52.7				14.3				16.5	137
128	0.0	56.6			17.6	11.1				19.0	
136	0.0	52.9				12.6				15.0	131
141	0.0	47.8			15.1	11.0				15.5	139
143	0.0	50.3			17.2					16.9	125
144	0.0	52.8			17.2	12.3				17.3	138
133	0.4	63.5			18.6	16.1			32.5	22.5	
62	0.5	71.4			20.3	19.0		53.7	36.1	28.7	135
89	0.5	67.2	77.3		20.4	17.4		54.8	37.5	26.7	133
134	0.5	62.7			18.0	15.7				23.6	
88	0.75	73.2	77.3		23.4	18.8		51.2	37.9		133
14	1.25	68.8			20.3	20.3			33.8	29.6	135
121	1.25	74.1	84.8	60.8	20.2	18.7		53.0	33.8	28.8	136
101	1.5	77.4	84.3	63.1	25.0	20.5		55.1	36.1	34.3	
7	1.75	78.3	80.0	62.8	21.3	21.8		55.5	35.4	30.4	141
123	2.0	78.0	84.8	61.3	23.3	19.3		56.5	36.7	31.2	136
150	2.5	77.2			23.2	19.4				31.3	126
96	4.0	88.1		68.9	27.0	25.3				38.7	122

TABLE 35: *Infant and child mandibular measurements (mm) (cont.)*

Burial	Age	ML	BicB	BigB	RB	SH	BH	BimB	BfB	RH	GA°
21	8.0	101.8		71.9	27.9	27.5	21.6		40.7	43.2	129
1	9.0	101.8		86.0	31.0	30.0	21.3		42.0	47.0	124
37	9.0	108.7	110.8	84.2	32.5	26.9	23.3	75.9	46.6	49.6	124
32	11.0	105.4	105.3	94.0	25.9	27.3	20.3	81.6	44.8	44.1	140
11.0	12.0	113.0	112.0	93.8	30.4	29.3	25.2	75.8	46.4	49.5	126
12.1	12.0	78.8			30.3		21.1			47.7	126
17.0	12.0	110.7	111.0	90.9	30.3	29.9	26.2	77.7	47.6	49.4	134
20.0	12.0	114.0			32.0	29.1	25.4			51.6	126

Key as for table 33

TABLE 36: *Dental measurements, maxilla (mm)*

Females	MD 11	MD 21	BL 11	BL 21	MD 12	MD 22	BL 12	BL 22	MD 13	MD 23	BL 13	BL 23	MD 14	MD 24	BL 14	BL 24
4					7.9		6.8			7.6		8.8				
15										7.2		7.3	6.2	6.9	8.8	8.7
19									8.0		8.2		7.6		9.6	
25										7.4		8.8		7.6		10.1
31									8.0		8.5					
35									7.6		8.5		7.5	7.4	10.5	10.3
36						8.2		6.9	8.5	9.0	9.0	9.0	7.7	7.8	10.0	10.2
39									7.8		7.6		6.9	6.7	9.1	8.4
47									7.8		8.5		7.3	7.0	9.4	9.4
58	9.4	9.0	7.6	7.4		6.8		6.5		7.1		8.4		7.3		9.7
60	8.0	7.8	6.3	6.4									7.5	7.6	9.7	9.9
64									7.3		7.4			6.7		9.3
73													7.6	7.7	9.2	9.7
83	8.5	8.7	7.1	7.6	7.2	7.8	6.0	6.6					7.5	7.5	9.4	9.0
87		9.1		7.6	6.5	7.0	6.2	6.6	7.8	8.0	7.7	7.7	7.8	8.0	10.0	9.9
102					6.6		6.3						6.6		8.6	
107					6.6		6.5									
109	8.9		7.0						7.2	7.2	8.1	8.2	7.2	7.2	9.1	9.7
112	8.9	8.9	7.6	7.4		7.0		6.4					7.8		10.4	
140	8.2	8.5	7.0	6.9	6.8	6.6	6.6	6.1	7.2	7.4	7.7	7.4	7.2	7.4	8.8	9.1
142									7.3	7.8	8.0	7.9	7.5		9.6	

Key for tables 36-39:

M-D mesiodistal; B-L buccolingual

Tooth numbers are according to the Fédération Dentaire Internationale (1971) system; the first digit represents the quadrant - 1 = right maxilla, 2 = left maxilla, 3 = left mandible, 4 = right mandible. The second digit represents the tooth in the quadrant, numbered from the midline 1 = central incisor, 2 = lateral incisor, 3 = canine, 4 = first premolar, 5 = second premolar, 6 = first molar, 7 = second premolar, 8 = third molar.

TABLE 36: *Dental measurements, maxilla (mm) (cont.)*

Females	MD 15	MD 25	BL 15	BL 25	MD 16	MD 26	BL 16	BL 26	MD 17	MD 27	BL 17	BL 27	MD 18	MD 28	BL 18	BL 28
4	7.0		9.8		11.4	12.0	12.1	12.4								
13										10.2		10.5				
15	5.9	6.5	8.8	8.7	10.2	9.8	11.2	11.0	9.8	10.1	11.3	11.2				
19	7.3		10.0		10.8	11.5	11.6	11.5	9.3	9.3	10.5	10.5	9.0	9.0	10.5	10.5
25					10.1	10.8	11.7	11.3	9.0	10.0	12.3	11.2	8.6	9.2	10.8	10.3
26													9.4		10.4	
31	6.2	6.8	8.9	9.0	10.8	10.2	11.7	11.6	9.9	9.5	11.2	10.8	8.6	9.0	10.2	10.0
35	7.0	7.7	9.8	9.8	11.5	11.5	12.2	12.1	11.2	12.1	12.3	12.6	7.8	9.0	10.7	11.8
36	7.6	7.8	10.7	10.5	11.2	10.9	12.6	12.5	11.1	11.1	12.4	13.0	10.4	10.6	12.4	12.4
39	6.8	6.8	8.8	8.6	9.8	10.0	11.0	10.9	10.0		11.2					
47	6.2	6.2	9.3	9.4	10.6	12.1	11.5	11.5	9.6	10.0	11.2	11.6	8.5	7.7	11.1	11.3
58					10.5	10.8	11.6	11.3	9.2		11.5		8.8		9.7	
60	6.6	6.7	9.5	9.5	10.3	11.4	10.8	11.2	9.4	10.0	9.4	10.7	9.2	8.8	9.8	9.5
61									10.3		11.2					
64	6.3	6.6	9.3	9.1	10.0	9.7	11.2	11.5	9.0	10.0	11.4	10.9	9.0	9.3	10.7	10.3
73	7.0	7.6	9.6	9.7	10.8		11.2		9.8	9.4	11.2	10.7		8.5		11.0
77									12.5		11.7					
79									9.0		11.3		9.1		11.3	
83	7.1	6.5	9.0	8.9					9.6	9.8	10.6	10.6	9.8	9.7	10.3	10.6
87	7.5	7.3	9.9	9.9	11.2	11.0	12.2	12.4	11.5	10.0	11.9	12.0	10.6	11.6	11.2	12.0
94													9.5		10.7	
102	6.0		8.2		10.0		10.8		8.9	9.1	10.6	10.3	7.6	8.1	8.9	9.4
107										10.2		11.4				
109	7.0	7.2	9.4	9.4		10.4		11.7	10.2	10.5	11.5	11.4				
110										10.8		11.7				
112	7.8		10.2							10.3		11.7		7.8		9.3
140	6.5	6.5	8.6	8.4	10.7	11.8	11.3	11.3	10.0	10.5	10.6	10.3	10.8	10.5	11.1	11.2
142										10.8		11.4				

Males	MD 11	MD 21	BL 11	BL 21	MD 12	MD 22	BL 12	BL 22	MD 13	MD 23	BL 13	BL 23	MD 14	MD 24	BL 14	BL 24
8														7.5		9.7
23										7.9		7.9				
24						8.5		7.1	7.2	7.7	8.5	8.3				
29					7.0		6.7							7.9		10.5
38										8.4		8.7		7.5		9.7
42														7.0		9.5
43										7.0		8.0		7.1		9.4
57														7.0		9.0
72	9.4	9.7	8.0	8.3						8.3		8.8	7.7	7.9	10.1	10.0
86									8.2	7.9	8.8	8.8	7.6	7.0	9.7	9.6
90	9.8		7.7		7.5	7.5	6.8	6.6	8.0	8.3	9.0	9.0	7.0	7.5	9.2	9.4
92									8.1		8.7					
100	8.4		6.9						8.4	8.5	8.5	8.5	7.4	7.9	9.9	10.0
103	8.5		7.5						7.6	7.7	8.7	8.2	7.0	6.8	9.4	9.3

TABLE 36: *Dental measurements, maxilla (mm) (cont.)*

Males	MD 11	MD 21	BL 11	BL 21	MD 12	MD 22	BL 12	BL 22	MD 13	MD 23	BL 13	BL 23	MD 14	MD 24	BL 14	BL 24
117	9.5	9.4	7.6	7.7		7.9		7.0	8.6	8.5	8.6	8.9	8.0	8.0	9.9	9.9
120									8.0	8.0	7.9	8.1	6.6	6.8	9.5	9.1
129									7.8	8.0	7.5	7.9	7.1	7.1	9.6	9.4
131													6.4	6.6	9.2	9.8
132														7.9		10.0
147	9.0		7.0										7.4		10.0	

Males	MD 15	MD 25	BL 15	BL 25	MD 16	MD 26	BL 16	BL 26	MD 17	MD 27	BL 17	BL 27	MD 18	MD 28	BL 18	BL 28
8		6.7		9.0		11.5		11.7		10.0		11.5				
9													9.7		11.0	
23														9.3		10.9
24						11.9		12.3	9.8	11.0	12.2	11.7	8.6		12.0	
29									11.0		12.5		10.2	10.3	12.0	13.0
38	7.2	7.0	9.5	9.3									10.3	9.0	11.3	11.3
42	6.1	6.2	9.0	8.8										10.5		11.9
43	7.0	7.0	9.8	9.6					9.1	9.4	11.7	11.7		9.4		11.7
44						11.6		12.1	10.2	10.7	11.6	11.7		8.9		10.7
57	7.1	7.0	8.6	8.7		11.3		12.0	9.3	9.2	10.9	11.2	8.7	9.0	10.5	10.5
72									10.5	10.7	11.9	11.5	9.3	9.7	11.7	11.5
76		7.1		9.3	10.8		11.5		10.0		12.0			9.0		10.7
86					10.9	10.8	12.2	12.4	9.8	10.0	11.1	11.6				
90	6.7	7.5	9.4	9.4	10.9	11.2	12.2	12.0	9.8	9.9	11.7	11.9		10.0		11.7
92	7.2	7.6	9.4	9.6	10.8	11.2	12.2	11.9	9.0	9.0	12.2	12.6				
100		6.9		9.9					9.6	10.1	11.5	11.9	9.3		11.9	
103	6.0		9.2		10.3	10.9	11.7	11.3	9.3		12.0					
117	7.0	7.0	9.5	9.3	11.9	11.4	12.3	12.4	8.7	10.2	12.0	12.4	9.6		11.3	
120	6.5	6.8	8.9	8.5	11.0	11.3	11.2	11.3	8.0	8.7	10.2	10.8	8.5	9.0	9.4	10.2
129	6.6	6.8	9.4	9.4	10.2		11.7		10.1	10.0	12.1	11.8	9.0	9.7	11.0	11.7
131		5.8		8.8		9.9		10.7	9.2	10.3	11.1	10.7	8.7		10.3	
147	7.4	7.1	9.5	9.4	11.1		11.5		10.5	9.5	11.1	10.9		8.8		11.1

Children	MD 11	MD 21	BL 11	BL 21	MD 12	MD 22	BL 12	BL 22	MD 13	MD 23	BL 13	BL 23	MD 14	MD 24	BL 14	BL 24
1													7.2		8.3	
6																
11						6.6		6.0	7.4	7.6	8.3	8.2	7.3	7.6	8.3	8.6
12.1	8.5	8.3	6.8	6.7		8.1		6.7	7.5	7.6	7.7	7.6	6.3	7.0	7.7	7.7
17	8.7	9.0	8.0	7.9	7.2		6.6		8.1	8.4	8.6	9.0	6.5	6.5	10.2	9.7
20					8.0	8.0	6.5	6.8					6.7	7.0	9.2	9.5
21		9.2		7.3	7.4	7.1	6.6	6.6								
32	9.0	9.0	7.4	7.6		6.9		6.7	8.0	6.7	8.0	7.7	6.1	6.3	9.2	9.3
37	8.9	9.0	8.0	8.0	7.9	7.7	6.2	6.1	8.3	8.3	7.8	7.8	6.7	6.7	10.0	10.0

TABLE 36: *Dental measurements, maxilla (mm) (cont.)*

Children	MD 15	MD 25	BL 15	BL 25	MD 16	MD 26	BL 16	BL 26	MD 17	MD 27	BL 17	BL 27	MD 18	MD 28	BL 18	BL 28
1					11.0	10.9	10.8	10.4								
6						10.9		11.5								
11	6.3	6.8	8.5	9.5	10.7	10.1	10.7	10.2	9.3	9.0	11.0	10.1				
12.1	6.5	6.5	7.7	8.1	10.0	10.2	10.7	10.8	8.3	8.9	9.2	10.0				
17	5.8	6.2	10.0	9.3	10.8	10.6	11.8	11.7	8.8	8.2	11.4	11.1				
20	6.4	6.3	9.2	9.4	11.9	10.8	12.6	12.5	9.3	10.0	11.4	11.4				
21					10.4	10.2	11.6	11.8								
32	5.8	5.6	9.4	9.3	10.0	10.3	11.7	11.6	9.5		11.5					
37					11.1	11.4	11.8	12.1	10.0	9.4	11.5	11.9				

TABLE 37: *Dental measurements, mandible (mm)*

Females	MD 41	MD 31	BL 41	BL 31	MD 42	MD 32	BL 42	BL 32	MD 43	MD 33	BL 43	BL 33	MD 44	MD 34	BL 44	BL 34
4									7.2	7.3	8.2	7.9	7.8		8.6	
15						5.5		5.9		5.8		6.9		7.0		7.5
18									6.7		7.2					
19									7.0	6.8	7.2	7.4	6.8	7.0	8.1	8.0
25									7.0	7.0	8.1	7.9	7.4		8.2	
26													6.8	7.0	8.0	8.3
31									6.9		7.9		6.3	6.4	7.9	8.1
35														7.0		9.1
36									7.7	7.9	8.2	8.4	8.0	7.3	8.8	8.9
39																
47	6.0	6.1	6.2	5.8	6.3	6.7	6.1	6.1	6.9		7.5		7.1	6.9	7.5	7.5
56													7.1		8.0	
58									7.1	7.3	8.1	7.8	7.2	7.3	7.8	7.6
60	5.5	5.1	5.0	4.9	5.7	5.5	5.4	5.7	6.8	7.0	7.5	7.1	7.2		7.3	
64	5.8	5.2	6.0	6.0	6.3		6.1			6.6		7.7		6.9		7.4
73													7.4	7.4	8.0	8.6
77																
79														6.8		8.3
83						6.0		6.1					7.1	7.0	8.0	7.8
87	6.2	5.6	6.9	5.9	6.6	6.4	6.4	6.6	7.0	7.0	7.8	7.6	7.7	7.7	8.5	8.7
94													7.0	6.8	8.5	8.5
102																
107													6.9	7.0	8.6	8.8
109					6.7	6.3	6.3	6.2	7.4		8.0		6.9		7.8	
110																
112					5.8		6.0						7.3	7.4	8.7	8.5
140		5.9		6.0	6.2	6.3	6.4	6.2	6.4	6.3	7.3	7.3	7.1	7.3	8.1	7.8
142													7.3	7.2	8.0	7.9
154									7.1		6.5		7.8		8.3	

TABLE 37: Dental measurements, mandible (mm) (cont.)

Females	MD 45	MD 35	BL 45	BL 35	MD 46	MD 36	BL 46	BL 36	MD 47	MD 37	BL 47	BL 37	MD 48	MD 38	BL 48	BL 38
4	8.2		9.0		12.1	12.8	11.2	11.3	11.3	12.0	10.6	10.7	11.3	12.0	10.4	10.4
15	6.8	6.9	8.0	8.2						11.0		10.4	9.9		10.4	
18	7.3		8.4		11.9		11.1									
19	6.5		8.0		11.7		10.9		10.1		9.7					
25	7.1	6.8	8.5	8.5	11.5	11.8	11.2	11.1	11.0	10.4	10.7	10.7	11.0	10.7	10.0	10.6
26									10.0		9.6					
31	6.7	6.3	8.1	7.9	11.3	11.3	11.3	11.2	10.3		10.1		11.1	10.9	10.4	10.3
35	8.0	7.8	9.1	9.0	12.0		11.9		12.0	12.1	11.4	11.0				
36	8.0	8.0	9.1	8.8	13.1	12.2	12.4	12.2	11.1	11.2	11.3	11.1	12.4	13.3	12.0	12.2
39					11.5	11.1	10.9	11.0	11.0	11.2	10.1	10.2				
47					11.7	11.6	10.9	10.4	11.0	10.6	10.4	10.3	10.9	11.1	9.8	10.1
56									10.6	10.9	10.2	10.4	10.4	10.0	10.0	9.6
58	7.6	7.8	8.8	9.3	11.6	11.2	10.7	10.9	11.2		10.6					
60	7.3	7.0	8.2	8.2	11.2	10.0	10.6	10.3	10.4	10.0	9.7	9.6		10.2		8.9
64	6.6	6.6	7.4	8.0	10.7	11.3	10.6	10.5	10.0	9.9	9.8	9.8	10.3	11.0	9.9	10.3
73	8.0	7.8	8.9	8.6	12.0	12.3	11.8	11.5	10.0		10.4					
77	8.0	7.9	9.6	9.5									12.4		11.7	
79		7.7		8.4		11.0		10.7								
83	7.8	7.6	8.3	7.8	11.9	11.2	10.9	10.8	9.6	10.3	9.7	9.8	11.1	11.5	9.9	9.5
87	7.8	7.6	8.8	8.8	12.6	12.2	11.5	11.1	11.6	11.5	11.5	11.2		12.0		10.9
94	7.5	7.1	8.3	7.9	12.2	11.7	11.0	11.0	11.3	10.8	10.3	10.6	12.3		11.1	
102	6.8		7.9		11.1		10.6									
107	7.0	7.1	8.8	8.1												
109	7.7	7.3	8.0	8.2	12.4	11.8	11.2	11.5	11.1		10.9			10.6		11.6
110		7.1		8.3					11.3	10.6	10.2	10.3				
112	8.2	8.4	8.8	8.5					11.7	11.5	11.1	11.5				
140	7.4	7.3	8.0	8.3	11.9	12.1	10.6	10.9	10.5	10.8	9.7	9.8	11.1	11.4	10.0	9.8
142	7.2	7.2	8.5	8.3	11.2	11.3	11.1	11.0	11.5	11.2	11.1	11.1	10.0	11.0	10.4	9.7
154	7.9	8.3	8.8	9.2												
Males	MD 41	MD 31	BL 41	BL 31	MD 42	MD 32	BL 42	BL 32	MD 43	MD 33	BL 43	BL 33	MD 44	MD 34	BL 44	BL 34
8	6.2	6.2	5.8	5.9	6.4	6.2	6.4	6.5	7.8	8.0	8.0	7.5	8.3	7.8	8.1	8.5
9																
23									7.0	7.1	8.2	8.4	7.6	7.4	8.6	8.2
24						6.8		6.7	7.2	7.2	8.2	8.2		8.2		8.6
29		5.9		6.2		6.4		6.8						7.8		9.0
30	6.5	6.4	5.7	6.1	6.9	7.2	6.6	6.7	8.0		7.9		8.0		9.0	
38													7.7	7.8	9.3	9.1
42									7.0		8.3		7.2		8.0	
43										6.4		7.0		6.8		8.1
44																
57		6.2		5.8									7.1		7.9	
67																

TABLE 37: *Dental measurements, mandible (mm) (cont.)*

Males	MD 41	MD 31	BL 41	BL 31	MD 42	MD 32	BL 42	BL 32	MD 43	MD 33	BL 43	BL 33	MD 44	MD 34	BL 44	BL 34
72	5.2	6.2	6.9	6.8	6.5	6.3	7.3	7.0	7.2	7.4	9.1	9.1		7.7		9.0
76		5.3		6.0	6.0	5.9	6.1	6.4					7.0		7.8	
86		6.0		6.1		6.7		6.9	7.5	7.5	8.8	8.7	7.4	7.1	8.2	8.0
90									7.4	7.2	8.1	8.6	7.4	7.7	8.0	8.2
92	5.8	5.8	6.1	6.0	6.0	6.2	6.6	6.9	7.0	7.4	7.7	7.8	7.4	7.5	8.1	8.2
93													7.8		9.1	
100	5.3		5.9		5.3	5.8	6.4	6.2	7.4	7.3	8.1	8.3	7.0		8.9	
103	5.2		5.9		5.6	5.9	6.9	6.2	6.6	6.8	8.4	8.1	6.5	7.0	7.6	8.0
117	5.8	5.8	6.2	5.9	6.4	6.3	6.8	6.4	7.2	8.0	8.3	8.0	8.0	7.9	8.3	8.2
120					5.8		5.8						6.9	7.3	8.2	8.2
129										7.2		7.8	7.5	7.1	7.5	7.7
131					5.5	5.8	6.5	6.4	6.7	6.6	8.0	8.3	6.8	6.7	7.7	7.6
132	5.6	5.9	6.7	6.0	6.2	6.8	7.0	7.1	6.5	7.0	9.1	9.0	7.6	7.4	9.6	9.4
147									7.0	7.3	7.3	7.7	7.4	7.8	7.6	7.6
Males	MD 45	MD 35	BL 45	BL 35	MD 46	MD 36	BL 46	BL 36	MD 47	MD 37	BL 47	BL 37	MD 48	MD 38	BL 48	BL 38
8	7.1	7.9	8.0	8.1	11.7	12.1	11.6	11.7	11.0	12.4	11.0	11.1				
9	7.5		8.0		11.6		10.8		10.3	10.5	9.7	9.6	11.3	10.6	9.4	9.7
23		7.1		8.7		12.3		10.6		10.8		9.9	11.2		9.7	
24					12.4		11.4		11.8	11.1	10.9	10.8	11.5		10.9	
29	7.7	8.1	8.6	9.5									12.1		11.4	
30	7.9	7.6	8.9	8.7					11.0		11.3		12.0	11.5	11.5	11.1
38														11.0		10.4
42	7.0	7.3	8.2	8.0										10.9		10.2
43		7.2		8.7					11.1	10.5	10.9	11.7		10.7		11.0
44	8.3		9.0		12.8	12.8	10.8	11.2	11.0	11.6	10.9	10.8	10.6	10.5	10.4	10.2
57	7.4	7.3	7.9	8.1	12.4	12.1	11.4	11.3	10.6	10.6	10.6	10.5	10.3	10.6	9.8	10.3
67											11.3		10.7	11.6		10.6
72		7.0		9.6					10.8	11.1	11.0	11.0	11.9	10.7	11.1	10.9
76									10.8	10.9	11.0	10.3		10.6		10.9
86	6.8	7.0	8.1	8.2	11.5	12.0	11.8	11.3	10.7	10.4	9.9	9.9	11.1	11.0	10.0	9.7
90	7.7	6.8	8.3	8.3		11.5		10.8	11.1	11.3	10.7	10.9	11.7	12.0	10.7	11.3
92	7.2	7.5	8.7	8.6	11.8	11.7	11.0	11.5	11.7	11.5	11.1	10.9				
93																
100	7.1	7.0	8.8	8.7					10.5	11.0	10.0	10.4	11.2		10.4	
103	7.0	7.0	7.8	8.2	10.9	10.8	11.1	11.1	11.2	11.0	10.9	10.5	11.0		10.6	
117	8.0	7.8	8.6	8.4	12.0	12.2	11.2	11.3	10.6	11.2	11.0	11.2	11.4		10.6	
120	7.2	7.0	8.3	8.2	11.5	11.4	10.8	10.9	9.6	10.0	9.9	10.5	10.3	10.0	10.0	9.8
129	7.2	7.4	8.0	7.9		11.6		10.5	12.0	10.5	10.2	10.3	11.8	12.2	10.7	10.4
131	7.0	6.6	7.7	7.6	11.5	10.8	10.5	10.6	11.0	10.3	10.2	10.3	10.0	10.3	9.8	10.1
132	7.3	7.6	8.8	9.6					11.4		11.6					
147	6.8	7.8	8.0	8.1	11.8	12.0	11.1	11.1	10.7	10.5	10.7	10.4	11.2	11.0	10.7	10.5

TABLE 37: *Dental measurements, mandible (mm) (cont.)*

Children	MD 41	MD 31	BL 41	BL 31	MD 42	MD 32	BL 42	BL 32	MD 43	MD 33	BL 43	BL 33	MD 44	MD 34	BL 44	BL 34
1													7.4		8.4	
6		6.2		5.9		6.7		6.5								
11	5.2	5.3	5.8	6.0	5.6	5.3	5.7	6.0		6.0		7.4	6.7	6.7	6.8	7.7
12.1	5.6	5.9	5.7	5.8	6.4	6.4	6.2	6.4		6.7	7.3	7.0	6.7	7.0	8.6	8.8
17	6.0	5.6	6.2		6.1	5.6	6.3	6.2	7.0	6.8	8.3	8.1	7.0	7.2	8.7	8.7
20	5.9	5.8	5.8	6.0		6.1		6.5	7.0	6.4	7.6	7.6	7.5	6.7	7.9	8.0
21	5.3	5.5	5.7	5.7	6.2	5.6	5.1	5.4								
32	5.4	5.7	5.9	5.9	6.2	6.2	6.1	6.3	7.0	7.1	7.2	7.3	6.7	6.1	8.0	8.1
37	6.3		6.1		7.0	7.0	6.4	6.4	7.6	7.6	7.5	7.5		7.2		8.3

Children	MD 45	MD 35	BL 45	BL 35	MD 46	MD 36	BL 46	BL 36	MD 47	MD 37	BL 47	BL 37	MD 48	MD 38	BL 48	BL 38
1	6.7		7.0		11.3		10.6									
6						12.2		10.6								
11	7.6	7.5	7.9	8.3	10.8	11.2	10.3	10.0	10.3	10.1	10.3	10.0	10.1		8.3	
12.1	7.5	7.1	8.8	8.4	10.2	10.9	9.5	9.4	10.0	9.8	9.2	10.0				
17	6.7	6.9	9.0	8.6	11.0	11.0	11.3	11.3	10.5	10.2	10.5	10.7				
20	7.7	7.4	8.4	8.1	10.8	10.6	10.8	11.1	10.0	11.1	10.1	10.7				
21					11.5	11.8	10.8	10.7								
32	6.7	7.2	8.5	8.4	11.0	11.0	11.1	11.3	10.0	9.5	10.4	10.4				
37					10.6	10.7	10.9	11.6	10.7	10.4	11.1	11.1				

TABLE 38: *Dental measurements, maxilla: summary statistics*

Females							Males						
	Left			Right				Left			Right		
	n	mean	s.d.	n	mean	s.d.		n	mean	s.d.	n	mean	s.d.
MD 1	6	8.70	0.50	6	8.70	0.50	BL 1	6	7.20	0.50	6	7.10	0.50
MD 2	6	7.20	0.60	6	6.90	0.50	BL 2	6	6.50	0.30	6	6.40	0.30
MD 3	9	7.60	0.60	11	7.70	0.40	BL 3	9	8.20	0.60	11	8.10	0.50
MD 4	14	7.30	0.40	15	7.30	0.50	BL 4	14	9.50	0.60	15	9.50	0.60
MD 5	13	6.90	0.50	17	6.80	0.60	BL 5	13	9.30	0.60	17	9.40	0.60
MD 6	15	10.90	0.80	16	10.60	0.50	BL 6	15	11.60	0.50	16	11.50	0.50
MD 7	20	10.20	0.70	20	10.00	1.00	BL 7	20	11.20	0.70	20	11.30	0.70
MD 8	14	9.20	1.10	16	9.20	0.90	BL 8	14	10.70	1.00	16	10.60	0.80
Males													
MD 1	2	9.55	0.21	6	9.10	0.57	BL 1	2	8.00	0.42	6	7.45	0.42
MD 2	3	7.97	0.50	2	7.25	0.35	BL 2	3	6.90	0.26	2	6.75	0.07
MD 3	12	8.02	0.43	9	7.99	0.42	BL 3	12	8.43	0.41	9	8.47	0.47
MD 4	16	7.34	0.47	10	7.22	0.50	BL 4	16	9.64	0.39	10	9.65	0.32
MD 5	14	6.89	0.46	11	6.80	0.46	BL 5	14	9.21	0.40	11	9.29	0.34
MD 6	11	11.18	0.52	9	10.88	0.49	BL 6	11	11.83	0.53	9	11.83	0.40
MD 7	15	9.91	0.65	17	9.64	0.74	BL 7	15	11.59	0.54	17	11.64	0.59
MD 8	13	9.43	0.56	11	9.26	0.63	BL 8	13	11.30	0.74	11	11.13	0.81

TABLE 38: *Dental measurements, maxilla: summary statistics (cont.)*

Children	Left			Right				Left			Right		
	n	mean	s.d.	n	mean	s.d.		n	mean	s.d.	n	mean	s.d.
MD 1	5	8.90	0.35	4	8.78	0.22	BL 1	5	7.50	0.52	4	7.55	0.57
MD 2	6	7.40	0.62	4	7.63	0.39	BL 2	6	6.48	0.34	4	6.48	0.19
MD 3	5	7.72	0.68	5	7.86	0.39	BL 3	5	8.06	0.57	5	8.08	0.37
MD 4	6	6.85	0.46	7	6.69	0.44	BL 4	6	9.13	0.85	7	8.99	0.93
MD 5	5	6.28	0.44	5	6.16	0.34	BL 5	5	9.12	0.58	5	8.96	0.88
MD 6	9	10.60	0.44	8	10.74	0.63	BL 6	9	11.40	0.77	8	11.46	0.68
MD 7	5	9.10	0.66	6	9.20	0.59	BL 7	5	10.90	0.83	6	11.00	0.90
MD 8	0	0.00	0.00	0	0.00	0.00	BL 8	0	0.00	0.00	0	0.00	0.00

TABLE 39: *Dental measurements, mandible: summary statistics*

Females	Left			Right				Left			Right		
	n	mean	s.d.	n	mean	s.d.		n	mean	s.d.	n	mean	s.d.
MD 1	5	5.58	0.43	4	5.88	0.30	BL 1	5	5.72	0.47	4	6.03	0.78
MD 2	7	6.10	0.46	7	6.23	0.37	BL 2	7	6.11	0.28	7	6.10	0.35
MD 3	10	6.90	0.58	13	7.02	0.32	BL 3	10	7.60	0.44	13	7.65	0.51
MD 4	18	7.09	0.29	20	7.21	0.40	BL 4	18	8.17	0.51	20	8.14	0.40
MD 5	21	7.41	0.55	23	7.45	0.54	BL 5	21	8.47	0.48	23	8.49	0.51
MD 6	17	11.58	0.65	20	11.78	0.56	BL 6	17	11.02	0.46	20	11.12	0.48
MD 7	17	10.94	0.62	22	10.85	0.65	BL 7	17	10.50	0.56	22	10.41	0.60
MD 8	13	11.21	0.87	13	11.09	0.85	BL 8	13	10.30	0.89	13	10.46	0.71
Males													
MD 1	10	5.97	0.31	8	5.70	0.48	BL 1	10	6.08	0.28	8	6.15	0.43
MD 2	13	6.33	0.44	11	6.05	0.48	BL 2	13	6.63	0.30	11	6.58	0.42
MD 3	15	7.23	0.44	15	7.17	0.42	BL 3	15	8.17	0.56	15	8.23	0.48
MD 4	17	7.47	0.42	19	7.40	0.46	BL 4	17	8.33	0.53	19	8.29	0.62
MD 5	19	7.32	0.41	18	7.34	0.42	BL 5	19	8.48	0.57	18	8.32	0.41
MD 6	13	11.79	0.58	12	11.83	0.51	BL 6	13	11.07	0.37	12	11.13	0.38
MD 7	19	10.91	0.56	20	10.95	0.54	BL 7	19	10.58	0.51	21	10.70	0.52
MD 8	16	10.95	0.60	18	11.18	0.61	BL 8	16	10.44	0.50	17	10.45	0.60
Children													
MD 1	7	5.71	0.29	7	5.67	0.41	BL 1	6	5.88	0.12	7	5.89	0.20
MD 2	8	6.11	0.59	6	6.25	0.45	BL 2	8	6.21	0.37	6	5.97	0.49
MD 3	6	6.77	0.55	4	7.15	0.30	BL 3	6	7.48	0.37	5	7.58	0.43
MD 4	6	6.82	0.42	6	7.00	0.37	BL 4	6	8.27	0.42	6	8.07	0.70
MD 5	5	7.22	0.24	6	7.15	0.50	BL 5	5	8.36	0.18	6	8.27	0.73
MD 6	8	11.18	0.55	8	10.90	0.40	BL 6	8	10.75	0.74	8	10.66	0.56
MD 7	6	10.18	0.55	6	10.25	0.30	BL 7	6	10.48	0.44	6	10.27	0.62
MD 8	0	0.00	0.00	1	10.10	0.00	BL 8	0	0.00	0.00	1	8.30	0.00

TABLE 40: *Individual long bone lengths (mm) and stature calculations (cm)*

	Humerus		Radius		Ulna		Femur		Tibia		Equation used	Stature estimate
	Left	Right	Left	Right	Left	Right	Left	Right	Left	Right		
Females												
4			222					397	331	332	F+T	151.0
13								416		360	F+T	157.7
15		282	222		244	244		386	331	329	F+T	149.2
18	251	255	214	215	237			381			F	147.6
19		280		218			399		335		F+T	151.8
25	275	275	223	221	245	244	407	408	342		F+T	153.9
26	297	299	242			258			361	360	T	158.7
27	280		227								H+R	152.5
31	292	296	236	237		268	413	407	344	341	F+T	154.4
35	292	292	236						359		T	158.3
36	284		227	227					350	350	T	155.6
39	277	279	212	213	237	237	391	394	316	315	F+T	148.1
40				220			411	416		340	F+T	154.5
45	301	301	246	244				428	379	377	F+T	161.9
47	304	309	237	239	259	258	432	432		375	F+T	162.0
56	258	263	201	202		217				301	T	141.1
58	300	303	245	238	257		432	429	357	361	F+T	159.6
60	286	290	232	234	246	247	414			342	F+T	154.8
61	290	289		238			257	413			F	155.9
64	291	289	238	237	260	266	417	411	361	362	F+T	157.6
73	277	278	219	219		241	381	380	333	330	F+T	148.7
77	302	303	236	239	253	260	417			353	F+T	156.8
79		292	233	235	253	254	412	408	348	348	F+T	155.1
83	277	277	218	218	233	233	392		340	341	F+T	151.5
87	285	283			242		407	407	344	342	F+T	154.0
94		281	222	221	245			410			F	155.1
102	288	288	216	218	236	236	395	395	336	331	F+T	151.0
107	281	284	228			247	409	406		349	F+T	154.9
109	295	291	236	236			401		349	350	F+T	154.1
110	273		215	215	233	232	396	398	330	327	F+T	150.6
112	307	307	237	238	256	255		441		374	F+T	163.2
113		281		226		249					H+R	152.5
122		288		226							H+R	153.6
140	275	279	227	228	252		410	409	346	346	F+T	154.8
142	290	294	225	224		248			340	341	T	152.8
154							430				F	160.3
Males												
9	297	294	240	242	264			407	350		F+T	157.2
22	310	310	253		274	272	432	430	363	362	F+T	162.4
23	319	318	267	269	288	286	456	454	389	390	F+T	169.6
24	290	273	231	233		255	398		335	335	F+T	153.8
28	280	279		230	251	253		407	343	347	F+T	156.5
29			244	246			439	440		364	F+T	163.8
30	302	302	241	241	266	263	414	411	354	355	F+T	158.6

TABLE 40: *Individual long bone lengths (mm) and stature calculations (cm) (cont.)*

	Humerus		Radius		Ulna		Femur		Tibia		Equation used	Stature estimate
	Left	Right	Left	Right	Left	Right	Left	Right	Left	Right		
Males												
38	307		241	238		263				345	T	158.1
42	325			259		282	447	447	375		F+T	166.4
43										345	T	158.1
44			232		257	256	410			352	F+T	157.9
57	320	323	255	262			451	448	379		F+T	167.3
67	299	297	252	255			420	412	372	370	F+T	161.4
72	313	316		266		288	440	441	387	390	F+T	167.4
74	330	328	273	275	294		457		380	383	F+T	168.8
76	290					260		400		342	F+T	155.1
86	315	315		259		277	437	438	368		F+T	164.1
90	325	320	261	260		287	459	457	384		F+T	169.3
91	320		255	260							H+R	167.9
92	307	310	245	249		274	420	419	353		F+T	159.4
93	308	309	261	262	280	281	440	443	381	383	F+T	166.6
100	300	300	240	242	263	260	431	424	350	350	F+T	160.1
103	305	311	249	249	263	269	430	428	360		F+T	161.7
115	310	307	254	257	279	279			374	378	T	166.6
120	313	312	245	245	265		437	437	356	358	F+T	162.4
129	280	280	237	238	250	250	403	400	337	335	F+T	154.4
131	291	292	230	233	247	250	409	406	325	327	F+T	153.8
132	290	294	257	259	280	279	435	433	371	363	F+T	163.4
147		340								395	T	171.9
152	320	316	246		264	265	438				F	163.9

Key: F: femur. T: tibia. H: humerus. R: radius

TABLE 41: *Adult long bone lengths - summary statistics*

		Females						Males					
		n	mean	s.d.	min.	max.	L vs R	n	mean	s.d.	min.	max.	L vs R
Humerus	Left	26	285.69	13.42	251.00	307.00	p=0.01	26	306.38	13.85	280.00	330.00	
	Right	29	287.17	12.21	255.00	309.00		23	306.35	16.18	273.00	340.00	ns
Radius	Left	28	227.57	10.86	201.00	246.00	ns	23	248.22	11.35	230.00	273.00	
	Right	27	226.89	10.53	202.00	244.00		24	251.21	12.39	230.00	275.00	p=0.01
Ulna	Left	17	246.35	9.15	233.00	260.00	ns	16	267.81	13.64	247.00	294.00	
	Right	20	247.55	12.61	217.00	268.00		21	269.00	12.61	250.00	288.00	p=0.01
Femur	Left	21	408.52	13.47	381.00	432.00	ns	22	431.95	17.48	398.00	459.00	
	Right	21	407.57	16.18	380.00	441.00		21	427.71	18.17	400.00	457.00	ns
Tibia	Left	21	344.38	13.96	316.00	379.00	ns	22	363.00	18.10	325.00	389.00	
	Right	26	345.27	17.88	301.00	377.00		21	360.43	20.01	327.00	395.00	ns

TABLE 42: *Adult limb proportions, summary statistics*

		Females						Males					
		n	mean	s.d.	min.	max.	L vs R	n	mean	s.d.	min.	max.	L vs R
Brachial index	Left	24	79.9	2.3	75.0	85.3	ns	21	81.3	2.8	76.9	88.6	p=0.02
	Right	24	79.4	2.2	75.7	84.3		20	82.7	2.5	78.5	88.1	
	L + R	18	79.5	2.4	75.4	84.8		17	82.2	2.6	78.4	88.4	
Crural index	Left	14	84.6	1.9	80.8	87.4	ns	19	84.2	2.2	79.5	88.6	ns (p=0.09)
	Right	18	84.8	2.2	80.0	88.1		14	84.8	2.5	80.5	89.8	
	L + R	10	84.3	2.0	80.4	87.3		11	84.7	2.8	80.0	89.2	
Inter-membral index	Left	11	69.4	1.4	66.4	70.8	ns	16	70.1	1.1	67.9	72.0	p=0.03
	Right	11	68.8	1.4	66.9	71.3		11	69.9	1.1	67.5	71.6	
	L + R	7	68.9	1.2	66.8	70.5		9	69.9	0.8	68.7	71.3	
Upper Limb (brachial):							Lower limb (crural):						
Mean (mean humerus*100/mean radius)							Mean (mean femur*100/mean tibia)						
Left/right (mean humerus L/R*100/mean radius L/R)							Left/right (mean femur L/R*100/mean tibia L/R)						
Intermembral: Mean (mean femur+mean tibia*100/mean humerus+mean radius)													

TABLE 43: *Adult femoral shaft circumference, mass estimates and stature:mass ratio*

	Femoral shaft circumference		Mass estimate	Stature: mass ratio
	Left	Right		
Females				
13		79.0	45.89	3.44
18	74.0	75.0	41.20	3.58
39	76.0	79.0	44.30	3.34
45	82.0	84.5	50.53	3.20
58	83.5	80.5	49.15	3.25
60		73.0	39.69	3.90
61	91.0		59.52	2.62
73	79.5	81.0	47.24	3.15
77	84.0	85.0	51.94	3.02
79	83.0	84.0	50.81	3.05
83	75.0		41.71	3.63
87	83.0	83.0	50.26	3.06
102	81.0	79.0	46.97	3.21
107	73.0	74.5	40.44	3.83
109	78.0		44.83	3.44
110	76.0	76.0	42.74	3.52
140	77.0	78.0	44.30	3.49
Males				
9	80.0	83.0	48.60	3.23
23	84.0	87.0	53.07	3.20
29	87.0		54.80	2.99
30	82.0	81.0	48.60	3.26
38		89.0	57.14	2.77
42		90.0	58.32	2.85

TABLE 43: *Adult femoral shaft circumference, mass estimates and stature:mass ratio (cont.)*

	Femoral shaft circumference		Mass estimate	Stature: mass ratio
	Left	Right		
Males				
57	92.0	90.0	59.52	2.66
67	88.0	89.0	56.55	2.79
72	90.0	89.0	57.73	2.90
74	94.0		63.18	2.56
86		90.0	58.32	2.89
90	88.0	89.0	56.55	2.74
93	91.0	90.0	58.92	2.87
100	87.0	88.0	55.38	3.03
103	81.0	81.0	48.05	3.32
120	83.0	82.0	49.70	3.22
129	82.0	80.0	48.05	3.37
131	84.0	84.0	51.37	3.24
132	88.5	86.0	55.09	2.95

TABLE 44: *Mass estimates: summary statistics*

		Females						Males					
		n	mean	s.d.	min.	max.	L vs R	n	mean	s.d.	min.	max.	L vs R
Femur midshaft circumference	Left	15	79.7	4.8	73.0	91.0	ns	16	86.3	4.2	80.0	94.0	ns
	Right	14	79.4	3.9	73.0	85.0		17	86.3	3.7	80.0	90.0	
Mass		17	46.6	5.1	39.7	59.5		19	54.7	4.5	48.1	63.2	
Stat.:mass ratio		17	3.3	0.3	2.6	3.9		19	3.0	0.2	2.6	3.4	

TABLE 45: *Additional postcranial measurements (mm): upper limb and body*

	Humerus head maximum diameter		Humerus midshaft maximum diameter		Humerus midshaft minimum diameter		Humerus midshaft circumference		Bi-iliac diameter
	Left	Right	Left	Right	Left	Right	Left	Right	
Females									
4	39.1	39.2	21.6	21.8	13.3	13.9			254.0
13	36.8								
15		40.4		21.7		17.5			
18	35.1	36.8	19.2	20.2	13.2	13.5	55.0	57.0	227.0
19				20.3		15.9			
25	36.9	37.0	19.9	19.7	15.8	15.9			
26	40.7	41.4	20.9	21.2	16.5	16.4	62.0	62.0	
27	39.5		21.1	20.8	14.2	14.0			
31	38.7	39.2	20.0	20.7	13.9	14.2			265.0
35	42.5	43.2	21.4	21.8	14.8	15.5			
36	40.7		21.7		15.1				235.0
39	41.7	41.4	20.2	20.2	14.0	13.9	60.0	58.0	
40	41.1		20.8	20.3	14.6	14.7			
45	42.9	43.1	23.4	23.1	15.7	15.6	66.5	67.0	256.0
47	41.8	42.0	20.3	20.2	15.0	15.3			261.0

TABLE 45: *Additional postcranial measurements (mm): upper limb and body (cont.)*

	Humerus head maximum diameter		Humerus midshaft maximum diameter		Humerus midshaft minimum diameter		Humerus midshaft circumference		Bi-iliac diameter
	Left	Right	Left	Right	Left	Right	Left	Right	
Females									
56	36.7	36.4	17.8	18.0	12.4	12.6	54.0	53.5	276.0
58	40.9	41.6	21.1	22.2	16.4	17.6	68.0	69.0	
60	39.5	39.6	19.4	20.6	14.7	15.4	58.5	61.0	
61	41.7	43.0	23.7	23.4	16.5	15.8	73.0	68.0	241.0
64	38.1	38.2	20.2	19.9	14.5	14.6			
73	39.8	39.4	22.1	21.7	14.7	16.4	64.0	66.0	
77	43.0	43.4	19.9	20.4	15.4	15.4	60.0	61.0	266.0
79	39.9	41.3	21.5	21.0	15.3	15.0	63.0	61.0	237.0
83	37.8	38.3	20.4	20.0	14.6	14.8	59.0	60.0	
87	40.0	39.9	22.6	21.5	15.4	15.3	65.0	64.0	
94				20.9		15.2		61.0	
102	38.7	38.1	19.8	20.3	14.3	14.8	58.0	58.0	
107	39.4	40.0	19.6	19.4	14.3	14.4	58.5	59.0	
109	40.7	41.8							
110	38.7		20.0	21.3	13.3	13.1	59.0	59.0	242.0
112	42.9	42.5	22.6	22.0	14.8	16.4	63.0	62.0	
113			21.7	22.3	15.3	15.3	68.5	64.0	
122	39.1		20.2	20.2	12.6	12.4	57.0	57.0	
140	38.9	38.9	20.3	19.8	14.0	14.0	60.0	59.0	
142	39.5	39.4	20.7	20.7	15.1	14.7	60.0	60.0	
154									
Males									
9	41.9		21.9	22.2	15.7	17.0			261.0
22	45.0	44.1	21.8	22.4	17.3	18.3	67.0	69.0	259.0
23		43.7	24.9	24.9	17.7	18.2	74.0	75.0	
24	41.8	41.8	22.4		17.4		70.0		
28	42.1	39.6	23.2	24.0	15.7	15.4	68.0	69.0	
29	44.9								250.0
30	45.4	46.1	22.5	22.7	16.9	16.7	67.5	68.0	
38	46.5		22.7	24.2	16.0	17.1	68.0	71.0	
42	46.1			24.6		17.8		74.0	
44									
57	47.8	47.2	25.0	24.7	17.1	16.5	73.0	73.0	
67	44.7	44.9	23.7	24.0	18.0	18.9	70.5		287.0
72	46.8	46.9	22.7	23.3	19.0	19.8	69.5	72.0	250.0
74	48.5	48.5	25.4	25.9	20.4	20.2	77.0	77.0	274.0
76	43.8		24.9		18.6		73.0		
86	45.4	46.8	23.1	23.9	20.1	20.1	74.0	75.0	
90	47.9	49.4	23.8	25.2	19.0	19.2	71.0	72.0	
91	50.1		25.0		18.5		79.0		
93	46.3	45.9	24.9	24.6	19.1	17.8	76.0	77.5	
100	44.2	45.1	21.6	22.8	17.6	18.4	61.0	68.5	263.0
103	45.3	46.2	21.4	22.2	16.7	15.9	64.0	65.0	
115	48.0	48.1	23.6	24.9	18.3	17.8	74.0	72.0	
120		45.2	23.8	23.9	16.8	16.5	69.0	70.0	

TABLE 45: *Additional postcranial measurements (mm): upper limb and body (cont.)*

Males	Humerus head maximum diameter		Humerus midshaft maximum diameter		Humerus midshaft minimum diameter		Humerus midshaft circumference		Bi-iliac diameter
	Left	Right	Left	Right	Left	Right	Left	Right	
129	42.2	43.7	23.8	25.0	16.3	15.8	67.0	69.0	
131	45.9	45.2	24.1	24.6	17.5	17.5	70.0	72.0	
132	44.6		26.0		17.0		73.0		
147				20.0		15.6		61.0	
152	45.8	47.3	20.9		15.7		61.5		

TABLE 46: *Additional postcranial measurements (mm): lower limb*

	Femur oblique length		Femur midshaft medio-lateral diameter		Femur midshaft antero-posterior diameter		Tibia medio-lateral diameter at nutrient foramen		Tibia antero-post. diameter at nutrient foramen	
	Left	Right	Left	Right	Left	Right	Left	Right	Left	Right
Females										
4		392.0		23.7	23.5	23.3	20.9	21.3	27.9	27.9
13		416.0		24.6		24.0	20.5	20.6	31.4	32.9
15		380.0		24.4		26.2	22.0	23.8	29.4	30.7
18		380.0	23.4	22.7	23.3	24.7				
19	390.0		23.6	23.0	23.2	23.7	22.9		30.6	
25	405.0	408.0	22.8	22.8	25.2	25.1	23.3	23.3	27.4	27.9
26							21.9	21.3	32.2	31.6
27								19.7		31.1
31	410.0	405.0	25.2	24.9	23.6	24.2	21.3	21.7	26.7	27.3
35							22.2	22.3	30.6	30.8
36							23.5	22.5	31.1	29.6
39	390.0	391.0	24.2	23.9	22.8	21.7	20.3	20.7	27.8	27.6
40	410.0	413.0	25.8	24.5	23.4	24.7	21.6	22.5	27.3	30.5
45	427.0	425.0	24.2	25.0	27.2	28.2	24.9	24.2	33.2	34.2
47	427.0	424.0	24.0	23.0	27.4	29.1	23.1	23.0	29.6	29.8
56								18.6		27.4
58	426.0	425.0	25.0	25.0	27.3	28.9	20.9	20.5	33.7	33.2
60		405.0		21.6		24.7	19.2	20.1	32.1	32.0
61	412.0		26.9		30.3		21.9	22.4	32.3	32.4
64	414.0	408.0	25.6	25.2	24.7	24.8	20.2	18.9	30.4	29.5
73	377.0	376.0	24.5	25.2	25.5	25.7	23.4	23.9	27.1	27.8
77	412.0		25.1	26.0	26.6	26.6	23.2	22.6	32.9	30.6
79	408.0	404.0	24.7	24.4	27.9	28.2	22.3	21.4	31.2	30.8
83	389.0		22.5		24.7		21.4	21.3	28.9	28.8
87	404.0	401.0	26.5	26.6	25.7	26.0	19.0	19.5	30.9	30.3
94							20.4	19.4	28.0	29.3
102	394.0	394.0	25.7	25.1	24.5	23.8	19.9	19.4	28.1	28.0
107	404.0	400.0	22.3	22.3	22.3	23.3	21.8	20.5	28.2	27.5
109	397.0		23.5		25.2		20.1	22.1	30.2	29.5
110	392.0	391.0	22.1	23.6	24.7	24.9	21.0	20.0	29.1	29.7

TABLE 46: *Additional postcranial measurements (mm): lower limb (cont.)*

	Femur oblique length		Femur midshaft medio-lateral diameter		Femur midshaft antero-posterior diameter		Tibia medio-lateral diameter at nutrient foramen		Tibia antero-post. diameter at nutrient foramen	
	Left	Right	Left	Right	Left	Right	Left	Right	Left	Right
Females										
112							23.0		33.1	
113								22.6		32.4
122							22.3	20.4	28.0	27.5
140		403.0	23.6	23.5	24.6	24.9	19.7	19.2	30.3	29.7
142							21.2	21.7	29.9	32.1
154										
Males										
9		405.0	23.9	24.6	26.3	27.1	23.1	22.1	34.8	33.2
22	430.0	427.0	26.9	25.0	27.6	28.8	24.3	24.1	33.2	34.0
23	452.0	450.0	26.9	26.4	29.0	28.2	25.2	24.7	36.3	34.9
24	395.0		25.9	25.7	27.7	27.9	24.1	24.1	35.4	34.6
28		403.0	25.3	25.8	25.9	26.3	25.3	24.8	32.4	
29	435.0		24.5		29.6		22.3	23.9	34.5	36.2
30	410.0	408.0	25.2	24.5	25.9	25.7	22.2	22.0	31.1	31.6
38		422.0		26.4		28.6	23.7	23.2	34.4	33.0
42		441.0		27.5		29.7	27.0	26.0	33.9	33.4
43										
44										
57	449.0	445.0	27.2	25.5	30.6	29.9	25.1	26.5	35.9	35.3
67	416.0	410.0	26.6	26.4	28.2	29.6	24.5	24.4	34.2	34.1
72	437.0	437.0	27.4	27.5	29.5	29.8	24.2	23.6	35.7	34.9
74	451.0		26.3		32.6		25.8	25.8	37.8	36.6
76							23.1	23.3	35.5	33.2
86		436.0		25.7		30.3		24.9		36.6
90	454.0	452.0	26.3	26.0	28.2	29.6	23.5	22.1	34.3	35.0
91										
92										
93	439.0	440.0	27.5	26.6	30.6	30.9	22.1	22.6	33.4	32.0
100	425.0	421.0	26.0	26.4	28.7	28.8	25.0	25.1	31.8	32.6
103	425.0	424.0	25.2	24.9	25.7	26.2	18.5	19.8	30.0	30.4
115							20.7	20.4	35.6	33.7
120	431.0	432.0	25.3	24.3	26.8	27.4	22.7	23.9	34.1	33.3
129	399.0	393.0	25.7	25.0	26.9	26.0		24.6		34.2
131	404.0	401.0	26.2	26.3	26.2	27.0	25.3	24.6	34.7	32.9
132	430.0	429.0	26.1	25.1	28.9	28.4	21.3	21.2	31.9	31.2
147							21.0	20.8	35.7	35.6
152	435.0							20.5		32.3

TABLE 47: *Adult postcranial measurements, summary statistics*

		Females					L vs R p value	Males					L vs R p value
		n	mean	s.d.	min.	max.		n	mean	s.d.	min.	max.	
Humerus head	Left	31	39.8	2.0	35.1	43.0	0.01	24	45.5	2.2	41.8	50.1	ns
	Right	26	40.2	2.1	36.4	43.4		19	45.6	2.3	39.6	49.4	
Humerus	Left	30	14.7	1.0	12.4	16.5	0.04	24	17.6	1.3	15.7	20.4	ns
midshaft min.	Right	32	15.0	1.2	12.4	17.6		21	17.6	1.5	15.4	20.2	
Humerus	Left	30	20.8	1.3	17.8	23.7	ns	24	23.5	1.4	20.9	26.0	0.0001
midshaft max.	Right	32	20.9	1.1	18.0	23.4		21	23.8	1.4	20.0	25.9	
Humerus	Left	21	61.5	4.7	54.0	73.0	ns	23	70.3	4.6	61.0	79.0	0.006
midshaft circum.	Right	22	61.2	3.9	53.5	69.0		19	71.1	4.0	61.0	77.5	
Bi-iliac diameter		11	250.9	15.5	227.0	276.0	-	7	263.4	13.3	250.0	287.0	-
Femur oblique	Left	19	404.6	13.9	377.0	427.0	0.04	18	428.7	18.0	395.0	454.0	0.002
length	Right	20	402.1	14.5	376.0	425.0		19	425.1	17.6	393.0	452.0	
Femur midshaft	Left	21	24.3	1.4	22.1	26.9	ns	19	26.0	1.0	23.9	27.5	0.02
medio-lateral	Right	22	24.1	1.2	21.6	26.6		20	25.8	0.9	24.3	27.5	
Femur midshaft	Left	22	25.2	2.0	22.3	30.3	0.02	19	28.2	1.9	25.7	32.6	ns
antero-posterior	Right	22	25.3	1.9	21.7	29.1		20	28.3	1.6	25.7	30.9	
Femur midshaft	Left	15	79.7	4.8	73.0	91.0	ns	17	86.6	4.2	80.0	94.0	ns
circumference	Right	14	79.4	3.9	73.0	85.0		16	86.1	3.7	80.0	90.0	
Tibia medio-lat.	Left	31	21.6	1.4	19.0	24.9	ns	23	23.5	2.0	18.5	27.0	ns
at nutrient foram	Right	32	21.3	1.5	18.6	24.2		26	23.4	1.8	19.8	26.5	
Tibia ant.-post.	Left	31	30.0	2.0	26.7	33.7	ns	23	34.2	1.8	30.0	37.8	0.02
at nutrient foram	Right	32	30.0	1.9	27.3	34.2		25	33.8	1.6	30.4	36.6	

IV. SKELETAL AND DENTAL EVIDENCE FOR HEALTH

HEALTH is here defined in a wider sense than is indicated by the term 'palaeopathology' which, if strictly interpreted, implies the study of individual skeletal pathology, and possibly the drawing of inferences about disease aetiology and prevalence in the population. The possibility of extracting a more inclusive picture of the health of prehistoric peoples from their skeletal remains than a simple description of, and extrapolation from, those ailments which affect the skeleton has been increasingly recognised (eg Cohen and Armelagos 1984, Cohen 1989, Wood *et al.* 1992). The integration of such non-specific evidence of health as infant and child mortality, skeletal and dental evidence of growth disruption, achievement of growth potential, adequacy or inadequacy of bone mass, and adult longevity, while potentially reflecting disease load in a population, widens the picture beyond that of specific disease. In this expansion in the depiction of the health of prehistoric people, the emphasis of the interpretation is on community health, which can be a positive as well as a negative condition. The definition of health as used here includes good as well as ill health.

The skeletons are the primary source of data, but this study extends beyond the morphological response to genetics and environment to consider the implications of demography and epidemiology. The 'biocultural' approach taken involves the integration of non-skeletal evidence of environment, social structure and physical activities with the skeletal evidence. The model on which this study is based is set out in figure 38.

THE EVIDENCE OF HEALTH FROM SKELETONS

The approach taken in determining health from skeletal remains is very different from that of a clinician or even a pathologist conducting a postmortem. In comparison with the soft tissues, the reactions of the skeleton and dentition to both external and internal factors are constrained by the nature of their mineral component. As a dynamic part of the living organism, bone is continuously undergoing remodelling in the process of maintaining mineral density and homeostasis, and in reacting to external influences such as infection and biomechanical stress. Specialist cells, osteoblasts and osteoclasts, are responsible for the formation and resorption of the bony structure. This process is usually in balance. Skeletal reaction to disease is limited to variations in the rate of interaction of these bone cells. This may be through alteration in the rate of bone formation or resorption, or a combination of both processes. In infancy and childhood, there is relative flexibility in the structure because of the modelling the bones are undergoing during growth. Bony reactions can significantly alter skeletal morphology. Once growth ceases, the opportunities for bony reaction are comparatively limited as the rate of cellular activity is reduced. Nevertheless, there can be bony response to external or internal influences as remodelling continues throughout life. Teeth are not subject to the same process of renewal during life, except where previously demineralized enamel may be remineralized in the early stages of this process. The reaction of the dentition to environmental or

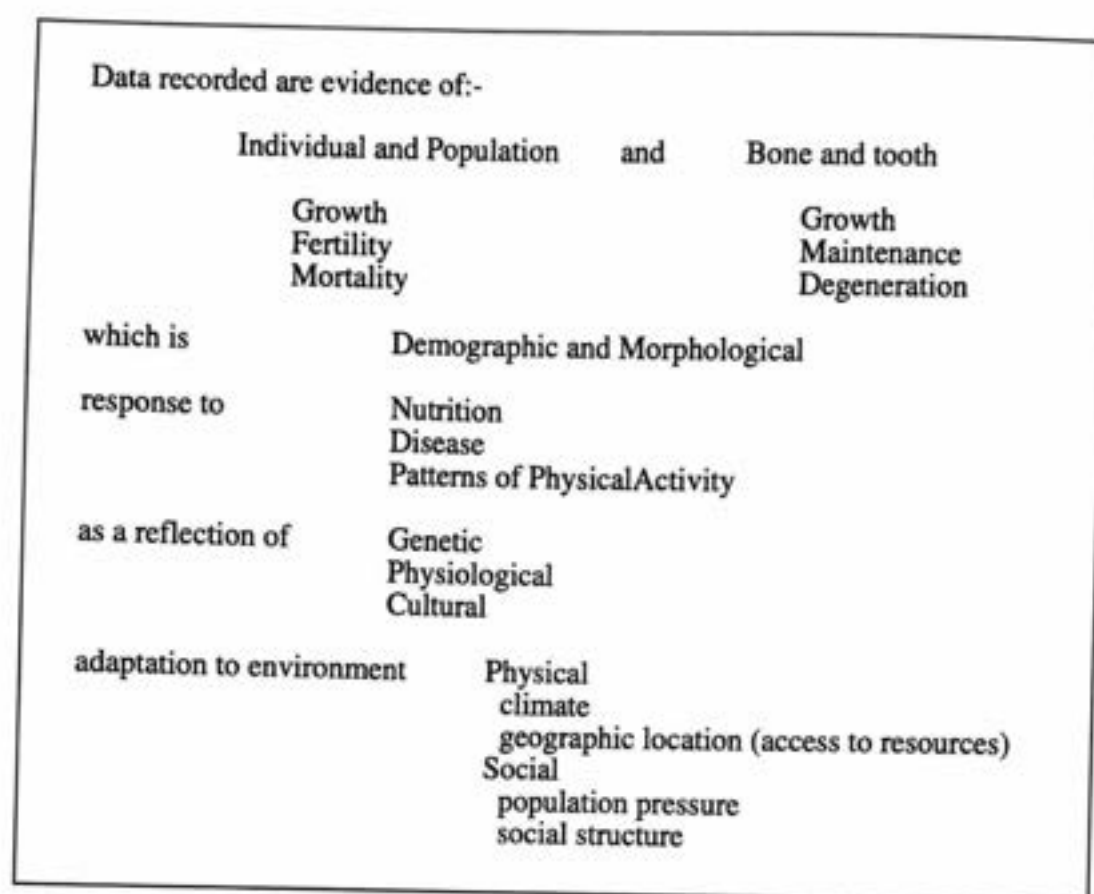


FIG. 38. Model for this study of health and disease in prehistoric humans

mechanical stress is therefore limited to alteration in the quality or shape of the tooth during growth, or to destruction of the structure once it is fully formed.

Because of these limitations, most of the skeletal characteristics indicative of health cannot be interpreted as responses to specific factors. Responses to inadequate nutrition cannot necessarily be differentiated from a systemic response to illness. Indeed, the synergistic interaction of malnutrition and disease means that the two conditions will often produce the same skeletal response or ameliorate or exacerbate an existing reaction. The aetiology of skeletal responses indicative of health can often only be inferred from the patterns of distribution within the population or from evidence of external factors such as diet or environment.

Some diseases do produce specific, identifiable, skeletal pathology. These form the basis for sections on palaeopathology in many skeletal reports, but they cannot be presumed to provide an approximation to the reality of health of the skeletal population. It is possible that the condition was not a significant factor in the health of the individuals concerned or the population as a whole.

In many diseases, there will only be a skeletal response when the condition becomes chronic. The presence of such pathology in a skeletal collection may be indicative of high levels of morbidity in the population, but it is also possible that survival of the affected individuals indicates better health than those with the same disorder who died in the acute stage of the disease. The absence of skeletal evidence of ill health offers the possibility that death was in response to an acute condition, whereas '...evidence of skeletal disease indicates a sufficiently adequate immune response to insure survival to the chronic stage.' (Ortner and Putschar 1981:44). This gives rise to the 'Osteological Paradox' (Wood *et al.* 1992), whereby skeletons with pathological lesions may represent individuals with better health than those who succumb to a potentially fatal illness. The interpretation of skeletal pathology is far from straightforward. The one response to ill health which is universal is the ultimate response of death. Although cause of death is rarely obvious, age at death is an important non-specific indicator of health, particularly in populations where most, if not all, individuals die at an age which is decades younger than the maximum potential human life expectancy of approximately 100 years (Acsádi and Nemeskéri 1970).

Beyond these considerations of the extent to which health is reflected in the skeleton, is the fact that the skeletal sample cannot be assumed to be representative of the living population contributing to the cemetery. To the extent that bones and teeth reflect health, each skeleton is evidence of the health of that individual at the moment of death. Some characteristics, particularly those in adults which reflect their health in childhood, are indicative of the health of the living population. Beyond this, with the exception of fatal accidents, the individuals represented by the skeletons have suffered fatally from disease. The evidence from their skeletal remains is, therefore, potentially biased in that it could show poorer health than that of the living population. This is particularly important in considering the infants and children, where the impact of physiological disruption on the individual is greater and the bias therefore potentially stronger than with adults (Cook and Buikstra 1979, Cook 1981).

Although the representation of health obtained from the skeletons does not equate with that of the living population, the data nevertheless provide sufficient detail to serve as the basis for comparing individuals and groups within the sample. Some types of data can also be compared with other sites, although factors such as the age dependency of many indicators and the question of comparability of age estimations between observers, and the extent of the genetic component in controlling many aspects of development, maintenance and degeneration, have to be considered in any intersite comparison.

Identification and recording

The methods available for identifying and describing bony reaction involve gross and radiographic observation, and microscopic analysis of thin sections. The information obtained from all these methods must be considered in concert, as each will provide a different perspective on the organic response of the skeleton. As the author did not participate in the excavation in 1985, none of the data presented was collected in the field. The lack of opportunity to examine the skeletons *in situ* was not as disadvantageous as would very often be the case with excavations of burials, as the excellent preservation of the material and the lack of intercutting of graves, together with the

exemplary skill of the excavators, resulted in almost complete recovery of individual skeletons. I believe that no vital information was lost.

In this chapter and the following, skeletal and dental evidence of health has been subdivided into two categories:

1. Chapter IV - Health. In this context 'health' is defined in a more restricted sense than that used in the overall objective of this study, as it is confined to those characteristics which are indicative of the general state of health rather than any specific condition. They may be either positive or negative. The evidence is presented in two sections, covering the period of growth (health during childhood) and maintenance (health during adulthood). This includes:

(a) Health during childhood:

- Growth - the achievement of growth potential and sexual maturation.
- Growth disruption - skeletal and dental responses to disruption of growth.
- Infant and child mortality.

(b) Health during adulthood:

- Maintenance of bone mass, fertility, and adult longevity.

2. Chapters V and VI - Disease. The negative evidence of health in the form of skeletal and dental pathology.

All evidence for each individual will be considered in the ultimate assessment of that person's health, as the skeleton represents a biological organism and many responses to environmental stress will be systemic. The identification of the aetiology of any particular reaction must be made in the light of all evidence available. Few skeletal reactions can be shown to be the consequence of isolated or discrete episodes of environmental stress or of accidents. The evidence is then considered for the population as a whole, to provide a representation of the health of the community.

HEALTH DURING CHILDHOOD

Growth

A primary source of evidence of childhood health is growth and growth disruption:

In human ecosystems, the impact of the environment is felt most forcibly on the growing child, particularly in the early years of life. (Malcolm 1974:329)

A child's growth rate reflects, better than any other single index, his state of health and nutrition.....a well designed growth study is a powerful tool with which to monitor the health of a population, or to pinpoint subgroups of a population whose share in economic and social benefits is less than it might be. (Eveleth and Tanner 1976:1)

These statements refer to living populations, where health studies can cover multiple indices of growth such as height, weight, skeletal and sexual maturity. Long bone lengths from children in a skeletal collection are not as useful as an indicator of growth rates for a number of reasons. Growth is a dynamic process which is ideally recorded from a longitudinal rather than cross-sectional sample of a group. The children in a skeletal collection not only represent a cross-sectional sample, but are further biased in that they are not necessarily representative of the living population. In addition, the genetic input in population differences in growth rates and body proportions (Eveleth and Tanner 1976) limits the use for comparison of available modern standards of diaphyseal length to genetically similar skeletal populations. There is also the complication of sexual dimorphism in older children. Where there has been no estimate of sex of these individuals, variations in growth rates as a result of other genetic or environmental factors cannot be distinguished from sex differences. Some estimate can be made of the relative growth rate of infants and young children within a population through a comparison of diaphyseal length and cortical thickness with dental age, where diaphyseal length has not been used in estimating age at death (Cook 1979, Huss-Ashmore 1981, Cook 1984, Goodman *et al.* 1984). The relative growth rate and timing of growth spurts can be compared with modern standards (Huss-Ashmore 1981). This requires a sample of different ages to form a basis for statistical comparison (Goodman *et al.* 1984) and the numbers available from this site are inadequate for this purpose.

Diaphyseal lengths of children from Khok Phanom Di aged from dental calcification are used as a general indication of relative growth rates. The lengths of the diaphyses of the long bones of children between the ages of five to fourteen years are listed in table 15. Skeletal growth in two children (burials 1, 12.1) was retarded compared with others of the same age. Both were buried in MP7. Burial 20 was comparatively tall. The only comparative prehistoric Thai data on diaphyseal length of long bones is from a single individual from Ban Na Di. This is from a seven-year-old child, with a femoral diaphyseal length of 268 mm. This is comparable with children of the same age from Khok Phanom Di.

Adult stature

Adult stature provides evidence of the achievement of growth potential which has the advantage over other measures of growth of not being age dependent. It is representative of the proportion of the population who were healthy enough to survive the vulnerable period of childhood. Stature is one of the metric parameters which has traditionally been calculated from skeletal groups (Wells 1970), but recognition of its use as an indicator of juvenile health in archaeological populations is relatively recent (Huss-Ashmore *et al.* 1982). It has been used to identify nutritional and social variations over time and within populations (Haviland 1967, Nickens 1976, Larson 1982, Cook 1984).

As with diaphyseal length in children, the comparison of adult stature is valid between genetically similar populations where the potential for growth in height is similar (Goodman *et al.* 1984). The effects of sex differences can be eliminated through the use of sex-specific stature calculation equations. The achievement of the genetic potential for height is strongly mediated by environmental influences during the growth period. This has been demonstrated by recent secular increases in height in a number of diverse populations (Tanner 1962, Eveleth and Tanner 1976, Wurm 1984),

through increases in mean heights of migrants compared with non-migrants from the same populations (Hulse 1968, cited in Cavalli-Sforza and Bodmer 1971), and in comparisons between urban and rural samples of closely related populations (Andrews 1943). In each of these circumstances, improved nutrition and living conditions have been cited as reasons for the increase in mean stature (van Weiringen 1978).

The preferential maintenance of growth rates in external bone dimensions at the expense of cortical thickness during periods of malnutrition or other environmental stress in children has been shown (Garn *et al.* 1964b, Himes *et al.* 1975). Low mean statures in a group therefore reflect severe or chronic problems, although there is some suggestion that the age at which environmental stress is suffered can be critical in determining the duration of the effects of slowing or cessation of bone growth. Interference in growth rates during infancy has been cited as having a more permanent effect than problems later in childhood when catch-up growth occurs at the time of recovery (Prader *et al.* 1963, Briers *et al.* 1975, Steegman 1985), although Alleyne *et al.* (1977) suggest that this 'critical period' concept in humans is not as clear cut as has been suggested from studies on animals. The slowing of skeletal growth and skeletal maturation during the adolescent growth spurt does not necessarily allow sufficient time for 'catch-up' before growth ceases (Frisancho *et al.* 1970, Malcolm 1979).

The method used in calculating adult stature is given in chapter III and the results of the individual calculations are listed in table 40. Summary statistics and comparative data are provided and discussed in chapter III. As the biological relationships among the available samples of prehistoric Thai and between prehistoric and modern groups are unknown, genetic variation cannot be discounted in assessing the possible reasons for differences in stature among these groups. However, in the absence of evidence to the contrary, the relatively short mean stature of the males at Khok Phanom Di compared with their prehistoric counterparts suggests that they suffered sufficient ill health in childhood to have disrupted growth to the extent that they failed to achieve genetic potential. This suggestion is reinforced by the comparison with the more heterogeneous samples of modern Thai. The females at Khok Phanom Di either did not suffer to the same extent, or were able to recover more quickly.

Sexual dimorphism

The degree of dimorphism in parameters which measure growth in a population may be an indicator of the differential susceptibility of males and females to environmental stress, as it has been suggested that males are less buffered (Tanner 1962, Wolanski and Kasprzak 1976, Stini 1985), although Stinson (1985) questions the extent to which this has been proved. Stini (1985) suggests that the greater variability in male morphology and the greater requirements in energy and protein to sustain their bulk exposes them to more risk, whereas females are less morphologically variable and less susceptible to environmental stress because of the need to survive pregnancy and lactation. Therefore, in groups where children are subject to environmental stress the degree of dimorphism will be reduced, as males fail to fulfil their growth potential while females deviate less from the genetically determined levels. Some studies have shown that males are more affected by factors such as malnutrition than females and that catch-up growth after a period of growth disruption is more rapid in females (Greulich 1951, Stini 1969, Tanner 1962, Tanner 1978, Malina *et al.* 1985).

Despite this, there are potential complications of socio-cultural factors such as sex differences in physical activity patterns in childhood and the protection of one sex (generally males) from environmental stress (Goodman *et al.* 1984). From an extensive study of sexual dimorphism in growth and development, Stinson concludes:

Given the complex cultural environment in which humans live, the hypothesis has little predictive value on a general level.....Explanations based on the idea that populations experiencing nutritional stress should have lower levels of sexual dimorphism do not seem to adequately explain the existing variation, probably because sexual dimorphism is influenced by a number of cultural practices as well as by genetic factors. (Stinson 1985:142)

Another note of caution in regard to the genetic component of sexual dimorphism, from Stini:

The magnitude of sexual dimorphism in humans should not be overstated. As in so many other biological properties, the within-sex variation for such continuous traits as weight, stature, fat mass, lean body mass, is sufficiently great to produce a substantial zone of overlap of the ranges of values for the sexes. (Stini 1985:219)

Nevertheless, an analysis of dimorphism in population is warranted, as the extent of sex differences may offer explanations for, or be explained by, interpretations of non-skeletal data from the site.

Modern studies on living individuals include such indicators as sex differences in mortality and morbidity, and both pre- and postnatal growth, but these require the sex of children to be known. Stature is the most commonly used measure of dimorphism in skeletal analyses (e.g. Cook 1984, Rathbun 1984). Sex differences in the timing and intensity of the adolescent growth spurt are chiefly responsible for dimorphism in stature (Tanner 1962, Stini 1985). The later and longer growth spurt in males allows greater growth in both pre-adolescent and adolescent periods and differences in body proportions, with males having relatively longer limbs than females (Tanner 1962, Marshall 1978, Stini 1985). However, Eveleth (1975), from a large study (n=264 populations, each of 50–200 individuals), concluded that sexual dimorphism in stature is too strongly genetically controlled for comparison between genetically different populations to be a valid indication of environmental effects. As with the use of stature itself as a comparative growth parameter, sexual dimorphism in stature is most valid as an indicator of intrapopulation variation. In addition to changes in the degree of dimorphism, the variation in both male and female stature needs to be considered. The degree of dimorphism can vary with changes in the mean stature for either sex, which reflects the complexity of the factors involved. Larson (1982, 1984) for example, has suggested that a relatively greater decrease in female stature during the transition to agriculture in a Georgian population, producing increasing dimorphism over time, has reflected an increase in environmental stress on females through an alteration in the sexual division of labour rather than any change affecting the entire population. The low sexual dimorphism in stature at Khok Phanom Di, described in chapter III, may therefore reflect increased sensitivity of males to environmental stress during infancy and childhood. Greater bilateral asymmetry in body proportions in males, also described in chapter

III, is possibly also attributable to environmental stress, although sexual dimorphism in dimensions which reflect local remodelling in response to biomechanical factors may indicate that all sexual dimorphism in morphometrics is attributable to sexual division of labour. Further investigation is required before the explanation can be offered.

Sexual maturation

A source of evidence of growth rates in respect to sexual maturation which is widely recorded on living populations is the onset of menarche in adolescent females. The evidence that the onset of menarche is environmentally as well as genetically controlled is similar to that of skeletal growth. Frisch (1978) hypothesised a relationship between nutrition, as evidenced by body weight, and menarche, and stimulated a continuing discussion on the question. The evidence, both direct and indirect, is that malnutrition delays menarche (Bongaarts and Potter 1983). There is considerable evidence for reduction of menarcheal age in industrial countries, with a mean reduction of 0.3 years each decade over the period 1840-1970 (Tanner 1962, Eveleth and Tanner 1976) which has been attributed to environmental, primarily nutritional, improvement. An earlier age at menarche has been recorded in higher than in lower socio-economic classes in other countries, and in urban rather than rural groups (Eveleth and Tanner 1976). There is also evidence for basic genetic control, although the over-riding environmental influences make the extent of genetic input difficult to quantify (Garn and Bailey 1978, Marshall 1978). However, apart from the biological possibility of conception, there are so many cultural, social and other biological factors involved in the age of first pregnancy that, unless the individuals involved are relatively young compared with the age that could be expected on the basis of modern data, the evidence of health from this source is not necessarily useful.

The skeletal evidence of menarche is the indirect evidence of parity from the pelvis. This gives some evidence of the maximum age of onset of ovulation, and the deduction of an allowance for the period of adolescent subfecundity gives a very approximate indication of the maximum age of the onset of menarche. There are many socio-cultural factors which may influence the correlation of these events.

The length of the period of adolescent subfecundity is variable and is the result of the irregularity and proportion of anovulatory cycles in the years immediately after menarche. Evidence available from the !Kung hunter-gatherers is that the proportion of anovulatory cycles is sufficiently high to result in an average period of four years from the age of the concurrent events of menarche and marriage until the birth of the first child (Kolata 1974). May (1978) suggests that the length of the period is 'roughly independent' of the age at menarche. An allowance of 3-4 years for this period in the Khok Phanom Di population seems reasonable.

The basis of the identification of parturition from the pelvis is discussed in the section on fertility. There are eight females aged less than 25 years at death, and all had pelves sufficiently intact for assessments to be made of pregnancy markings at both the dorsal pubic bones and the preauricular grooves of the ilia. Table 16, which lists the grades of pitting for all females, provides the data. The youngest female, B140, aged 17 years at death, has no pitting at the dorsal pubis and no preauricular groove. The pitting on the two individuals aged 19 years at death (burials 25, 60), after allowance for the period of adolescent subfecundity, places the age of menarche at 15-16 years.

Mean age at menarche in prehistoric New Zealand Polynesians has been estimated to be c. 16 years, and the age at first conception c. 19 years (Visser 1986). No other estimates for prehistoric groups have been located, although Houghton and Wiriayaromp (1984), on the basis of the presence of evidence of pregnancy on the pelvis, suggest that a 17-year-old at Ban Na Di may have reached menarche at 15 years.

There is a wide range of data available on the age at menarche in living populations. In the nineteenth century menarche occurred at about 15 years in England and 17 years in Norway and Finland (Eveleth and Tanner 1976). The secular reduction in age at menarche in 'developed' societies since the mid nineteenth century has markedly reduced this mean age. Eveleth and Tanner (1976:216) list mean ages at menarche of numerous populations throughout the world from data published during the 1950s, 60s and 70s. In populations from Asia, the mean ages vary from 12.5 years in affluent Hong Kong to 13.3 years in lower class Hong Kong. Within this range are samples from Burma (13.2 years), Singapore (12.7–13.0 years) and urban Japanese (12.9 years). In most European populations the mean age is 13.0 years. In more traditional societies, the age at menarche remains higher. In rural Indians and Sri Lankans it is closer to 14.5 (± 0.13 – 1.73) years (Wilson and Sutherland 1953, Eveleth and Tanner 1976). In tribal societies in Africa (Tanzania, Rwanda and Nubia) it ranges from 15 to 17 (± 0.16 – 0.30) years and in New Guinea from 15.5 to 18.4 (± 0.29 – 0.35) years (Eveleth and Tanner 1976). In the Dobe !Kung hunter-gatherers in the Kalahari Desert, it is c. 16.5 years (Howell 1979).

Stahlie (1960) reported that the average age of the first child in modern Thai women was 22 years 4 months, and that less than 5 per cent of births were to women aged less than 19 years. Births to 15 and 16 year olds were very rare. Stahlie does not discuss cultural or social influences on the age of first parturition, but it is not necessarily a reflection of physiological ability to conceive.

In comparison with the age at menarche in traditional societies, the estimated minimum age of c. 15–16 years in Khok Phanom Di females is at the younger end of the range.

Growth disruption

Skeletal evidence of acute or chronic physiological disruption of growth is a non-specific indicator of health which may reflect a systemic reaction to nutritional or specific pathological processes. The extent to which the skeleton responds to these problems is not necessarily the same for all individuals. There is some genetic input to the response and it can be subject to existing health, including lowered resistance through repeated or chronic illness. The degree of genetic resistance or vulnerability cannot be assessed, except in cases of inherited illness which are known to interfere with the immune system. Disruption of growth may be evident on the long bones in the form of 'Harris lines' and on the dentition in the form of enamel defects.

Skeletal - Harris lines

These are seen on radiographs as lines or bands extending across the medullary cavity of long bones. The term 'Harris' lines (Harris was one of the early researchers in the field (1926, 1931, 1933)) is used here for brevity, because it is commonly used and recognised and because it does not carry any implications as to formation processes or appearance, as do some of the other names used such

as 'lines of arrested growth', or 'transverse lines'. Their description as lines is a reflection of their radiographic appearance, rather than their actual form. On sectioned bone, they can be seen to vary from single strands of bone to lattices or plates. As they parallel the growing ends of bones, and in some bones these surfaces form different angles at different stages of development, the lines are not necessarily at right angles to the long axis of the bone but may appear, for example, as oblique chevrons, as in the distal femur and humerus (Garn *et al.* 1968).

Two factors are involved in the formation of Harris lines, an adverse event which has sufficient physiological effect on the individual to interfere with bone growth, and recovery and resumption of growth. The bony response initially involves the cartilagenous epiphyseal plate. Growth ceases, the plate becomes thin, loses its normal structure and ceases to provide the template for normal osteoblastic activity. The osteoblasts continue production of bone at a relatively low rate, forming a transversely-oriented structure beneath the epiphyseal plate. During the recovery period, resumption of normal osteoblastic activity coupled with a four to nine day lag in the maturation of the cartilage cells results in the thickening of the transverse bone. Bone growth at the normal orientation resumes with the reconstruction of the cartilaginous template (Garn *et al.* 1968, Steinbock 1976). Following the subsequent movement of active growth from the site, the transverse structure remains, and becomes visible radiographically.

The relationship between periods of growth disruption signified by Harris lines and episodes of malnutrition or disease has been investigated. Attempts to correlate episodes of poor health with line formation in modern human populations have failed to show a correspondence with clinical disease, although there is always the difficulty of identifying subclinical disease or malnutrition (Dreizen *et al.* 1964, Marshall 1968). Gindhart (1969) found that lines formed after 25 per cent of episodes in modern children. The exact circumstances in which lines form remains unclear, although there is general acceptance that low-order correlations exist between line formation and episodes of environmental stress (Garn *et al.* 1968, Buikstra and Cook 1980, Mensforth 1981).

Other points relevant to the interpretation of the lines are that they are more likely to form during the vulnerable period of early infancy, but these are less likely to be retained into adulthood (Garn *et al.* 1968); there is no difference between lines formed after acute or chronic growth disruption (Steinbock 1976); and lines are more frequent in males than in females (Garn *et al.* 1968). Although the presence of lines indicates a period of recovery, their absence does not indicate that the individual did not suffer from environmental stress. Many lines may indicate periodic, perhaps seasonal, stress whereas fewer lines may indicate either that growth disruption was absent or that it was chronic. The consideration of the data with other indicators may clarify this point. The complexity of the aetiology and persistence of lines indicates that care needs to be taken in the interpretation of their presence and prevalence in skeletal populations. The interpretation should be approached with caution, and with due consideration of the range of other factors which may explain the patterns observed (Steinbock 1976, Goodman *et al.* 1984, Hummert and van Gerven 1985, Martin *et al.* 1985).

Lines were identified and recorded on both child and adult skeletons from Khok Phanom Di. They are most common in the distal tibia (Garn *et al.* 1968), and as this is the site most frequently used in other skeletal analyses, it is also used in this study. Antero-posterior radiographs, with the bone supported to maintain the correct orientation where necessary, were taken of all complete,

undamaged tibiae from individuals aged over three months. Where both tibiae from an individual were included, the bone with the greatest number of lines visible was used in the analysis.

Criteria for identifying lines vary considerably between observers. Methods used include counting lines extending a quarter or more of the distance across the bone (Goodman and Clark 1981), lines extending halfway or more across the bone (Maat 1984), lines extending 30 per cent or more across the bone (Clark and Mack 1988), or all lines visible, regardless of length (Simpson 1979, Crownover 1984). In practice, the identification of lines is not always totally objective, as the density of the bone can effect the clarity of the bone on the radiograph, regardless of the exposure used. The degree of remineralization of the skeletons from Khok Phanom Di has compromised the definition of structures in the bones most highly remineralized, and highlighted the presence of lines in those least remineralized. The criterion used was to count the lines extending at least a quarter of the distance across the bone, as lines of this size are more likely to be apparent on all radiographs and so the observations would be standardized. Only lines obviously transverse were counted, also to reduce the chance of including images which are artifacts of bone preservation. As the criteria used in analyses from other sites are variable, use of this minimum size of line does not further devalue intersite comparisons. Radiographs were taken of tibiae from 20 adult females, 24 adult males and 10 infants and children.

In addition to using presence or absence of lines as an indicator of growth disruption, further information may be obtained by estimating the age at which they formed (Martin *et al.* 1985). This may give some clues as to aetiology, if there are patterns of growth disruption at specific stages of development within the population. Several methods of estimating the age at the time of growth disruption have been devised. These are based on the size of the diaphysis at the time as indicated by the distance of the Harris line from the distal end of the diaphysis. Wells (1967b) made estimates based on regular growth increments within five-year periods between 2 and 16 years of age. Allison *et al.* (1974) and McHenry and Schulz (1976) also used regular growth increments to estimate annual growth. More refined methods were later developed by Simpson (1979), Hunt and Hatch (1981), Clarke (1982) and Maat (1984). These are all similar, and based on data on age-specific diaphyseal lengths from modern growth studies on white Americans by Maresh (1955) and Gindhart (1973). They make allowances for the variation in annual growth during infancy and childhood, including sex differences in growth rates, and take into account the differential growth at distal and proximal ends of the bone. There are variations among the authors in the suggested annual growth rates, but the methods have basic similarities. Clarke, Hunt and Hatch, and Maat assign lines to specific ages, and Simpson has used five-year intervals.

The system of Maat (1984) was used in this study. Although there may be differences in age-related growth velocities between the white American reference populations and the Khok Phanom Di population, lines have been assigned to specific ages of formation in this study, as this provides evidence of the age pattern of line formation. These are not seen as absolute ages of formation, and there will be no attempt to correlate the assessed age of formation with that of other indicators of growth disruption (this point is discussed further in the section on dental developmental defects). The range extends only to the age of fifteen, and although growth does not cease then, increments in the final years are minimal, and no lines were identified in the diaphyses adjacent to the line of epiphyseal fusion.

TABLE 48: *Number of individuals with Harris lines on the distal tibiae and the number of lines formed at each year of life for all adults and for both sexes*

Age when lines formed	Females		Males		Total	
	n individuals	n lines	n individuals	n lines	n individuals	n lines
<1	11	12	14	17	25	29
1	3	3	3	3	6	6
2	5	5	3	3	8	8
3	1	1	3	3	4	4
4	2	3	0	0	2	3
5	2	2	0	0	2	2
6	3	3	0	0	3	3
7	3	3	0	0	3	3
8	5	5	0	0	5	5
9	5	8	1	1	6	9
10	3	4	4	4	7	8
11	5	6	1	1	6	7
12	2	2	0	0	2	2
13	1	1	6	6	7	7
14	0	0	4	4	4	4
15	0	0	1	1	1	1
16	0	0	0	0	0	0
Total	20	58	22	43	42	101

The adults with Harris lines and the ages at which lines were formed are listed in table 65. Table 48 lists the number of individuals with Harris lines and the total Harris lines recorded by estimated age of formation for all distal tibiae. Of the 44 individuals, 42 had at least one line. The two individuals with no lines were both male. Table 49 summarises the mean number of lines formed by sex and age at death. Females have more lines per individual than males at the 10 per cent level of significance. The expected reduction in numbers of lines with increasing age and remodelling is not evident and there are no significant differences with age.

Figure 39 illustrates the proportions of individuals with lines at each age of formation for both sexes. This clearly shows a high proportion of lines formed in infancy, with 55 per cent of individuals (50 per cent of females, 58 per cent of males) having lines formed within the first year of life. Both sexes have bimodal peaks in line formation, later in childhood. The first peak in the females occurs at the age of 8–9 years and the second at the age of 11 years. The two male peaks occur approximately two years later. These age periods approximate the periods of maximum growth dur-

TABLE 49: *Prevalence of Harris Lines on distal tibiae by age at death for all adults and for both sexes*

Age	Females			Lines per individual Males			All adults		
	n	Mean	s.d.	n	Mean	s.d.	n	Mean	s.d.
<30 years	11	2.7	3.1	12	1.8	1.1	23	2.2	2.3
30+ years	9	3.1	2.3	12	1.8	1.4	21	2.4	1.9
Total	20	2.9	2.7	24	1.8	1.2	44	2.3	2.1

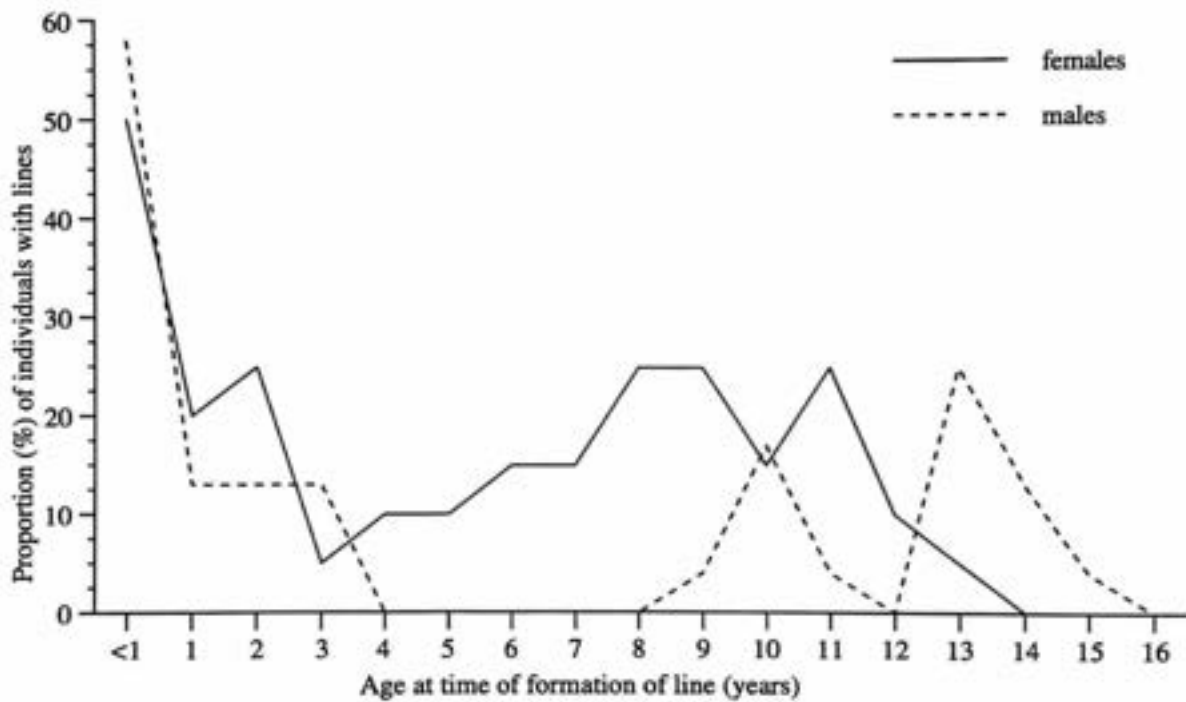
Females > males $p = .079$ 

FIG. 39. Proportion of adults with Harris lines formed at each year of growth

ing infancy and during the prepubertal growth spurt. Those individuals with lines formed in later childhood suffered more periods of growth disruption.

Eight of the 10 children with intact tibiae had Harris lines. The two unaffected individuals were aged less than one year at death. Table 50 lists the Harris lines recorded by the estimated age of formation for all distal tibiae. Individual data are listed in table 65. Three children had lines formed

TABLE 50: *Number of children with Harris lines on the distal tibiae and the number of lines formed at each year of life*

Age	n individuals with lines	n lines	Age	n individuals with lines	n lines
0	3	5	1	6	7
2	1	2	3	1	2
>3	0	0	Total	8	16

within the first year of life. The remainder of lines were all formed during the second year, with the single exception of B137. Apart from B137, the children do not have more lines than the adults. Compared with adults, the episodes of growth disruption in children appear to have occurred later in infancy and older children did not have episodes associated with the prepubertal growth spurt.

Harris lines are commonly included in reports of prehistoric populations, although the methods of defining and counting them varies considerably between authors, as previously discussed. The evidence from different populations is not necessarily directly comparable. Another problem is that not all authors use the tibia in making the assessment of line formation, introducing another element of variation in the data. Comparison is made here between the prevalence of line formation in the Khok Phanom Di adults and that of the prehistoric groups which are geographically closest, and a selection of other skeletal samples. The data for adults are summarised in table 51. At prehistoric sites in Kentucky, Cassidy (1984) found a mean number of lines per tibia of 11.3 at the pre-agricultural Indian Knoll site and 4.3 at the agricultural site of Hardin Village. Wells (1967b) reports mean numbers of lines per individual ranging from 1.6 to 5.1 in the tibiae of a series of seven Saxon and medieval groups from Britain. Within these groups, between 40 and 90 per cent of the individuals had lines.

The criteria used for identifying lines varies among the observers, with the Ban Na Di and Dickson Mounds samples based on the same principle as that used at Khok Phanom Di, counting lines that extended a quarter or more of the distance across the shaft. The Maori and Namu samples included all lines, regardless of length, and the Non Nok Tha and Niah Cave samples included lines which were parallel to the plane of the epiphyseal cartilage, and "of sufficient length to confirm transverse rather than oblique angulation *vis à vis* the shaft of the bone" (Crownover 1984:15). The number of lines at Ban Na Di and Non Nok Tha must be considered as minimum numbers, as in both cases the condition of the bone was poor, with considerable postmortem damage. The difficulty of making comparisons of mean numbers of lines among small samples is clear in the high standard deviations in the smaller samples. The two females from Ban Na Di are an extreme example, with one individual having 17 lines and the other a single line.

There are relatively few data from adults in modern populations, but Garn and Schwager (1967), in a study of the distal tibiae of 136 adults (87 females, 49 males) aged 25–50 years from the Fels Research Institute in Ohio, found lines on 14 per cent of males and 30 per cent of females. In 110 older individuals, aged 50–86 years, the percentages were lower at 8 per cent of males and 14 per

TABLE 51: *Summary of Harris lines in adults in prehistoric South East Asian and other skeletal populations*

	Females				Males			
	N	% with lines	lines ¹ mean	s.d.	N	% with lines	lines ¹ mean	s.d.
Distal Tibiae								
Khok Phanom Di	20	100	2.9	2.7	24	92	1.8	1.1
Ban Na Di	2	100	9.0	11.3	5	80	1.0	0.0
Namu, Taumako, Solomon Islands	7	86	5.9	4.0	28	86	3.2	3.0
Maori	101	-	5.9	5.1	81	-	5.5	5.2
Kulubnarti, Nubia	84	60	-	-	67	66	-	-
Dickson Mounds, Illinois	43	63	1.2	1.2	65	74	1.5	1.3
Femora²								
Non Nok Tha	-	-	-	-	5	80	4.0	4.2
Niah Cave, Sarawak	6	83	7.0	6.1	5	100	6.8	5.0

¹ Mean and standard deviation of number of lines per individual, ² "...burials possessing a right or left femoral fragment of at least 7 cm in length." Crowner 1984:48. Sources of Data:- Khok Phanom Di - this study; Ban Na Di - Reassessed from radiographs held in the Anatomy Department, University of Otago Medical School; Non Nok Tha and Niah Cave - Crowner 1984; Namu - Houghton, n.d.a. and unpublished data; Maori - Simpson 1979; Dickson Mounds - Goodman and Clark 1981; Kulubnarti - Hummert and Van Gerven 1985

cent of females. They counted lines extending 50 per cent of the distance across the shaft in this study, but in a later report, with a sample of 186 women aged 20–86 years (Garn *et al.* 1968), they counted lines as extending at least 5 mm into the tibial shaft. In this group 53 per cent of the individuals aged 20–50 years (n=112) had lines, which gives some indication of the effect of variation in the criteria for identifying lines. They do not report the number of lines per individual.

In the Khok Phanom Di population a greater proportion of individuals had lines than almost any other group, including the other prehistoric samples, but the mean number of lines per individual was relatively low.

Sex differences in the mean number of lines are minimal in the Dickson Mounds population (Goodman and Clark 1981) and in the Maori (Simpson 1979), but in the Namu group (Houghton, n.d.a) the females had more lines than the males. In the Ohio group, Gindhart (1969) found that the frequency of line formation was higher in males, but that the line persisted longer in females. At Khok Phanom Di females had on average more lines than males. This suggests that they had more episodes of ill health in childhood, although other possible explanations include the longer persistence of lines in females and the faster recovery of females from disruptive episodes. Assuming both sexes had the same degree of exposure to factors resulting in growth disruption, the females could

then have experienced more episodes compared with chronic disruption in males. A third possibility is the influence of cultural or social factors. Girls and boys may have been fed diets of differing quality, or one sex may have been expected to make a contribution to the work of the community at an earlier age and therefore to have been subject to more physical stress, such as fatigue. Together with the higher mean age at death of females, their more frequent recovery from growth disruption could be a reflection of the osteological paradox, therefore indicating that they had better health as they were able to recover from whatever caused the disruption.

The age at which the lines were formed is of interest in comparing the rate and pattern of line formation among populations. The high prevalence of line formation during the first year of life in the Khok Phanom Di people is not recorded in other samples. Two studies using samples from prehistoric Amerindian populations found the peak frequency of line formation at around two years of age (Goodman and Clark 1981 - Dickson Mounds, Clarke 1980 - Dickson Mounds and Mesa Verde), with less than 5 per cent of lines formed before the age of one year. In modern samples, Gindhart (1969) recorded peaks of line formation at about two to two and a half years of age, although all these lines were resorbed before adulthood. Dreizen *et al.* (1964) and Blanco *et al.* (1974) in studies of Guatemalan groups found peaks at about four years of age. In the Maori, Simpson (1979) used five-year age groupings and found a peak frequency during the ages of 5–10 years, with a mean of twice as many lines forming then as during the age ranges of 0–5 years and 10–15 years. In the Kulubnarti population Hummert and van Gerven (1985) recorded lines formed during the first year of life only in children who died before the age of seven years. The secondary peaks of line formation during later childhood recorded in the Khok Phanom Di population were also found in the Dickson Mounds population (Goodman and Clark 1981), at the age of 13 years in both sexes, although Clarke (1978, cited in Hummert and van Gerven 1985) records no increase in line frequency up to the age of 12 years in his study which included individuals from the same site (Dickson Mounds) as that reported by Goodman and Clark. In the adults from the Kulubnarti population Hummert and Van Gerven (1985) found very few lines formed before the age of 10 years, but at subsequent ages there was an increase from c. 5 per cent of the population with lines to over 50 per cent at the age of 16. Dreizen *et al.* (1964) and Gindhart (1969) found very few lines after the age of five years in the modern groups.

Sex differences in the pattern of ages of formation are very similar in the Khok Phanom Di and Dickson Mounds (Goodman and Clark 1981) groups. In both, a greater percentage of lines formed in infancy in the males, and there was an earlier adolescent peak in line formation frequency in the females. This pattern conforms with sex differences in the susceptibility of infants to growth disruption and in the age at onset of the period of adolescent growth (Tanner 1978).

The potential for variation resulting from the method of defining lines, the variety of physiological factors affecting their formation and retention into adulthood, and the differing state of preservation of the skeletal samples cannot be ignored in explaining the line formation rates and age patterns, but the correspondence of the patterns in the Khok Phanom Di and Dickson Mounds groups with periods of peak growth velocity, including the sex differences in the age of peak adolescent growth, lends biological validity to these results.

In summary, ignoring the complexities of methodology, the proportion of individuals with Harris lines in the Khok Phanom Di adults suggests that a greater proportion of the group suffered from

episodes of growth disruption than in other populations, particularly early in infancy, although each individual suffered relatively few episodes. Females may have had more episodes of disruption or shorter episodes, with more frequent recovery than males.

No directly comparable data for children in prehistoric populations have been located. As the number of lines which may have formed on any long bone will be to some extent dependent on the length of the bone, the prevalence of Harris lines in a population will be influenced by the age structure of the subadult skeletons, reducing the value of any comparison between populations.

Dental developmental defects - enamel hypoplasia, opacities and discolourations

The dentition is a source of information on health during childhood which has advantages over the evidence available from bone:

- Teeth are generally well preserved in archaeological sites. They are more durable than bone and less susceptible to postmortem damage.
- They provide a permanent record of the early growth period, from the ages of one to six years in the permanent dentition and from the antenatal period to the age of one year in the deciduous dentition. Dental enamel does not undergo the same sort of remodelling as bone.
- Adult dentitions can be included in the sample, except where attrition or caries have damaged the crown. The possibility of using evidence from individuals who have survived childhood is a major advantage, as these data will more closely reflect that of the living population. The bias of the mortality-selected child individuals is reduced, the sex of the individuals can be estimated, and the prevalence of developmental defects can be correlated against evidence of growth disruption and achievement of growth potential.

As Goodman *et al.* say:

An important step is the conversion from a recording of hypoplasias on teeth to a recording of the number and time of development of growth disruptions per individual so that statistical analysis may focus not on teeth as the unit of analysis but on individuals, and not on hypoplasias as datum but on growth disruptions. (Goodman *et al.* 1980:5)

Defects in dental enamel may be either inherited or developmental in origin. Inherited defects can generally be distinguished from developmental defects in that, although the form of the defects may have some similarities, the whole crown of the tooth is usually involved, including staining of the enamel (Hillson 1986). Developmental defects generally appear in the form of transverse defects in the thickness of the enamel on the crown, reflecting a period of disturbance of the increment of enamel on the tooth crown (El-Najjar *et al.* 1978, Cook and Buikstra 1979, Hillson 1986). The defect may be so extreme as to result in complete absence of enamel. Staining or opacities are generally patchy or associated secondarily with the transverse defects (Pindborg 1970).

There are two stages in the development of the enamel, with the formation of a primarily organic matrix and subsequent mineralisation during the maturation phase (Rose *et al.* 1985, Hillson 1986).

Ameloblasts are the cells which form enamel. Disturbance of their activity during matrix formation produces structural defects which may be visible either on the tooth surface or microscopically on sectioned teeth. Macroscopic defects, termed 'enamel hypoplasia', can vary from continuous transverse linear bands or lines to discontinuous linear pitting. The defects may also be single or multiple, with each representing an episode of disruption in the enamel formation process. There is no evidence for a direct relationship between the appearance of the defect and the duration and severity of disruptive episodes (Rose *et al.* 1985, Hillson 1986) and individual response will vary.

No two individuals with similar causative agents of enamel hypoplasia are likely to show the same pattern of expression on the crown surface. (El-Najjar 1978:191)

Neiburger (1990) suggests that there is an hereditary influence on the formation of hypoplasia, and that some populations or families will have susceptible dentitions while others show no evidence, even during severe malnutrition.

The disturbance of enamel maturation, the phase when most of the mineralisation of the tooth takes place, produces defects of the enamel which are described variously as opacities or discoloured enamel (Ainamo 1982) or hypocalcification (Cook and Buikstra 1979, Blakey 1981, Blakey and Armelagos 1985, Hanson 1987). These defects may appear with or without hypoplasia, but have not received as much attention in archaeological studies as enamel hypoplasia, except where they appear in the deciduous dentition (e.g. Cook and Buikstra 1979, Blakey 1981, Hanson 1987). This is possibly because they are often difficult to differentiate from postmortem staining.

The level of the defect on the tooth crown has been used as evidence of the age of the individual at the time of formation of the defect. This enables the construction of age specific patterns of disruptive episodes in a group.

There is a wide variety of environmental stresses which may produce defects including malnutrition (Goodman *et al.* 1989), disease episodes, and local trauma (Cutress and Suckling 1982, Pindborg 1982, Neiburger 1990).

The study of developmental defects and the estimation of the age at which developmental disruption has occurred in skeletal groups has been based on single teeth (for example, McHenry and Schulz 1976, Clarke 1982) on all teeth (Goodman *et al.* 1980) or on selected teeth (Perzigian *et al.* 1984). Ideally all teeth should be examined for defects, as different types of teeth are vulnerable in varying degrees to developmental disruption (Goodman and Armelagos 1985) and the developmental ages of tooth crowns vary. In the Khok Phanom Di dentitions, the buccal or labial surfaces of all teeth, both deciduous and permanent, were examined for enamel defects to ensure that all types of defect would be recorded.

In most studies of developmental defects in skeletal groups, the description of the defects is limited to that of linear enamel hypoplasia, and the recording and analysis is as of single, discrete events (e.g. McHenry and Schulz 1976, Clarke 1982). The lack of a standard definition was noted by Goodman *et al.* (1984), but the DDE Index (Ainamo 1982) developed for use in clinical studies can be used as a basis for classification in skeletal groups, and has been adapted in studies on living populations (for example, Goodman *et al.* 1987). A similar coding system has been used on the Khok Phanom Di population. This was modified to record the various types of defect and combinations of defect found as follows:

0. Normal enamel, DDE Code 0.
1. Linear enamel hypoplasia - horizontal, discrete, continuous grooves, DDE Code 4.
2. Linear enamel hypoplasia - horizontal, discrete, pitted grooves DDE Code 3.
3. Non-linear hypoplasia - areas of uneven enamel, usually not pitted but roughened, often on the gingival area and generally with indistinct vertical orientation of the deficiency (Fig. 40A), DDE Code 4.
4. Non-linear hypoplasia - discrete areas of missing enamel, generally circular and less than 1 mm in diameter (Fig. 40B) DDE Code 6.
5. Discoloured enamel, staining or hypocalcification - brown, usually horizontal but not continuing across the entire buccal/lingual surface of the tooth, DDE Code 7.
6. Combination of 1 + 3 (Fig. 40C-D).
7. Combination of 1 + 5.
8. Combination of 3 + 5.

The age of occurrence of the event or events which caused the disruption has been assessed by various methods by different authors. In the most commonly used method, the tooth crown is divided vertically into sections which are deemed to represent time periods during the growth of the tooth (Schulz and McHenry 1975, Cook and Buikstra 1979, Hillson 1979, Goodman *et al.* 1980, Maat 1984, Blakey and Armelagos 1985). The age at which the disruption occurred is then calculated from the position of the defect on the crown. One method of allocating the age is by dividing the tooth into equal sections based on the average length of the formation period for the tooth type (Rose 1977, Goodman and Armelagos 1985). As teeth are different sizes in different individuals because of varying growth periods, rather than varying growth rates (Moss and Moss-Salentijn 1977), this method assigns different amounts of tooth surface to each time period in different individuals, depending on the size of the tooth (Cook 1981). Another method is based on the developmental sequence of teeth of Massler *et al.* (1941) (Goodman *et al.* 1980, Maat 1984, Rose *et al.* 1985) but this carries many assumptions as to average lengths of the developmental period and the developmental sequence, which is population specific. Cook (1981), in a histological study, bases defect analysis on frequencies per mm to avoid these problems. This produces an assessment of disruption patterns without allocating ages, but would be applicable only to intact (unworn) teeth, as measurement must begin from the point of initial calcification, on the cusp of the tooth. The assessment of the age-related pattern of developmental disruption in the Khok Phanom Di dentitions has been based on the canines, both mandibular and maxillary, as these provide the largest sample of teeth with defects which are linear and therefore most likely to be developmental in origin. The age of formation is assessed on the basis of the position of the defect on the crown, using the method of

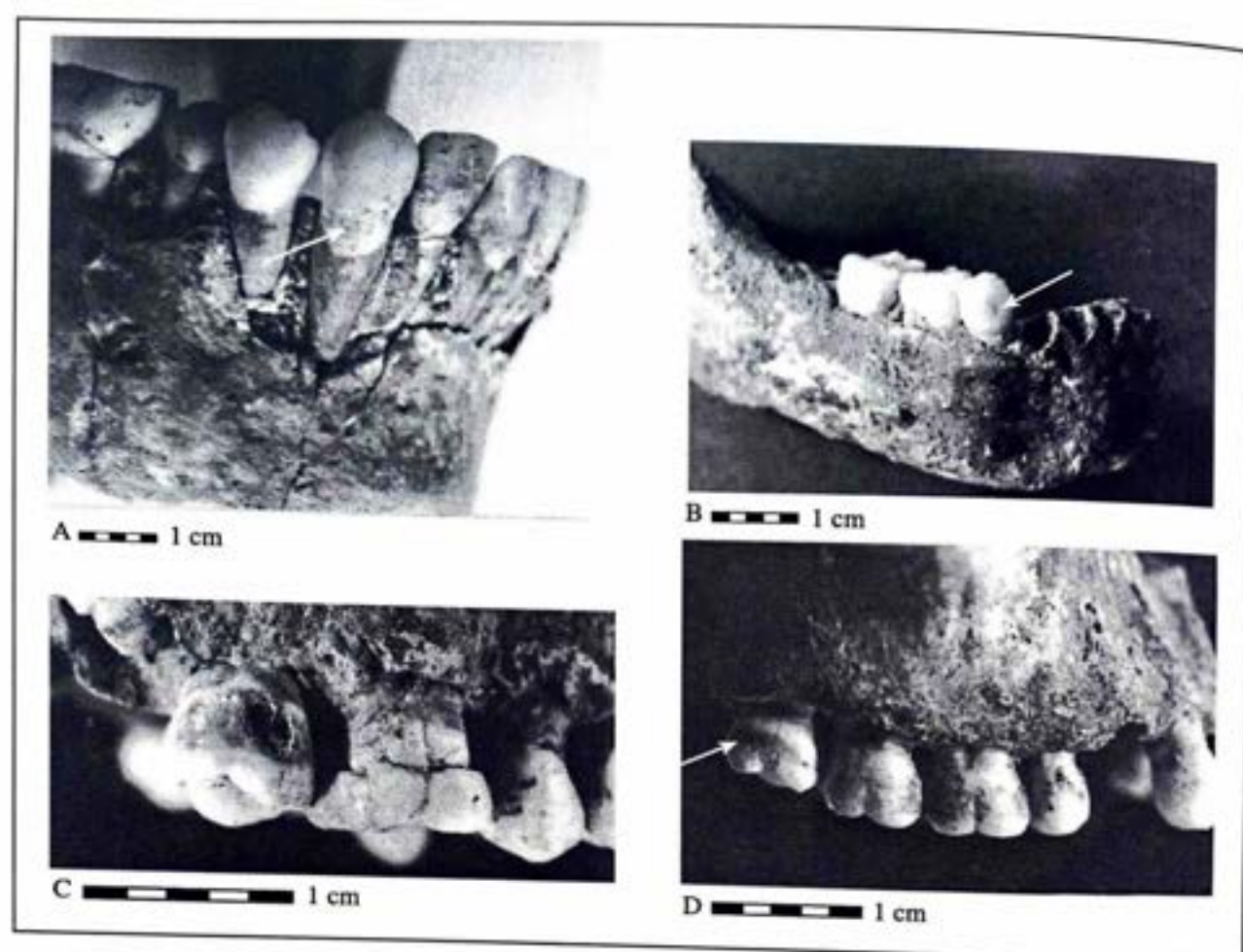


FIG. 40. A: Mandibular teeth with enamel hypoplasia code 3 on the canine (burial 8, 15 years, male?) B: Mandibular deciduous dentition with enamel hypoplasia code 4 on the canine (burial 96, 4 years) C: Permanent maxillary second molar with enamel hypoplasia and discolouration code 6. The hypoplasia is in the gingival third of the tooth. The hole in the discoloured enamel represents postmortem enlargement of a carious lesion. The damage to the adjacent tooth is postmortem and the alveolar bone has postmortem erosion D: Permanent maxillary third molar with enamel hypoplasia and discolouration code 6. In this tooth the enamel is pitted and close to the crown of the tooth

Goodman *et al.* (1980). This was adapted from the method developed by Swärstedt (1966) on the basis of the developmental sequence of Massler *et al.* (1941). The distance of the defect from the cemento-enamel junction is measured and translated into age of formation using the diagram illustrated in figure 41.

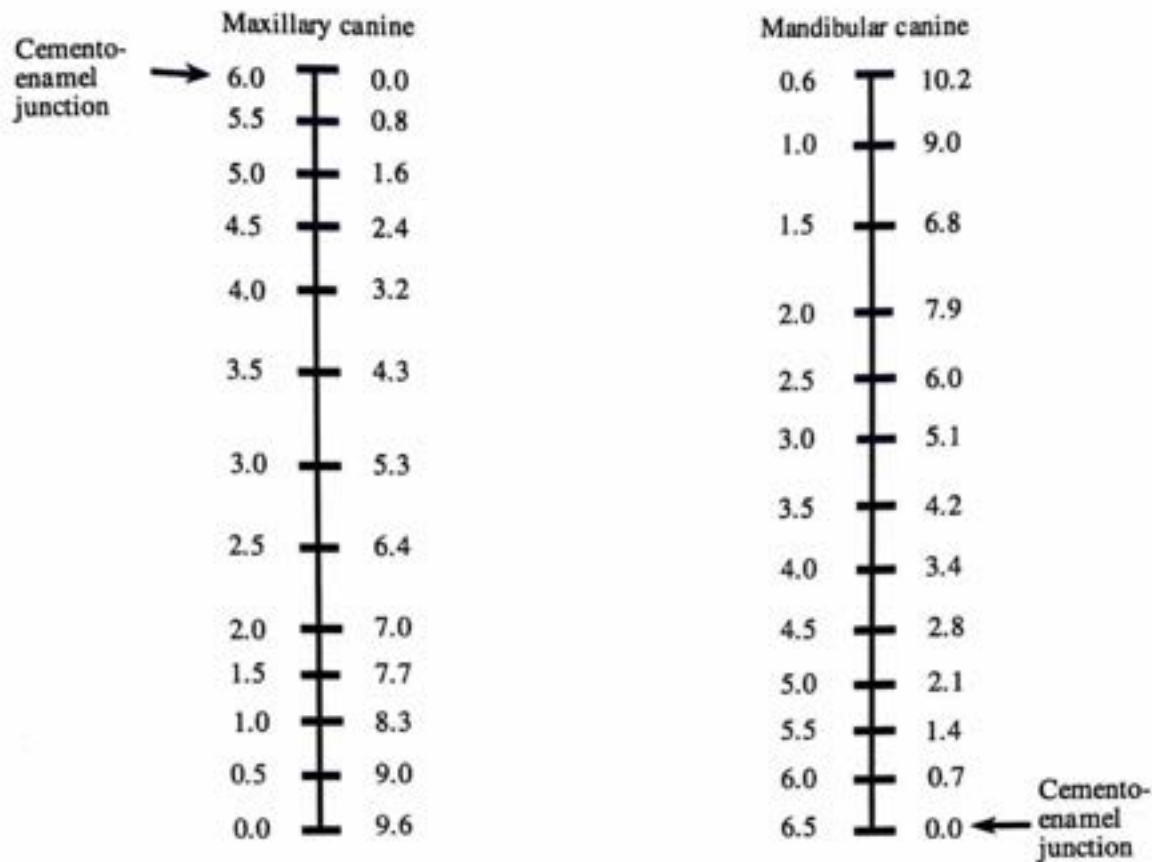


FIG. 41. Mineralisation diagram for the enamel of the permanent canines. This is the basis on which the age at which linear enamel hypoplasia formed was calculated. The numbers to the right of each line are the lengths of the tooth crowns in mm from the cemento-enamel junctions. The numbers to the left of each line are the corresponding ages at which the sections of enamel developed (modified from Goodman *et al.* 1980:520)

In the case of discrete, linear or pitted hypoplasia (codes 1, 6), the measurement is of the defect, but with the irregular hypoplastic enamel (code 5), the measurement is of the beginning of the defective area, as in all cases this continued over the remainder of the crown, to the cemento-enamel junction. It is clear that the application of a standard which is based on a very different population with a possibly different developmental sequence cannot provide accurate data, and that measurement from the point which represents the end of enamel formation rather than the beginning will introduce inter-individual variation in the age assessments. As with Harris lines, the ages estimated are not regarded as absolute but provide a basis for the analysis of patterns of disruption. All teeth were examined under magnification, with oblique lighting, and measurements were taken to the nearest 0.1 mm with dial calipers. The examination of the dentition was confined to macroscopic

defects, as the sectioning of teeth necessary for microscopic analysis was not attempted for this study.

Of the 68 adults, 65 had at least one intact tooth, but 13 individuals with fewer than 10 intact teeth (less than one-third of the dentition) were excluded from the analysis, to reduce sampling error. The 52 (26 females, 26 males) individuals included in the analysis have a mean number of 21 intact teeth. All individuals except one female (B35) and one male (B92) had defects on at least one tooth. Details for each individual are not listed because of the bulk and complexity of the data. Defects have been classified in groups of linear and non-linear and these groups have been separately analysed. Table 52 summarises the results.

Seventy-one per cent of adults had both linear and other enamel defects, and 38 per cent had at least one tooth with multiple defects. A majority of defects were classified as linear hypoplasia, occurring either alone or in combination with non-linear gingival hypoplasia, and the latter was the next most frequently occurring defect. The frequencies and patterns of enamel hypoplasia do not differ significantly between males and females.

Table 53 summarises the rates of defective enamel formation by tooth category. This shows that the teeth with the most defective enamel were the maxillary incisors and the canines in both jaws, and the second and third molars, particularly the maxillary third molars. The patterns of different types of defect vary among different teeth categories, and between the maxillary and mandibular dentitions. Both upper and lower incisors have virtually only linear defects. The defects in the canines are predominantly linear, but other types were also recorded. A high proportion of the defective teeth, particularly in the mandible, have multiple defects. The proportion of defects which are linear decreases towards the posterior teeth, and the third molars have virtually only non-linear defects, with the multiple defects on the maxillary third molars comprising combinations of virtually only non-linear defects. The proportion of teeth with multiple defects also increases towards the posterior dentition, except on the mandibular third molars.

The estimation of the ages at which the defects formed and, by implication, the ages at which growth disruptions were suffered are summarised in table 54. The data for linear hypoplasia show the highest prevalence of recorded defects over the ages of three to four years, and the highest number of defects per individual at the ages of one and three years. As this sample includes adults of all ages, wear on the occlusal and lingual surfaces of the teeth, particularly in older individuals, may have obliterated some of the defects on enamel formed in the early years. The data for non-linear hypoplasia show that the onset of formation of the defective enamel was at the ages of three and four years on the canines.

Table 55 shows the number and proportion of individuals with defects in those who died before the age of 30 years compared with those who died after this age. This shows that the individuals who died at a younger age had a higher number of defects extant on the enamel formed at early ages, which may reflect the lower degree of attrition of the enamel near the cusps of the teeth. Despite this, the total figures show that those who died before the age of 30 years did not have more episodes of growth disruption in total than those who survived longer.

The same data recorded for both permanent and deciduous dentitions of children are summarised in tables 56-57. All children with fully or partially erupted teeth in either dentition are included in the data, although in all cases either or both dentitions were incomplete or minimally represented.

TABLE 52: *Number and proportions of adults with at least one tooth with each type of developmental defect, and number and proportions of teeth with each type of defect*

Defect types*	All adults		Females		Males		Teeth		
	n	%	n	%	n	%	n	%	%
							all teeth defective teeth		
1 LEH - continuous lines	44	85	23	88	21	81	223	21	49
2 LEH - pitted lines	1	2	1	4	0	0	2	0	0
Total Linear defects	45	87	24	92	21	81	225	21	49
3 Non-linear EH - gingival	31	60	13	50	18	69	137	12	29
4 Non-linear EH - discrete	7	13	3	16	4	15	10	1	2
5 Discolouration	14	26	11	42	3	16	40	4	9
Total non-linear defects	38	73	18	69	20	77	187	17	40
Individual teeth with multiple defects									
6 Continuous LEH and gingival EH	14	26	6	23	8	31	41	4	9
7 Continuous LEH and discolouration	1	2	0	0	1	4	2	0	0
8 Gingival EH and discolouration	10	19	4	15	6	23	11	1	2
Total multiple defects	20	38	7	27	13	50	54	5	11
Total all defects	50	96	25	96	25	96	466	43	100
Summary of Individuals									
With both LEH and non-LEH	37	71	18	69	19	74			
With LEH only	9	17	6	23	3	11			
With non-LEH only	4	8	1	4	3	11			
Total	50	96	25	96	25	96			

* The numbers refer to the codes listed on Page 127; LEH = Linear enamel hypoplasia, codes 1,2; non-LEH, non-linear enamel hypoplasia, codes 3-5

There were four children with both deciduous and permanent teeth present. In three cases (burials 6, 21, 37), defects were recorded on the permanent teeth only, and in one case (B145) on the deciduous teeth only. This shows very different patterns of enamel developmental defects in the two dentitions. The principal defect visible in the deciduous dentition is discrete, non-linear hypoplasia (Fig. 40B). A few dentitions had teeth with discoloured enamel. The differentiation of teeth with discolouration of the enamel which was unequivocally developmental from those with discolouration which was postmortem was difficult. In some cases, the presence of caries in association with

TABLE 53: *Numbers and proportions of each tooth with enamel defects (adults, sexes combined). The teeth are numbered from mesial to distal (i.e. 1 = first incisor, 8 = third molar)*

Tooth	Maxillary teeth								Mandibular teeth							
	Total ¹		LEH ²		Other ³		Multiple ⁴		Total		LEH		Other		Multiple	
	n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	%
1	41	83	33	97	0	0	1	3	36	25	9	100	0	0	0	0
2	22	59	9	69	4	31	0	0	49	41	19	95	0	0	17	5
3	63	76	24	50	15	31	9	19	86	83	36	48	15	21	20	28
4	68	28	13	68	6	32	0	0	98	30	17	59	11	42	1	3
5	64	14	4	57	5	43	0	0	95	16	8	53	6	40	1	7
6	73	32	12	52	9	39	2	9	87	22	11	58	6	16	2	11
7	84	56	12	26	32	68	3	6	86	41	9	26	21	60	5	15
8	65	80	4	8	39	75	9	17	69	36	5	20	20	80	0	0

¹ Total = number of teeth in each sample, and proportion with enamel defects; ² LEH = number of teeth with linear enamel hypoplasia (codes 1,2, refer page 127) and proportion of defective teeth represented by these defect types; ³ Other = number of teeth with non-linear enamel hypoplasia and discolouration (codes 3-5), and proportion of defective teeth represented by these defect types; ⁴ Multiple = number of teeth with combinations of both linear and non-linear defects (codes 6-8) and proportion of defective teeth represented by these defect types

discolouration suggests the presence of an underlying enamel defect. It is possible that the source of the stain itself may have been postmortem, but that the presence of an existing hypoplastic defect of the enamel resulted in the selective staining of the defective areas. Figure 42 illustrates central maxillary deciduous incisors with enamel discolouration.

The proportions of individuals and of teeth with various defect types in the permanent dentition in the children is similar to that in the adult dentitions, except for a much lower prevalence of linear hypoplasia. The non-linear defects on the gingival part of the teeth follow the same pattern as that in the adults, although in some cases the enamel is also opaque.

The distribution of defect types between the dental arcades and among tooth types is summarised in table 57, although the relatively incomplete state of both deciduous and permanent individual dentitions means that these data are not as indicative of defect prevalence as those for the adult dentitions. In the deciduous teeth the canines, both upper and lower, are most commonly affected by the discrete enamel defects, and the maxillary incisors by staining. Upper and lower teeth are equally affected if all defects are considered. In the permanent teeth, the maxillary teeth have almost twice as many defects as the mandibular, with the molars having both gingival hypoplasia and multiple defects. Both upper and lower canines are affected by gingival hypoplasia.

As there are minimal numbers of permanent teeth with linear hypoplasia, no data have been collected on the age distribution of the events responsible for the developmental disruption.

The discrete defects on the deciduous teeth are predominantly on the lateral buccal surfaces, although some are on the lingual surfaces. The level of the staining of the anterior teeth varies from

TABLE 54: *Ages at which defective enamel was formed, based on the canine teeth of adults (n=51 individuals, sexes combined)*

Linear Enamel Hypoplasia				
Age (Years)	Individuals with defects		Mean defects per individual	Proportion of defects (%)*
	n	%		
<1	2	4	1.00	2
1	5	10	1.20	6
2	13	25	1.08	15
3	27	53	1.37	39
4	26	51	1.04	28
5	9	18	1.00	9
6	1	2	1.00	1
Total	36	71	2.67	100
Non-linear Enamel Hypoplasia				
<1	0	0		0
1	1	2		4
2	2	4		8
3	13	25		54
4	8	16		34
5	0	0		0
6	0	0		0
Total	21	41		100

*Proportion of all defects formed at each age of life

appearing only on the cusp of the tooth to extending down two-thirds of the crown but excluding the gingival area. Most of the crowns are formed prenatally (Schour and Massler 1941), and therefore the defects formed *in utero* or perinatally, with calcification normal from birth.

As with Harris lines, there is potential for significant interobserver variance in the defining and reporting of developmental defects of the dental enamel. Most reports of dental defects in skeletal populations cover only linear enamel hypoplasia in adult dentitions, and as this is the enamel defect least likely to be reported differently by different authors, the intersite comparison made here is restricted to that defect. Table 58 summarises the data available for linear enamel hypoplasia in prehistoric Thai and other skeletal populations.

Of the prehistoric Thai populations not included in this list, at Non Pa Kluay, Pietruszewsky (1988b) records hypoplasia on two of a total of 110 teeth in the sample, but does not say whether these were from one or two individuals. No mention is made of hypoplasia on the Ban Kao den-

TABLE 55: *Number of adults with canine linear enamel hypoplasia formed at various ages in relation to age at death, and the mean number of lines per individual*

Age at which defect formed (years)	Individuals <30 years at death		Individuals 30 + years at death	
	n with lines	mean lines per individual	n with lines	mean lines per individual
0	2	.11	0	.00
1	4	.21	2	.11
2	7	.37	7	.39
3	15	.79	23	1.28
4	13	.68	15	.83
5	2	.11	7	.39
6	0	.00	1	.06
Total	17	2.53	20	2.75



FIG. 42. Maxillary central incisors from burial 88 (age 9 months) with areas of discoloured areas of enamel which may represent developmental defects or have occurred postmortem. Both teeth have areas of enamel damaged on the distal lingual which may have been carious lesions. The root on the tooth on the right has postmortem damage

titions (Sangvichien *et al.* 1969), but lines are clearly visible on the upper first incisors and lower canines in the illustration of one individual in the plates in their report.

It appears that a much greater proportion of the Khok Phanom Di people than of other prehistoric Thai had suffered from episodes of growth disruption during early childhood, but the anecdotal nature of the recording of the prevalence of enamel hypoplasia and the relatively poor preser-

TABLE 56: Number and proportion of children with each type of developmental defect, and number and proportion of teeth with each type of defect. 'Individuals' designates those with at least one tooth with a defect

¹The numbers refer to the codes listed on page 127

Non-LEH = non-linear enamel hypoplasia, codes 3-5

vation of the dentitions from the sites other than Khok Phanom Di are likely to have affected the comparability of the data. In contrast, the percentage of individuals with at least one affected tooth from the other skeletal populations from the Pacific and North America is similar to that at Khok Phanom Di. In the reports of each of these groups the authors have identified a major pathological or nutritional problem. Over 80 per cent of the Namu population have evidence of tertiary yaws (Houghton, n.d.a), endemic malaria and malnutrition are suggested as having affected the Nebira people (Houghton n.d.b), and Simpson (1979) suggests that the prehistoric Maori were subjected to seasonal food shortages with associated opportunistic infection. The proportion of individuals at Dickson Mounds with linear enamel hypoplasia increased over time from 45 per cent to 80 per cent, and Goodman *et al.* (1980) ascribe this to nutritional deficiencies arising from a change in subsistence from hunting and gathering to maize cultivation.

In modern populations, Goodman *et al.* (1987) found 47 per cent of a sample of 300 Mexican children living in 'marginal' conditions had enamel hypoplasia, and they quote a selection of studies on English and New Zealand groups, who can be assumed to have had adequate diet and health care.

TABLE 57: Rates of enamel defect formation in children by tooth number (numbered mesial-distal), showing the number of teeth affected and the proportion of defective teeth represented by each defect type

Tooth	Discrete ¹		Maxillary Stain ²		Total ³		Discrete		Mandibular Stain		Total	
	n	%	n	%	n	%	n	%	n	%	n	%
Deciduous teeth												
1/2	1	2	8	21	9	23	0	0	6	15	6	15
3	5	13	1	2	6	15	7	18	2	5	9	23
4/5	1	3	5	13	6	16	0	0	3	8	3	8
Total	7	18	14	36	21	54	7	18	11	28	18	46
Permanent Teeth												
Tooth	LEH ⁴		Maxillary Other ⁵		Multiple ⁶		LEH		Mandibular Other		Multiple	
	n	%	n	%	n	%	n	%	n	%	n	%
1/2	3	6	2	4	1	2	4	8	0	0	0	0
3	1	2	7	15	1	2	0	0	9	19	0	0
6/7	1	2	8	16	6	13	0	0	3	6	2	4
Total	5	10	17	35	8	17	4	8	12	25	2	4
Total each arcade					30	62					18	38

¹Enamel hypoplasia code 4 - refer to page 127 for description of codes

²Discolouration (code 5)

³Total of teeth with defects

⁴Linear enamel hypoplasia, continuous (code 1)

⁵Gingival hypoplasia and discolouration (codes 3, 5)

⁶Combination of linear enamel hypoplasia and gingival hypoplasia (code 6)

reporting less than 5 per cent of children with enamel hypoplasia (Murray *et al.* 1984, Cutress *et al.* 1985, De Liefde and Herbison 1985).

The mean number of linear defects per individual is reported by Simpson (1979) for Maori. He found a mean of 1.90 (s.d.1.69) lines per individual, and Goodman *et al.* (1980) found a mean of 1.31 lines per individual in the Dickson Mound group. This compares with a mean of 2.67 lines on the Khok Phanom Di people, based on the canine teeth alone. The absence of sex differences in the proportion of individuals with lines at Khok Phanom Di is also evident in most other prehistoric populations. The equivalent of the non-linear enamel hypoplasia and discolouration which appears on the permanent dentitions of the Khok Phanom Di population is not reported in any other skeletal populations. The possible aetiology of these defects are not clear, as the distribution on individual teeth and across dentitions does not fit with developmental patterns.

TABLE 58: *Percentage of individuals with linear enamel hypoplasia in adults in prehistoric South East Asian and other skeletal populations*

	Females		Males		All adults	
	N	% with LEH ¹	N	% with LEH ¹	n	% with LEH ¹
Khok Phanom Di	24	92	21	81	45	87
Ban Na Di	13	21	12	42	25	27
Namu, Taumako	-	-	-	-	129	74
Maori	43	84	28	74	81	9
Nebira	-	-	-	-	33	100
Dickson Mounds	50	66	50	62	111	66
Indian Knoll	-	86	-	86	-	86
Hardin Village	-	73	-	92	-	-

¹Linear enamel hypoplasia

Sources of Data:- Khok Phanom Di - this study; Ban Na Di - reassessed from material held in the Anatomy Department, University of Otago Medical School; Namu, Taumako, Solomon Islands - Evans 1987; Maori - Evans 1987; Nebira, Papua New Guinea - Evans 1987; Dickson Mounds, Illinois - Goodman *et al.* 1980; Indian Knoll, Hardin Village, Kentucky - Cassidy 1984

In summary, the proportion of adults with enamel hypoplasia at Khok Phanom Di is high compared with other prehistoric groups, and the number of disruptive episodes is also greater, although the pattern of response of the dental enamel appears to be unusual.

As is the case with Harris lines on the long bones, the prevalence of visible enamel hypoplasia on the permanent teeth of children will be to some extent dependent on the developmental stage of the child, ie the number of teeth erupted, which is in turn dependent on the age at death of the children. Any comparison between populations requires that the sample subadults be of similar age. Accordingly, no comparative data are reported here.

Reports of dental enamel defects in the deciduous dentitions of other skeletal populations mainly cover only those types of defect classified either as discolouration (hypocalcification) or linear hypoplasia. The proportion of children with defective teeth in the Khok Phanom Di population (5/18 - 28 per cent) is relatively small compared with those in the Amerindian populations from Illinois reported by Cook and Buikstra (1979 - 50-60 per cent) and Blakey and Armelagos (1985 - 64 per cent), but if the sample from Khok Phanom Di is reduced to those aged under five years at death who have not yet lost any anterior teeth, the proportion increases to 38 per cent (5/13). Of the same age group, Cook and Buikstra found 80-95 per cent with defects. The proportion at Khok Phanom Di is closer to that reported from modern populations where the assessment is based on the anterior teeth only. Sweeney *et al.* (1969) found 42.5 per cent with linear enamel hypoplasia in a population with mixed nutritional status in Guatemala, and Enwonu (1973) found 21 per cent in poorly nour-

ished children in Nigeria. None of the defects in the Khok Phanom Di population is of the more severe form reported from some populations, where a large proportion of the crown has virtually no enamel (e.g. Cook and Buikstra 1979).

A high proportion of the defects formed during the prenatal period in both the skeletal and modern populations, a pattern also seen at Khok Phanom Di. Cook and Buikstra (1979) and Blakey and Armelagos (1985) suggest the interaction of nutrition and infection in the aetiology of the defects. Enwonu (1973) found an association with marginal maternal diet, including protein deficiency, and in at least one case the mother recalled having an acute dose of malaria during early pregnancy. Sweeney *et al.* (1969) found an association of the defects with prematurity and infections in the first month of life.

The other form of enamel hypoplasia found on the Khok Phanom Di teeth, discrete areas of deficient enamel on the buccal surfaces of mainly the canines, was not specifically reported by Cook and Buikstra or Blakey and Armelagos, but has been discussed in some detail by Skinner and Hung (1989). It has been found in 70 per cent of children from Upper Palaeolithic Europe (Skinner and Hung 1989), and has been seen in prehistoric New Zealand Maori and prehistoric Fijians (pers. observation). Skinner and Hung (1989) found the defect in 13 per cent of Indo-Asian children compared with 2.4 per cent of European children in a Canadian sample, and ascribe the defect formation to nutritional factors. They suggest that calcium deficiency in the mother and infant, which could be more prevalent in East Asian or Chinese where lactose intolerance is common, resulted in deficient cortical bone over the forming canine. Minor physical trauma could then result in damage to the ameloblasts of the developing tooth from about six months of age.

Correlation of skeletal and dental evidence of growth disruption

A number of authors have attempted to correlate the patterns of formation of dental defects with Harris lines (Wells 1967b, McHenry and Schulz 1976, Clarke 1982, Maat 1984). These studies have generally not shown high levels of correlation between the two indicators, which Clarke (1982) suggests is a reflection of susceptibility of the bones and dentition at differing stages of the disruptive event and the multifactorial aetiological nature of both conditions. The numbers of assumptions about rates and ages of bone and tooth development which are incorporated into the assessments of ages of the occurrence of the disruptions suggest that methodological errors are as likely explanations for the lack of correlation.

Infant and child mortality

The final response of the organism to environmental stress is death. Individual ages at death and group mortality rates provide a basic source of information on childhood health, and represent the only information from this age group which is not biased by the method of sample selection. If the sample is representative of the population, the skeletal collection provides accurate information on infant and child mortality in the living population.

The relative accuracy of age at death estimations for infants and children, compared with those for adults, offer the possibility of more precise analysis of age group characteristics. An example of this is the identification of weaning-age deaths and discussion of the causes in Cook (1979) and Cook and Buikstra (1979). In more general terms, the correlation between such indicators as

enamel hypoplasia and mortality rates in children is another source of information (e.g. Goodman and Armelagos 1988). The methods of assessing age at death in infants and children were detailed in chapter II.

TABLE 59: *Infant and child mortality rates*

	n	%
Age at death (in years)		
Neonates	47	31
< 1 year	16	10
1-4 years	11	7
Subtotal <5 years	74	48
5-9 years	7	5
10-14 years	5	3
Subtotal 5-14 years	12	8
Total <15 years	86	56

The individual age estimations are listed in table 13. Table 59 is a summary of infant and child mortality. The deaths of neonates and infants within the first year of life are shown separately, as these constitute a substantial proportion of child mortality. The remainder of the deaths are shown in five-year age groups. Figure 43 is a histogram of the frequency of total deaths at each year of age.

The histogram illustrates the very high number of deaths, primarily of neonates, during the first year of life. This group accounts for 41 per cent of all deaths and 60 per cent of the deaths of children. The death rate is much lower during the second and third years but overall, 86 per cent of all childhood deaths occur before the age of five. Between the ages of two and fourteen years, the numbers of deaths at each age form a small proportion of all deaths. Ten (71 per cent) of the fourteen deaths which do occur, are of individuals aged between eight and twelve years.

Comparative infant and child mortality rates from a selection of prehistoric sites and modern populations are listed in table 60. The comparative data used are those from sites where the proportion of infants is high enough to suggest that the sample is a realistic representation of infant mortality. Those sites with a low proportion of infants are excluded on the grounds that this may represent sampling error, as discussed in chapter II.

The level of young infant (<1 year) mortality at Khok Phanom Di is high compared with other skeletal samples (Table 60). Apart from the sample from prehistoric Lerna, in Greece, no other group exceeds 30 per cent for all infant deaths. Lerna is also the only other site providing data on the proportion of infants represented by neonates. Again it is at a similar level to that at Khok Phanom Di. The level of total infant mortality at Khok Phanom Di is also high compared with the samples of modern, non-affluent societies and with hunter-gatherers. In contrast, mortality in the age groups from 1-4 and 5-14 years, where sampling is less likely to influence the data, is at the lower end of

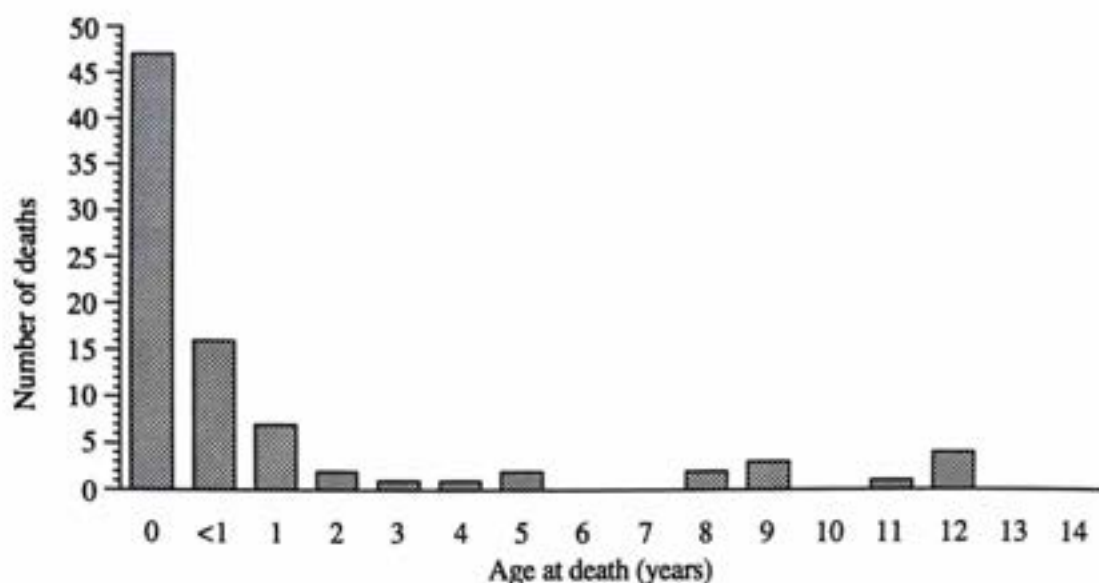


FIG. 43. Infant and child mortality

the ranges at other prehistoric sites. It is not significantly different from all other sites other than the Amerindian site of Hardin Village, which has significantly higher rates in both age groups. The mortality rates at Khok Phanom Di are also lower than in the modern hunter-gatherer groups. The total deaths of infants and children at Khok Phanom Di is within the same range as the total deaths at the other prehistoric sites and within the range of more modern groups. Temporal variations in infant and child mortality are discussed in chapter VIII.

SUMMARY - HEALTH DURING CHILDHOOD

Evidence from adult skeletons

The evidence of growth and maturation suggests that the inhabitants of Khok Phanom Di suffered episodes of ill health during childhood which were of sufficient severity and/or duration to interrupt growth to the extent that there was an ultimate effect on adult stature. The long-term effect of the growth disruption was greater in the males than the females, as indicated by the relatively short stature of the males compared with individuals from other prehistoric sites. Both the low sexual dimorphism in stature, again reflecting the relatively short stature of the males, and the early age at menarche in the females support the proposition that the long-term effect was greater in males than females.

The skeletal and dental indicators of growth disruption support the theory that a majority of individuals of both sexes suffered from episodes of ill health in childhood. The higher mean number of Harris lines recorded on the females appears to be at variance with the suggestion of more

TABLE 60: *Infant and child mortality at Khok Phanom Di compared with other skeletal populations and ethnographic and historical data. All mortality rates are expressed as a proportion of total deaths*

		% Dying at the following ages (years)							
	n	0	0-1	1-4	0-4	5-9	10-14	5-14	0-15
Skeletal									
1. Khok Phanom Di	154	31	41	7	48	5	3	8	56
2. Ban Na Di	57		24	9	33	7	2	9	42
3. Lerna	234	30	36	13	49	4	3	7	56
4. Indian Knoll	295		23	10	33			12	45
5. Hardin Village	296		13	20	33			20	55
6. Arroyo Hondo	120	26	16	42	10	5	15	57	
7. Skeletal			13-30						40-60
Ethnographic/ Historical									
8. Pre-industrial		10-40							30-70
9. 18th/19thC Europe		20-30							40-60
10. Modern hunter-gatherer		6-50*							20-50
11. Modern 3rd world		10-20							
12. Modern affluent		1-1.2							

* Mean 15-25; Data sources; 1. This study; 2. Houghton and Wiriyaromp 1984; 3. Angel 1971; 4-6. Cassidy 1984; 7. Acsádi and Nemeskéri 1970; 8. Weiss 1973a; 9-12. Multiple sources, listed in Cohen 1989

prevalent male ill health. This may reflect either greater persistence of lines in females, as already discussed, or it may support the evidence from stature by showing that the females recovered more readily and more often from episodes of ill health, producing more lines. Dental evidence of growth disruption is similar in both sexes.

The predominance of Harris lines in the Khok Phanom Di people in the first year of life rather than at ages two to four years as reported from other groups, suggests a different aetiology from the 'weaning age' stresses offered as an explanation for the high frequency in older infants (Clarke 1980, Goodman and Clark 1981). These are listed by Clarke (1980) as associated with the increasing mobility of 2-4 year olds and exposure to environmental pathogens, the nutritional and emotional stress of weaning, and physiological changes such as the decrease in internally regulated immuno-globulin levels and the loss of maternal immunity from milk. None of these factors should have an effect on infants during the first year of life. The loss, during the second six months of life, of the maternal immunity acquired in the passive transfer from the placenta is possibly more relevant. The definition of the ages of Harris line formation in periods of less than one year is stretching the validity of the method, but a large percentage (78 per cent) of the first-year disruptive episodes

in the Khok Phanom Di people do appear to have occurred later in the first year, the time at which the passive immunity reduces, rather than near the time of birth.

The sex differences in the second concentration of line formation during the peak growth periods of adolescence suggest that these may reflect the greater opportunity for line formation at these ages rather than an increase in disruptive episodes, although the latter cannot be discounted. The relatively low numbers of lines per person at Khok Phanom Di and the ages at which they occurred suggest that the disruptions were not seasonal, and therefore reflect discrete episodes of illness rather than cycles of nutritional inadequacy or disease. The ages at which the formation of the enamel was disrupted contrasts with Harris lines, in that the concentration of the highest proportion of defects on the canines is at the age of three in the Khok Phanom Di people, and the highest proportion of individuals with defects on all teeth at the ages of three-four years. The lack of correlation between the skeletal and dental indicators of growth disruption has also been illustrated in other populations (McHenry and Schulz 1976), perhaps reflecting the differing responses of the tissues to ill health rather than negating the assumptions about their aetiology. The enamel hypoplasia could reflect the pattern of growth of the tooth and the pattern of wear as much as the pattern of episodes of growth disruption, as previously discussed. According to the mineralisation diagram for the teeth in Goodman *et al.* (1980), the maxillary canine grows most rapidly during the ages of two-three years, and the mandibular canine during the ages of one-three years. The greatest opportunity for formation of hypoplastic enamel is therefore during these ages, and Rose (1977), who found a concentration of defects also at about the age of three in the Dickson Mounds population, suggests that the thinner enamel at this point on the tooth has ameloblasts which are more sensitive to growth disruption. The enamel closest to the occlusal surface is most likely to have worn in older individuals, erasing hypoplasia formed at the ages of one-two years, and resulting in the maximum extant defects in adults in the enamel formed at the age of three. An alternative explanation is that these defects represent the 'weaning-age' propensity for ill health already discussed.

The non-linear dental enamel defects appear most commonly on the canines, especially the mandibular, and on the second and third molars, especially the maxillary. Although the calcification of the crowns of these teeth is not contemporaneous in many populations (Ubelaker 1978) the mean eruption times of the canines and second molars in a small sample of modern Thai (Kamalanathan *et al.* 1960b) are within two years of each other. The pattern in the Khok Phanom Di burials raises the possibility that, if the gingival third of the crown of the relatively long canines, that of the second molars, and the occlusal half of the third molars were all forming at the same time, it is possible that a major event which caused disruption of growth produced the defective enamel on all these teeth. The ages at which the calcification would be occurring would be about seven to ten years, which coincides with the estimated ages of formation of the first of the two 'post-infant' peaks in Harris line formation in both sexes. The higher proportion of males than females with non-linear hypoplasia, in association with the probable later development of teeth in the males, strengthens the argument for contemporaneous development and disruption of the affected crowns, as the older the individual, the more likely the third molars are to be calcifying.

To summarise, the evidence of childhood health from adult skeletons shows that virtually all individuals at Khok Phanom Di suffered episodes of ill health during childhood. The most common ages of ill health were the first year of life, at about age three, and during the ages of six to

twelve years in females and nine to fifteen years in males. The males either suffered more severe episodes of ill health, or recovered less easily than the females, as their relatively short stature suggests inadequate catch-up growth. Compared with their prehistoric contemporaries in Thailand, the minimal comparative data suggests that childhood was a relatively difficult time for the people at Khok Phanom Di. The ubiquity of the episodes of growth disruption demonstrated by the adults is comparable with the patterns in other, more widely spread prehistoric peoples, in whom major health problems have been identified as listed in the discussion of dental enamel hypoplasia. This implies that the Khok Phanom Di population was faced with a ubiquitous health problem during childhood. The aetiology of this is discussed further in chapter VII.

Evidence from children

The evidence of health from individuals who died in childhood is biased as an indicator of the health of the living population. In conjunction with the evidence from adults, however, the data can be useful in describing the pattern of ill health in the group.

There is evidence for either skeletal or dental episodes of growth disruption from most children old enough to be tested. The pattern of growth disruption in children appears to differ from that in those who survived to adulthood, with skeletal disruption less prevalent in the first year of life and more common in the second year, although this may be a reflection of the method used in calculating the age of formation of the Harris lines. The dentition shows that linear enamel hypoplasia was less common in children, although prevalence of non-linear hypoplasia is similar in both children and adults. The lower prevalence of linear enamel hypoplasia is unexpected, as the teeth of children are less worn than those of adults and hypoplasia formed early in life is less likely to have been removed by attrition. This suggests that death in childhood is not necessarily evidence of chronic ill health. An exception is B137, who had multiple episodes of skeletal growth disruption in the three years before death. The majority of episodes of growth disruption identified by defects in deciduous teeth occurred pre- or perinatally and therefore reflect either maternal health or the trauma of birth. That the defects are minor and relatively few individuals were affected compared with infants in other populations has two possible explanations. The cause of the growth disruption may have not been widespread but it is more likely that the prevalence is a reflection of the high neonatal death rate, with those who were less healthy not having survived beyond birth.

Correlation of evidence of childhood health from adults and children

The evidence of health during childhood from both adult and child skeletons is similar in some respects. The predominance in adult skeletons of evidence of episodes of growth disruption in the first year of life coincides with the high proportion of child deaths which occur in that age group. The ages of the second peak of growth disruption in the form of Harris lines in adults also coincides with the second peak of child mortality at about 8-12 years, and it is possible that the non-linear dental enamel defects in adults also occurred at about this age. The dental evidence of growth disruption in the form of linear enamel hypoplasia does not correlate well between adults and children or with other indicators of ill health.

ADULT HEALTH

Longevity

Age at death is the primary indicator of adult health. In contrast to the death of a child, which is unequivocally an abnormal event, the death of an adult, particularly in the older age groups, has an element of inevitability. The relatively early ages at death of most prehistoric groups suggest that most clinically-recognized features of senescence would not have affected prehistoric people, but the concept of aging is relative to the context in which it is being discussed. Acsádi and Nemeskéri note in their discussion of human mortality that a maximal life span is that "which could be derived from the longest individual life span found under specific conditions" (1970:14). Age at death as an indicator of adult health in prehistoric groups is most validly viewed as longevity, or the extent to which the expected maximum life span has been achieved by each individual. Most studies of prehistoric groups use age at death as an indicator of group health, using the mean age at death of the group or mortality figures calculated in life tables. As discussed in chapter II, these tables introduce a number of assumptions about the demographic characteristics of the group being studied and because of this, the raw data on individual age at death should also be considered in the analysis of health. Although the identification of specific cause of death in an individual is rare, epidemiology may offer clues through patterns of health in explaining mortality patterns.

The problems and methods of assessing age at death have already been discussed in some detail in chapter II. The results of the individual age estimates are listed in table 13, and the data presented in the form of a histogram and life tables (Fig. 13 and Table 9). Mean ages at death are also given. Overall adult longevity is discussed briefly in the context of demography in chapter II.

Sex differences in frequencies of deaths in five-year age groups are shown in figure 44 and the life expectancies calculated in the life tables are shown graphically in figure 45. These illustrate slight sex differences in adult mortality patterns, with females living on average longer than males. The difference is significant at the <10 per cent level ($p=.094$). All but two males died by the age of 40 years, whereas eleven females survived at least to this age, and seven beyond 44 years.

The age and sex composition and representativeness of the samples from other prehistoric Thai sites has been discussed in chapter II. Mean ages at death calculated from the samples in which the estimation of age is in 10-15 year age groups are not directly comparable with the mean ages calculated from samples where ages are allocated in single years. For this reason, and because of inter-observer variation in methods of age estimation, comparison of longevity among sites is unfortunately less valid than is desirable. Table 61 lists mean ages at death (adults aged 17 years or greater) which are relatively comparable as they have all been calculated using the median of the 10-15 year age groups used by Pietrusewsky.

These data show that the mean age at death at Khok Phanom Di is lower than the mean ages at death at prehistoric sites in North East Thailand, but slightly higher than the other Central Thai site of Ban Kao. The mean ages at death were significantly different from Khok Phanom Di only at Non Nok Tha. At some sites females lived longer than males and at others the males lived longer, although with the exception of Ban Na Di ($p<.05$), none of the within-site sex differences is sta-

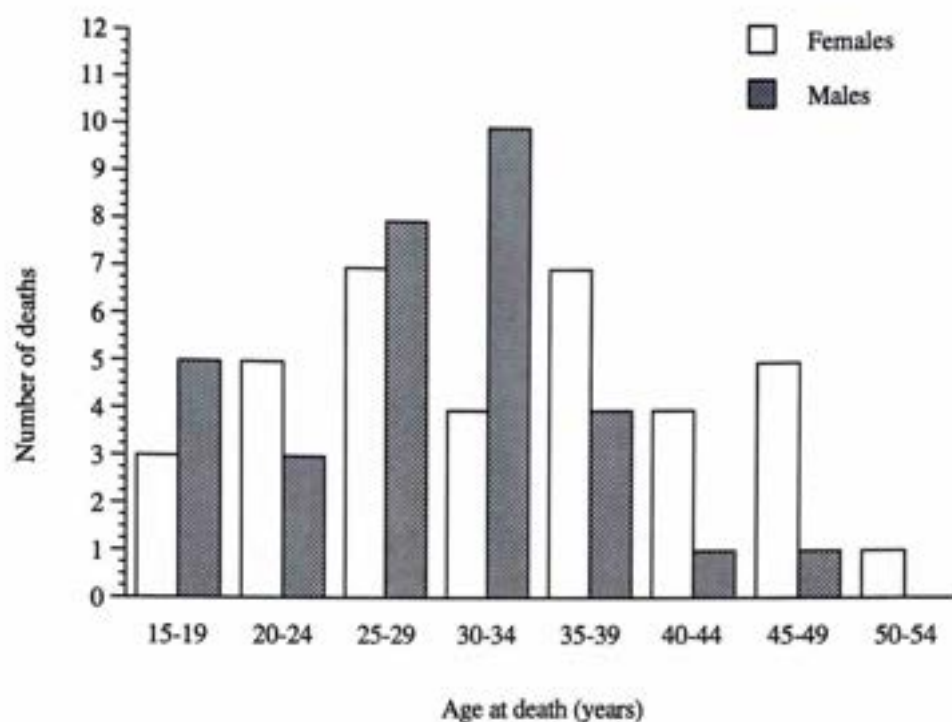


FIG. 44. Age at death of adults

TABLE 61: Mean age at death of adults (age > 16 years) of prehistoric Thai

	All adults				Females				Males			
	n	Mean	s.d.	Signif. ¹	n	Mean	s.d.	Signif.	n	Mean	s.d.	Signif.
Khok Phanom Di	66	31.39	8.54	-	36	32.13	9.32	-	30	30.50	7.56	-
Ban Na Di	33	32.71	10.50	ns	15	37.33	11.05	ns	18	28.86	8.51	ns
Non Nok Tha	92	34.16	8.05	<.05	39	33.87	8.37	ns	50	34.40	7.93	<.05
Ban Kao	29	29.52	8.87	ns	10	28.50	7.25	ns	13	30.73	9.43	ns

¹Statistical significance of difference between each site and Khok Phanom Di

Data Sources: Khok Phanom Di - this study; Ban Na Di - Houghton and Wiriayaromp 1984; Non Nok Tha - recalculated from data in Pietrusewsky 1974a; Ban Kao - recalculated from data in Sangvichien *et al.* 1969

tistically significant. The males at Non Nok Tha lived significantly longer than at Khok Phanom Di.

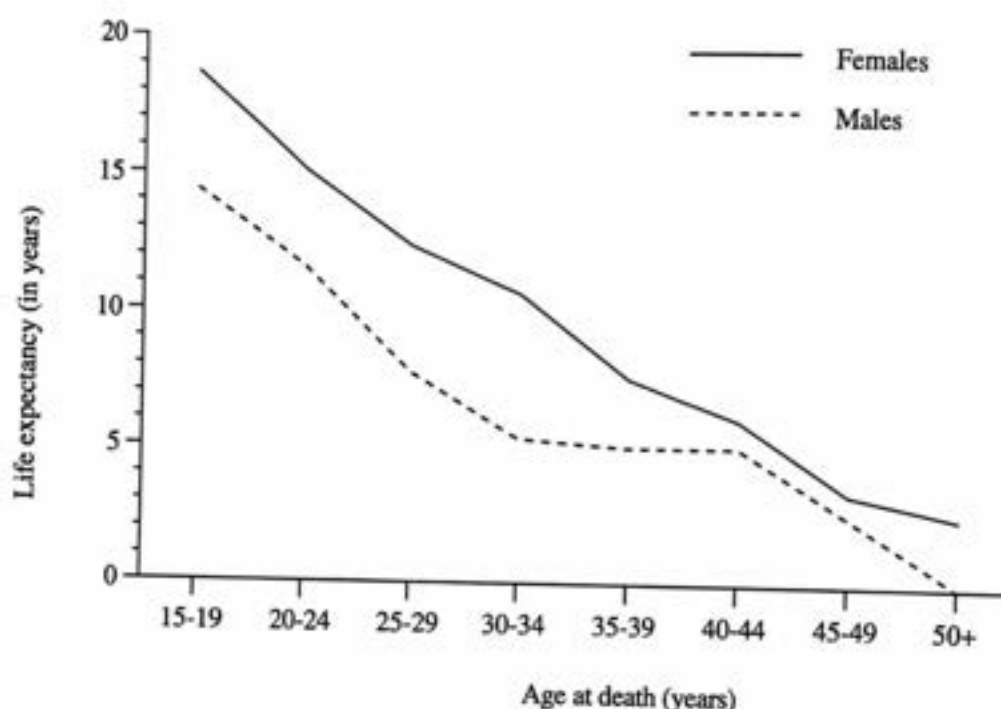


FIG. 45. Adult life expectancy (Ex) for both sexes as calculated in table 9

Cohen (1989: 209), in a review of mortality changes over time, notes that archaeological skeletal populations from all time periods have a strong tendency to a mean age at death in the thirties. Whether this is a reflection of the true ages at death of prehistoric peoples or of the biases of those estimating age at death is not clear.

Another measure of adult longevity, that of life expectancies at age 15 calculated in life tables, is even less directly comparable among the prehistoric Thai populations because of the complications introduced by the more statistically sophisticated nature of the calculations. Life expectancies at the age of 15 among a wide variety of prehistoric populations show that they fall within the ranges of 12 to 35 years (Table 62).

There are few comparative data on modern 'tribal' societies, but the mean and range of life expectancies at the age of 15 in fifteen contemporary hunter-gatherer societies is listed by Cohen (1989:200), and shows a range of 19–49 years, with an average of 28 years. Problems of establishing exact ages of individuals has often had some influence on these data, but the life expectancy is generally higher than the means calculated from skeletal populations. Most other data on life expectancy in modern populations are reported as life expectancy at birth. This is influenced by

TABLE 62: *Life expectancy (E) at age 15 at Khok Phanom Di compared with other prehistoric groups. Both sexes combined*

	n ¹	Mean E (15)	Range E (15)
1. Khok Phanom Di	1	16.6	-
2. Mesolithic	4	26.9	15.0-34.8
3. Neolithic	8	19.1	15.0-24.3
4. Hunter-gatherer	4	16.5	15.0-19.1
5. Protoagricultural	22	19.8	15.0-28.7
6. Modern hunter-gatherer	15	c. 28.0	19.0-49.0

¹n = the number of populations

Data sources: 2,3. Acsádi and Nemeskéri 1970, summarised in Weiss 1973a:49; 4,5 Weiss 1973a: 49; 6. Cohen 1989:200. Note: Weiss (1973a) has set the minimum E(15) value to 15 years, as he does not believe it is possible for a population to have survived any length of time with a lower adult life expectancy

infant and child mortality. The complications of large population sizes with increased exposure to infectious diseases often compounded by malnutrition means that these data have little relevance to the experience of small, isolated, prehistoric societies. Life expectancy at the age of 5 at Khok Phanom Di is close to the lower end of the ranges listed in table 61, implying a relatively short lifespan compared with many prehistoric peoples.

Fertility

The ability and willingness to reproduce is a fundamental indicator of the 'health' of a population in the broadest sense of the term, with both fecundity and fertility reflecting a wide range of genetic and environmental factors, including psychological well-being. Low levels of fertility may reflect biological or socio-cultural limitations on conception and successful gestation and parturition. High levels of fertility indicate that the health of the population was sufficiently good to enable a high level of reproduction, regardless of the encouragement given by socio-cultural factors.

The biological limitations include the reproductive lifespan of females. This period starts with the onset of ovulation, after menarche and a period of adolescent sterility, and ends with the cessation of ovulation at menopause, or, in many prehistoric groups, at death at an age prior to the onset of menopause.

During this period, a number of factors can act individually or in concert to cause the cessation of ovulation and a reduction in fertility. These include physiological consequences of childbirth which in part determine the length of the birth interval, including the period of pregnancy itself and the postpartum infecundable interval which is primarily a result of lactational amenorrhea (Bongaarts and Potter 1983). Other causes of subfecundity include poor nutrition, which can delay the onset of menarche, or, in the case of acute starvation, such as in a period of famine, cause amenorrhea (Bongaarts and Potter 1983). Disease and high levels of physical activity can also re-

duce fecundity through amenorrhea (Frisch and McArthur 1974, McFalls and McFalls 1984, Glass *et al.* 1987, Schweiger *et al.* 1988). All these factors may interact to reduce fecundity, although their effectiveness varies with individual characteristics such as age. The speed of onset and duration of the stressor also influences the effect on fecundity (Bongaarts and Potter 1983). Levels of foetal, perinatal and infant mortality will affect the length of postpartum and lactational amenorrhea and the birth interval.

Influences on fertility other than those directly affecting the endocrinological function of females of reproductive age include socio-cultural factors affecting conception such as the frequency of intercourse and use of contraception. The amount of time during the reproductive lifespan which the females are involved in sexual activity can be affected by cultural practices such as controls on the age of first intercourse, marital practices, and availability of partners for widows, and psychological and biological factors such as the health of both partners. Levels of male infertility will also affect conception rates.

Apart from the complexity of factors involved in determining fertility levels, which makes its interpretation as an indicator of health equivocal, the methods of estimating fertility, both from individuals and at the population level, are not necessarily reliable.

The method of estimating individual fertility used here is from the alterations to the pelvis as a consequence of pregnancy. The aetiology of these 'parturition pits', the methods of recording and the results have already been discussed in chapter II. All females over the age of 17 have 'pregnancy pits', indicating a high level of fertility.

Maintenance of bone mass

Bone dynamics during adulthood have received a lot of attention in living populations over recent decades, largely because of the increasing prevalence of osteoporosis and consequent fractures in elderly women. In a healthy adult, the period from cessation of growth until about 35 years of age is a period of consolidation of the skeleton. Skeletal maturation is completed during this time with the fusion of the last epiphyses and skeletal elements such as the sternum and the anterior bodies of the sacral vertebrae. Skeletal mass increases both through apposition on endosteal and periosteal surfaces of cortices and through decrease in porosity (Garn 1970, Parfitt 1988).

Peak adult bone mass, representing the summation contributions of growth (90–95 per cent) and consolidation (5–10 per cent) is reached at about age 35–40 for cortical bone and probably earlier for trabecular bone. (Parfitt 1988:50)

Soon after this age, bone mass begins to reduce in the phenomenon described as 'age-related' bone loss. This is apparently part of the universal biological aging process, which acts through decreased osteoblastic activity. With continued bone resorption, mean loss from all sites in the skeleton in females from about the age of 35 and in males from age 40–45 is 0.3 per cent of peak adult bone mass per annum. This loss accelerates in females from about 45 years, with the perimenopausal reduction in oestrogen levels, to about 1.0 per cent per annum (Mazess 1982, Parfitt 1988). This accelerated rate of loss continues until about 70 years of age (Parfitt 1988). There are wide variations between individuals in this progression of bone mass increase and decrease (Parfitt 1988) as the result of a combination of factors. Genetic potential and biomechanical stress are

the primary determinants, and hormones, nutrition and disease are secondary, mediating factors (Heaney 1988).

Of the genetic factors, sex is the primary factor in inter-individual variation, with peak adult bone mass being about 25-30 per cent higher in males than in females (Parfitt 1988). This will in part reflect size, as larger individuals have higher bone mass, but will also reflect the variation which is apparent from infancy (Garn 1970). There are also suggestions of a genetically mediated variation between groups such as Blacks and Whites in North America, with the former having higher bone mass at all ages (Heaney 1986), and Chinese and Japanese living in the United States of America, who were recorded as having lower cortical thicknesses than Whites in equivalent socio-economic groups (Garn *et al.* 1964a). The principal action of genetic potential for lower bone mass is to lower peak adult bone mass.

The importance of biomechanical stress on the maintenance of bone mass is illustrated in cases of immobilization following surgery or trauma, and weightlessness during space flights, where increased loss of bone mineral results in loss of bone mass (Sinaki 1988). Wolff's Law states that form follows function in bone (Wolff 1986), and this is demonstrated, for example, where there has been a specific increase in the bone mass of the dominant arm of tennis players and favoured leg of athletes (Lane *et al.* 1988, Sinaki 1988). Physical exercise, particularly when it is long-term, can maintain and improve bone mass, even in the event of nutritional problems. Anorectic individuals who have regularly exercised do not lose more bone than controls (Lane *et al.* 1988). However, Plato and Norris (1980), in a study of grip strength and measurements of the second metacarpal on a living population, found a positive correlation with cortical thickness but not with medullary width, which suggests that achievement of maximum potential bone mass during the period of growth and early adulthood may be affected by biomechanical factors, but that age-related bone loss is not influenced.

Diet and nutrition influence bone mass, both through failure to achieve genetic potential during the period of growth as was discussed in the section on adult stature, and through variation in the rate and timing of the pattern of increase and decrease in adults. Nutritional deficiencies which have received most attention in clinical studies are those relating to calcium. Heaney (1988) notes that the evidence for high levels of calcium intake maintaining bone mass is equivocal. It appears that bones respond differentially to calcium uptake, and that individuals have differing levels of calcium requirements in bone mass maintenance. Phosphorus, especially in proportion to calcium, vitamins D and C and trace elements such as manganese, copper, and zinc are all also potentially involved in skeletal mass maintenance. Garn (1970) notes that more generalized protein-calorie deficiencies will result in acceleration of loss of bone mass in adults. In the presence of protein-calorie malnutrition, childhood growth in height will be maintained at the expense of growth in bone mass, even to the extent of withdrawal of mineral from the cortex.

Apart from metabolic disorders such as rickets and scurvy which reflect nutritional deficiencies, diseases which affect bone mass include some of the hormonal and haematopoietic diseases (Ortner and Putschar 1981).

The extent to which failure to achieve genetic potential for bone mass during the growth phase can be made up during the adult consolidation period is not clear. Briers *et al.* (1975) and Graham *et al.* (1982) record that catch-up growth in cortical thickness will occur if there is recovery after

chronic and severe undernutrition during infancy. Long-term nutritional deficiencies may predispose to lesser peak adult bone mass and greater relative age-related loss.

The measurement of bone mass in comparisons among modern populations and prehistoric groups requires consideration of all the factors implicated in the modification of formation and remodelling rates. The age structure of the populations is consequently of primary importance, as is diet, levels of habitual physical activity and morbidity rates. As most adults in most prehistoric populations appear not to have lived beyond the age at which peak adult bone mass is reached in modern adults, it is possible that the age-related bone loss, including the post-menopausal loss in women, which is such a problem in modern health, did not affect prehistoric groups to any great extent. The results of some studies suggest that the pattern and timing of loss differs in prehistoric populations (Dewey *et al.* 1969, Ericksen 1982) but a number of potential variables, including problems with skeletal aging, cannot be discounted in these cases. Whether the lower cortical thicknesses in the prehistoric populations involved represent a lower peak adult bone mass rather than a greater rate of loss of bone needs to be considered. This could reflect genetic factors, nutritional or pathological stress during growth and adulthood, or biomechanical factors rather than degeneration with biological aging.

Trabecular bone, particularly in the vertebral bodies, is first affected by inadequate bone formation or increased bone resorption (Arnold *et al.* 1966, Johnston *et al.* 1985), but cortical bone is most commonly used for the assessment of bone mass in skeletal collections as it is usually better preserved and allows more precise measurement. The processes of bone remodelling involved in the maintenance of adult cortical bone mass vary in the rate of activity across the thickness of the cortex. During periods of bone loss, least resorptive activity occurs near the periosteum, and the rate increases towards the endosteal surface. The greatest activity takes place at the endosteal surface and in resorptive phases this results in the connection of the resorption spaces with the marrow cavity, with the effect of increasing the width of the marrow cavity and thinning of the cortex. Throughout life, even during periods of resorption, there is very slight apposition of bone on the periosteal surface which helps to maintain the mechanical integrity of the bone. Remodelling of intracortical bone, which increases bone porosity, requires histological examination of bone, but the thickness of the cortex can be simply measured from antero-posterior radiographs. This can then be normalized for body size by relating cortical thickness to the diameter of the shaft (Barnett and Nordin 1961).

It has been suggested that the proportion of the bone cross-section occupied by cortical area is a better descriptive measure (Martin and Armelagos 1979). Garn (1970) offers a formula for calculating per cent cortical area in second metacarpals from cortical thickness, but this assumes that the bone is round, an uncommon occurrence in prehistoric bones. The alternative is direct measurement of the bone. Van Gerven *et al.* (1969) also advocate direct rather than radiographic measurement on the grounds that errors can occur in the latter through the inability of the radiograph clearly to reveal endosteal porosity, and that the assumption must be made that the medio-lateral cortical walls exposed on the radiograph represent the mean cortical thickness for the entire bone. However, direct measurement of cortical area requires cutting of the bone to expose the full diaphyseal cross-section, and this is not necessarily practical nor desirable in a skeletal series. Recent developments of non-invasive methods of measuring cortical thickness include, besides radiogram-

metry, single and dual photon absorptiometry, computed tomography and other methods of nuclear medicine to measure bone density (Mazess and Wahner 1988). The application of these methods involving high technology is less practical in the study of archaeological collections than the simpler radiogrammetry. A comparison of methods on living individuals by Wishart *et al.* (1993) found that the calculations based on radiographs produced comparable results with those calculated by more sophisticated methods.

TABLE 63: *Cortical indices measured from radiographs of the second metacarpals in both sexes*

	Females		Males	
	Left	Right	Left	Right
n	26	19	21	20
Mean	59.22	59.15	63.01	63.48
Standard deviation	10.08	10.66	7.56	8.61

The bones most commonly used for the assessment of cortical thickness are the metacarpals and radius in clinical studies (Heaney 1986) and femur and metacarpals in archaeological studies, although the ribs, tibia and humerus have all been used. (Dewey *et al.* 1969, Van Gerven *et al.* 1969, Ericksen 1976, Cook 1979, Martin and Armelagos 1979, Simpson 1979, Frank 1981, Saitta 1981, Pfeiffer and King 1983, Cassidy 1984, Wiriayaromp 1984a). The variation in remodelling rates among different bones negates comparisons of data other than those using the same bone.

The thickness of the cortical bone in the Khok Phanom Di skeletons has been assessed radiographically using the second metacarpal. The metacarpal was used as, compared with the femur, tibia and radius, it is the least variable bone in cross-section and the measurements from the medio-lateral radiograph are more likely to give a closer representation of the entire cortical area. It is also relatively easily radiographed in large numbers because of its small size. The second metacarpal has been used because it is morphologically less variable than the other metacarpals (Garn 1970), and comparative data are available from other archaeological and modern clinical studies (Skrobak-Kaczynski and Lange-Anderson 1974, Saville *et al.* 1976, Simpson 1979, Cassidy 1984).

Measurements of the thickness of the cortex at the midshaft followed the method described by Garn (1970). Cortical thickness is derived from the equation: $C = T - M$, where C = cortical thickness, T = total bone diameter and M = medullary cavity width (Garn 1970). A method of obtaining comparative data by normalizing cortical thickness for bone diameter was calculated by Nordin (Barnett and Nordin 1961). It is calculated using the equation: $C/T = ((T - M) / T) 100$.

An initial series of metacarpals, radiographed by McGill (1986), were subsequently supplemented by a second series. Radiographs were repeated for a sample of bones to provide a comparison between the two series. The radiographs were taken on fine-grained paper with the metacarpals in the antero-posterior position. Measurements of bone length and calculation of midshaft point were made using graph paper over the radiographs on a light table to ensure accuracy. Measure-

ments at the midshaft on the first series were made manually, using fine-point dial calipers graduated to 0.05 mm. The second series were measured twice, once using the dial calipers and the second time using a Zeiss MOP-3 electronic Digitiser, with the tablet placed over a light table. The measurements subsequently used in the analysis were those made using the digitiser, as this method provided the most consistent results. The radiographs from the first series of metacarpals were re-measured using the digitiser to ensure comparability.

Individuals with the distal epiphyses fused were used, as the bone needed to be complete to measure length and obtain the midshaft point. This also ensured that the sexually dimorphic changes in cortical thickness during the growth spurt at puberty (Garn 1970) would not introduce another level of complexity into the interpretation of the results. Both left and right metacarpals were radiographed where these were available.

A total of 86 second metacarpals from 55 adults (29 females, 26 males) were radiographed. Both left and right metacarpals were available for 31 individuals, and the remainder comprised 16 left bones and 8 right bones. Table 63 lists the mean and standard deviations of the cortical indices (Nordin scores) for both sexes. Individual data are listed in table 66. The variation in the cortical index between right and left bones from the same individuals is not significant in either sex or with the sexes combined. Mean scores of left and right bones for each individual were used in the analysis. The difference between male and female scores is not significant.

Figure 46 illustrates individual cortical indices in relation to age at death. Regression analysis shows an apparent decrease with age which is significant in females ($p=.005$) but not in males.

As the second metacarpal is commonly used in radiographic assessments of cortical thickness, there is a wide range of data available for comparison, both from skeletal and modern populations, including a small sample from Ban Na Di. These data are summarised in table 64. In both sexes at Khok Phanom Di the mean cortical thickness is in the upper range compared with both skeletal and modern populations, particularly in the younger individuals. As the standard deviations are high, there are clearly individuals with cortices as thin as some of the other prehistoric skeletal samples, but the evidence suggests that the group as a whole had good cortical bone structure.

Some problems with the measurements from the radiographs were encountered, as in some cases the shafts of the bones were not cylindrical but varied in shape with variation in the development of the ridges marking the attachments of the dorsal interosseous muscles. The angle at which the bone rested during the exposure of the radiograph appeared to have the potential to influence the extent to which the development of the muscle attachments, rather than the basic cortex, was represented in the image. The definition of the endosteal surface, particularly in older individuals where resorption was evident, also caused some problems in the measurement of the medullary cavity. The question remains as to the extent to which the deviation of the shaft from the cylindrical shape assumed in using the radiographic measurement influences the measurement. In a study of a series of modern metacarpals, Horsman and Kirby (1972) found the assumption that the shaft was round to be valid. In a series of radiographs from Norwegian timber workers, Skrobak-Kaczynski and Lange-Andersen (1974) found that the outer diameter of the shaft increased with age and that there was a slower rate of endosteal loss in these men than in Americans and Finns of the same ages. They concluded that this was brought about by the stimulation of hard physical work. Plato and Norris (1980) found a correlation between grip strength and total width of the shaft, cortical thickness, and

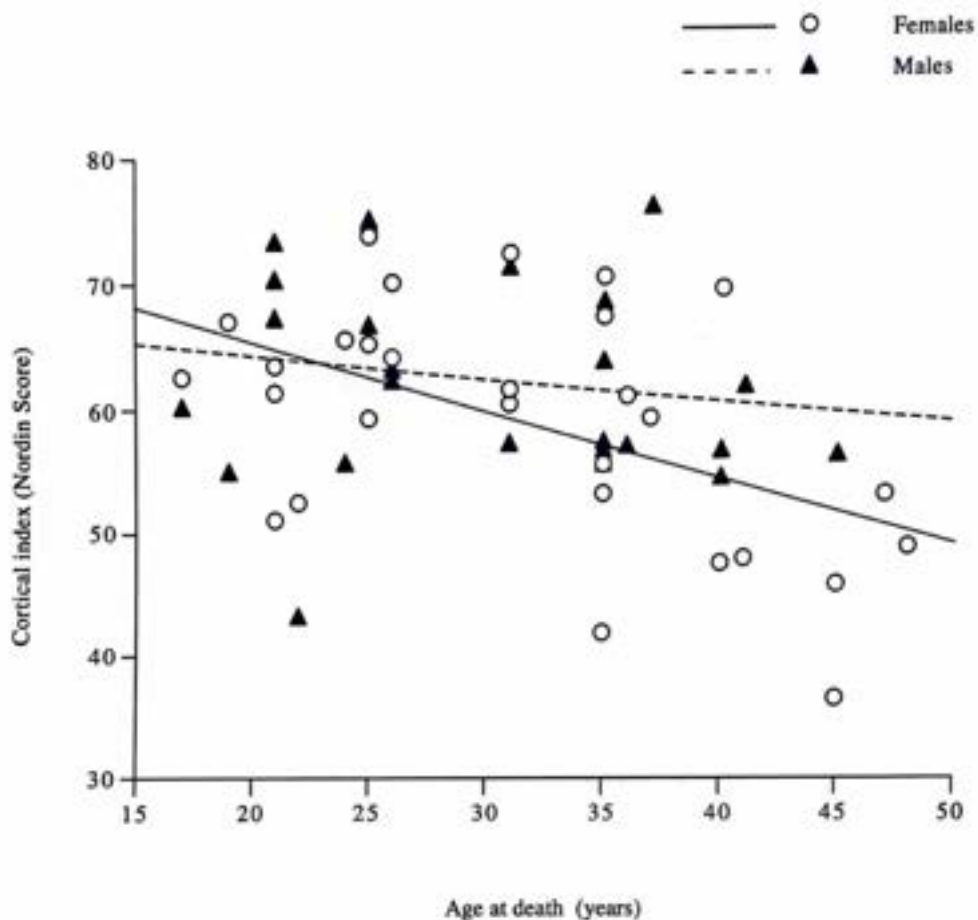


FIG. 46. Relationship of cortical thickness index (Nordin's score) with age in adults

cortical area. This they also attributed to the enhanced development of the bone through physical activity exerted early in life. In prehistoric peoples engaging in a high level of physical activity, both the shape of the shaft of any long bone and the thickness of the cortex may be affected to the extent that the bone does not reflect systemic bone growth and maintenance as a result of reduction in susceptibility to osteoporosis. The evidence of good cortical thickness in the Khok Phanom Di people and the well developed musculature may therefore reflect both strenuous physical activity involving the use of the hands and good systemic skeletal development, with the absence of nutritional deficiencies or illness which could have caused premature osteoporosis. The measurement of cortical area from sections of several bones from each individual would clarify this question.

SUMMARY - ADULT HEALTH

The good bone cortices in both sexes and universal fertility in the females suggests good overall health in adults. Despite this, they did not live as long as other prehistoric South East Asians. The apparent contradiction of these indices is discussed further, in conjunction with the evidence of pathology, at the end of chapter VII.

SUMMARY - ADULT HEALTH

The good bone cortices in both sexes and universal fertility in the females suggests good overall health in adults. Despite this, they did not live as long as other prehistoric South East Asians. The apparent contradiction of these indices is discussed further, in conjunction with the evidence of pathology, at the end of chapter VII.

TABLE 64: *Cortical thickness expressed in the form of the cortical index (Nordin Score) at Khok Phanom Di compared with other skeletal populations and ethnographic and historical data*

	Bone used	n	Age	Nordin Score	s.d.
Females, prehistoric skeletal					
1. Khok Phanom Di	Second metacarpal	29	all	59.0	9.6
		12	<30	63.1	6.5
		10	30-39	60.5	9.0
		7	>40	49.9	10.1
2. Ban Na Di	Second metacarpal	2	30-40	68.9	-
		2	'adult'	58.9	-
3. Namu	Second metacarpal	19	20-50	61.6	-
4. Maori	Second metacarpal	36	-	49.2	6.8
5. Indian Knoll	-	-	17-29	51.0	-
		-	30-49	36.0	-
6. Hardin Village	-	-	17-29	37.0	-
		-	30-39	33.0	-
Modern					
7. UK	Second metacarpal	-	20-40	63.0	-
8. US white	Second metacarpal	-	20-40	64.0	-
1. Khok Phanom Di	Second metacarpal	26	all	62.5	8.1
		12	<30	64.1	9.5
		12	30-39	61.6	7.1
		2	>40	59.2	4.0
2. Ban Na Di	Second metacarpal	4	17-29	61.4	-
		2	30-39	64.1	-
3. Namu	Second metacarpal	31	20-50	59.7	-
4. Maori	Second metacarpal	27	-	52.9	8.9
5. Indian Knoll	-	-	17-29	51.0	-
		-	30-49	47.0	-
6. Hardin Village	-	-	17-29	37.0	-
		-	30-49	37.0	-
Modern					
9. Norwegian (lumbermen)	Second metacarpal	28	20-29	57.0	-
		51	30-39	57.8	-
		85	40-49	55.9	-
		99	50-59	53.2	-
		49	60-69	52.1	-
10. Finnish (farmers)	Second metacarpal	-	20-40	63.0	-
(urban)		-	20-40	58.5	-
11. US whites	Second metacarpal	-	20-40	62.5	-
12. Guatemalans	Second metacarpal	-	20-40	63.0	-

Sources of data: 1. This study, mean of left and right in individuals with both metacarpals present; 2. Wiriyaromp, unpublished data, Anatomy Department, University of Otago Medical School; 3. Houghton, unpublished data (cited in Simpson 1979); 4. Simpson 1979; 5,6. Cassidy 1984; 7. Nordin, unpublished data (cited in Garn 1970); 8. Saville *et al.* 1976 (cited in Heaney 1988); 9-12. Skrobak-Kaczynski and Lange-Andersen 1974

TABLE 65: *Harris lines recorded from radiographs of individual adult distal tibiae, by age at formation. The numerals indicate the number of lines recorded at each age*

		Age (years) at which lines are calculated to have formed																Total	
	Age	0	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	Total
Females																			
4	26	1																	1
25	19			1															1
26	35	1								1	2	1	2						7
31	25			1	1	2	1	1	1	1		2	1						11
35	21							1		1	2	1							5
36	21	1																	1
39	25	1																	1
45	45	1								1	2								4
47	22	1																	1
64	21	1							1		1		1						4
73	25			1															1
77	26	1																	1
79	47	1																	1
83	30		1																1
87	24	1	1												1				3
102	35					1													1
107	40			1			1	1	1	1					1				6
109	31		1										1	1					3
110	36	2									1		1						4
142	31			1															1
Males																			
8	15	1																	1
9	30														1				1
22	21															1			0
23	30	1		1											1				3
24	25	2										1							3
28	21		1									1			1				3
30	34			1															1
38	33											1							0
42	32																		1
44	18											1							1
67	35		1																1
72	32		1												1				2
76	34	1													1		1		3
90	27	1																	1
92	18	1			1														2
93	42	3			1											1			5

TABLE 65: *Harris lines recorded from radiographs of individual adult distal tibiae, by age at formation. The numerals indicate the number of lines recorded at each age (cont.)*

		Age (years) at which lines are calculated to have formed																	Total
	Age	0	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	
Males																			
100	32	1																	1
103	26	1																	1
115	38	1																	1
117	16	1											1						2
120	21				1														1
129	27	1													1	1			2
131	25	1		1							1								3
132	34	1														2			3
Children																			
14	1.25																		0
20	12	1																	1
32	11		1																1
62	0.50																		0
63	1.75		1																1
88	0.75	2																	2
96	4		1																1
123	2		2																2
137	5		1	2	2														5
145	8	2	1																3

TABLE 66: *Individual cortical index (Nordin score) of the second metacarpals of adults*

Females			Males		
Burial	Left	Right	Burial	Left	Right
4	70.27		9		57.47
13		67.56	22	66.27	74.71
15	70.73	70.73	23	60.54	61.18
19	58.33	60.49	24		75.00
26	45.07	38.89	28		43.37
27		48.00	30	69.05	68.60
35	61.44		38		64.04
36	49.35	53.01	42		57.61
39	72.97	75.00	44	55.12	
40	59.42		57	65.98	67.90
45	37.34	35.71	67	54.74	59.77
47	53.93	51.28	72	72.34	70.83

TABLE 66: *Individual cortical index (Nordin score) of the second metacarpals of adults (cont.)*

Burial	Females		Burial	Males	
	Left	Right		Left	Right
56	45.83		74	61.17	48.10
58	55.71		90	65.82	60.92
60	67.08		91	56.38	
61	44.29	53.33	92	55.22	65.38
64	63.64	63.51	93	60.00	63.95
73	65.38		100	58.97	54.88
77	60.25	68.35	103	76.32	74.35
79	53.09		115	56.82	
87		65.71	120	65.88	68.97
94	73.61	71.62	129	59.46	65.52
102	53.33		131	54.12	57.61
107	47.62		132	55.79	
109	62.96	58.23	147	76.32	
110	60.61	61.76	152	76.39	
112	73.24	66.20			
140	67.11	58.11			
142	67.12	56.41			

V. SKELETAL PATHOLOGY

THIS chapter considers skeletal evidence for disease. As discussed in chapter IV, skeletal pathology reflects abnormal rates of cellular interaction in bone. Figure 47 is a diagrammatic representation of the causes and effects of processes that may produce skeletal pathology. Pathology is defined as 'the structural and functional manifestations of disease' (Anderson 1989) and is not used here as a synonym for disease.

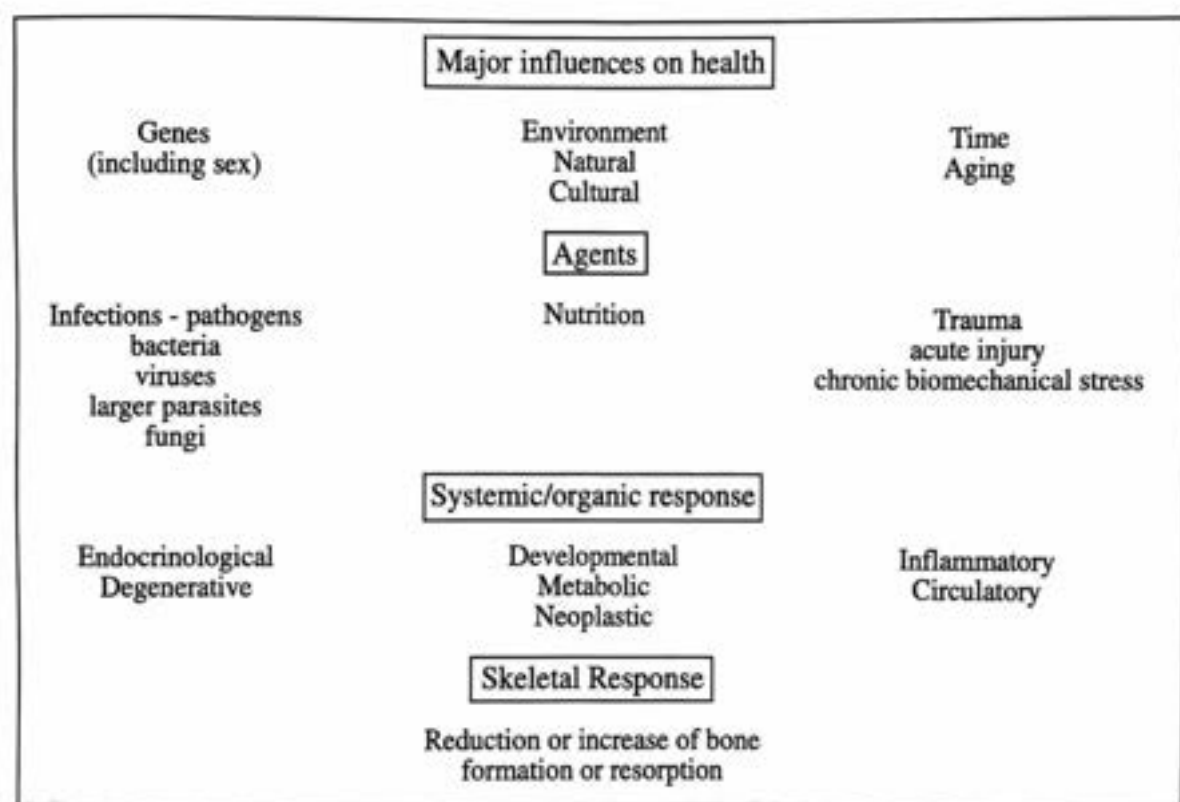


FIG. 47. Model of skeletal pathological processes

The trail starts at the major influences on the individual (or the population) which can result directly in disease or predispose susceptibility to disease. The agents are those factors that can produce a developmental defect, or produce a tissue response which is circulatory, inflammatory, neoplastic or metabolic. When the skeleton is affected, the response is reflected in alteration in the rates of bone formation or resorption. The reaction may be systemic or local (Ortner and Putschar 1981).

The first step in identifying reactive bone is to distinguish abnormal from normal morphology. The range of variation in normal bone morphology will depend on the population being studied. Individual skeletal responses will vary depending on such factors as age, sex, pre-existing state of health, and genotype. In the initial recording of skeletal evidence of health, it is necessary to have some understanding of the range of morphological variation which constitutes 'normal' in the population being studied, and for each sex and at different ages. Wells (1967a:12) refers to the 'morphological uncertainty' which arises when there is an inadequate knowledge of the range of normal morphological variation. This includes variation in morphological response to biomechanical stress from repetitive or strenuous physical activity. The point at which such remodelling becomes pathological cannot necessarily be objectively defined. Any 'pseudopathology' created by post-depositional processes must be identified and excluded from further analysis.

The next step is to describe the remodelling, with the aim of identifying the process involved. Precise description of the following provides a basis for this:

- Location on the skeleton - axial, appendicular, or on specific bones.
- Location on the bone - periosteal, endosteal, intracortical.
- The type of bone involved - cancellous, cortical.
- Description of the bone cell reaction - bone formation, resorption or a combination of the two.

The third step is to attempt to identify the disease involved. This is based primarily on clinically recognised skeletal pathology, supplemented by reference to the interpretation of patterns observed in other skeletal collections. Differential diagnoses are considered. The validity of the use of clinically recognised evidence in diagnosis depends on the assumptions that skeletal reactions are the same as those resulting from modern diseases, that there has been no evolution of diseases such that the condition no longer exists and is not known clinically, that observed symptoms may represent terminal stages no longer common because of therapeutic intervention and that more than one disease is involved (Klepinger 1983).

The identification of the disease involved (where this is possible) is an intermediate step in the present analysis. The final steps are identification of the factors involved in producing the conditions which allowed the disease to exist in the population and interpretation of the effects of the disease on individuals and the population. Active lesions, indicating active disease at the time of death, are differentiated from remodelled lesions which may reflect recovery from a disease. The epidemiology of the diseases in the population is discussed in conjunction with the results of the analysis of health in chapter VII.

GENERAL PATHOLOGY

The identification of pathology involved the meticulous examination of all surfaces for any reactive bone and remodelling, and recording of the distribution of the changes and their nature. The examination was supplemented by microscopic examination where appropriate. Where a reaction was

visible on the external surface and the internal structure was not visible, radiographs were taken to assist in the diagnosis. Joint degeneration is the most common bone pathology in human populations (Sokoloff and Hough 1985). Because of this ubiquity, the condition of articular surfaces of all adult joints was systematically recorded and is reported and discussed in a separate section.

Much of the pathology other than joint degeneration consists of diverse observations, recorded on single individuals. Pathology recorded on more than one individual is presented first, followed by the individual observations.

The most common non-articular pathology is on the orbital plates of the crania of a number of individuals, where the subperiosteal bone has grossly visible porosity and cribrotic bone formation. Of thirteen children aged between one and fourteen years with the orbital plates intact, ten had the pathology (77 per cent), including seven of the eight children aged over five years. These changes conform with the descriptions of the condition widely known as 'cribra orbitalia' and they were therefore classified using the scale of severity listed by Stuart-Macadam (1985: 392). Two had light, four had medium, and four had severe lesions. None of the children less than one year old ($n = 63$) was affected. Of the adults, three (of 57 with the orbital plates intact = 5 per cent) had lesions graded as light. These individuals, two males and one female, all died by their mid-twenties.* The basis of development of cribra orbitalia and its association with anaemia has been comprehensively demonstrated by Stuart-Macadam (1987b). Anaemia is considered in detail in a separate section later in this chapter. Figure 48 illustrates the cranium of a child with grade 3 lesions.

Pathology on the maxilla was recorded in three adults, all females (Table 67). The anterior (facial) surface of the bone was depressed, reducing the size of the underlying maxillary sinus. In two adults, there is active infection, evidenced by dental abscess cavities with cloacae draining into the maxillary sinus (Fig. 49). The evidence in the third adult suggests a similar cause, with the infection no longer active and the bone largely remodelled. The direct association with adjacent dental infection in two of the three cases suggests that this is the cause of the maxillary pathology.

There is also an infant (B123, 2 years) with a lesion on the anterior surface of the right maxilla, above the fully erupted deciduous first molar (Fig. 50). The surface of the bone is porous and reactive over an area of c. 8 x 10 mm. The roots of the tooth are almost fully exposed and the bone in the area between the root furcation is very porous and was actively resorbing. There is no obvious diagnosis for this lesion other than nonspecific infection. The tooth is not carious.

Four of the eight children aged over five years had enlarged nutrient foramina on the hand bones (Fig. 51), which suggests increased vascularity. This response has been recorded in cases of genetic anaemias such as thalassaemia, sickle-cell anaemia and variants, and in haemophilia, Gaucher disease, and leprosy (Fink *et al.* 1984, Lawson *et al.* 1984).

Among the adults, B23, a 30-year-old male, has the middle and proximal phalanges fused on one finger. The youth of the person and the lack of degeneration at other joints suggests the fusion is a result of trauma to the joint at an early age, rather than ankylosis as a consequence of age or activity-related joint degeneration. Burial 93, a 42-year-old male, has the middle and distal pha-

*During the initial examination of the adult skulls, using the classification of Hengen (1971) a much higher proportion of the adult skulls was recorded as having 'light' lesions (31/57) on the basis of minor, dendritic grooves in the area of the lacrimal fossa. These were subsequently excluded on the assumption that they represented impressions of blood vessels (lacrimal artery, superior ophthalmic vein) traversing the orbital roof rather than expansion of diploe within the bone.

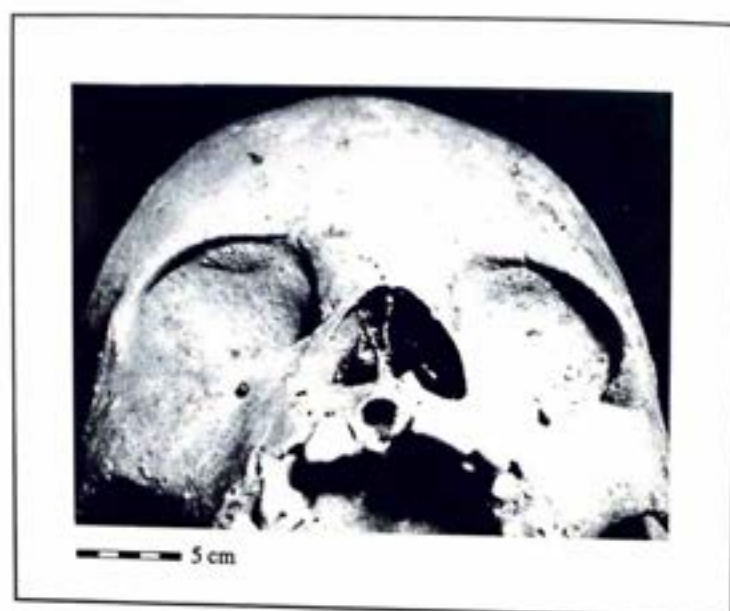


FIG. 48. The cranium of burial 21 (8 years) with cribra orbitalia of grade 3

langes fused on one finger. As this person is older and has severe degeneration on other joints in the hand, the fusion may reflect either trauma or the final stage of joint degeneration with the fusion of osteophytes. The distal joints of the fingers are more susceptible to age-related degeneration than the proximal joints, although females generally suffer from the problem more than males (Resnick and Niwayama 1981).

A number of individuals have single, localized, subperiosteal lesions. Burial 38, a 21-year-old male, has a discrete thickening of the cortex, *c.* 25 mm in length, on the subcutaneous dorsal border distal to the trochlear notch of the left ulna (Fig. 52A). The bone surface is smooth, apart from slight porosity. There is no evidence of pathology on the radiograph other than the thickening of the cortex. Burial 67, a 35-year-old male, has a cortical lesion on the lateral shaft of the right humerus, 30 mm in length and 9 mm in width, running diagonally along the bone just below the deltoid crest (Fig. 52B). The surface of the lesion is smooth, with no reactive bone. Burial 79, a 47 year-old female, has a slight cortical lesion on the lateral surface of the left fifth metacarpal. The surface of the lesion is smooth, with no reactive bone. Burial 129, a 27-year-old male, has a discrete cortical thickening on the subcutaneous anterior border of the right tibia, towards the proximal end of the bone (Fig. 52C). It extends 50 mm along the bone and halfway across the shaft, and is raised 2.5 mm from the subperiosteal surface. The surface of the lesion is smooth, with no reactive bone evident. The most probable basis for such localized, non-active lesions is trauma. All except the lesion on the humerus are on areas where the bone is subcutaneous and therefore vulnerable to trauma. Inflammation or infection of a wound in the thin layer of overlying soft tissue could easily be transmitted to the periosteum and produce periostosis. Resolution of the infection and healing of the soft tissue with remodelling of the bone could result in the long-standing, sclerotic, lesions observed. The

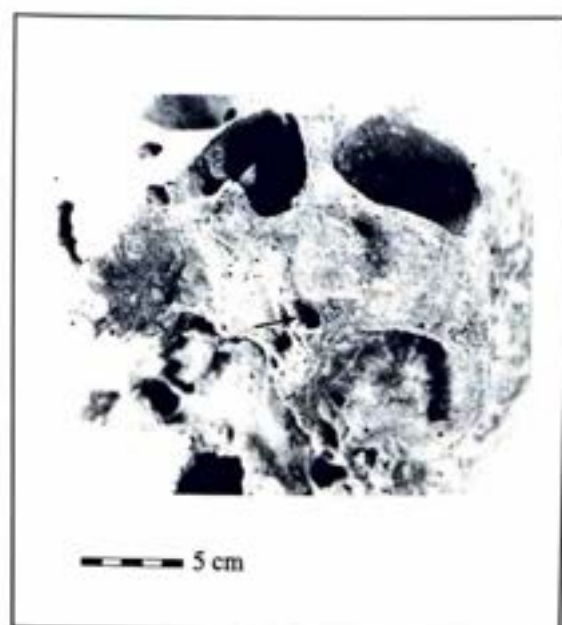


FIG. 49. Face of burial 79, showing gross collapse of the left maxilla over an area 25 x 20 mm, to a depth of 6 mm, below the infraorbital foramen, extending laterally almost to the zygomatic border and medially to the border of the nasal aperture. There is no reactive bone visible on the surface. All the left posterior maxillary teeth were lost during life and the alveolar crest is almost completely resorbed, but a cloaca opening from the alveolar crest into the maxillary sinus remains in the area of the left maxillary premolars (arrow)

thickening on the humerus of B67 would have been overlain by parts of either brachialis or triceps brachii muscles but as it is on the lateral aspect of the arm, inflammation secondary to severe trauma is still the most probable explanation.

As could be expected in a population, there is not only evidence of bone reaction to soft-tissue trauma, but also four cases of healed fractures of bone (Table 68). The clavicle can be fractured following a fall, when the upper limb is used to break the fall and the force is transmitted through the limb to the clavicle (Fig. 53). Burial 110 may have suffered an infection following the fracture, or sustained the fracture because the bone was weakened by infection. Osteomyelitis is suggested by the cloaca, although the small amount of reactive bone present may indicate that the infection was not active.

The rest of this section describes individual cases of pathology. An 18-year-old male (B44) has an area with porous reactive periosteal bone on the external table of the right parietal (Fig. 54). Periostitis can be either primary, associated with trauma or osteomyelitis, or secondary to a systemic disease such as anaemia or one of the treponematoses. It indicates stripping of the periosteum which has stimulated growth of the subperiosteal bone. It does not always indicate inflammation, and as is not restricted to infection, it is difficult to identify the aetiology (Ortner and Putschar 1981). A localized area of periostitis such as this may reflect trauma, such as a blow to the head resulting in

TABLE 67: *Lesions of the maxilla*

Burial	Age	Observation
77	26	Has a shallow depression on the left maxilla, 8 x 13 mm in size, lateral to the infraorbital foramen. There is reactive bone on the surface of the area, and an active periapical abscess on the tooth immediately below. There is a cloaca draining from the abscess into the maxillary sinus.
79	47	Has gross collapse of the left maxilla over an area 25 x 20 mm, to a depth of 6 mm, below the infraorbital foramen, extending laterally almost to the zygomatic border and medially to the border of the nasal aperture (Fig. 49). There is no reactive bone visible on the surface. All the left posterior maxillary teeth were lost during life and the alveolar crest is almost completely resorbed, but a cloaca opening from the alveolar crest into the maxillary sinus remains in the area of the left maxillary premolars.
102	35	Has a depression 12 x 14 mm in diameter, 4 mm deep, on the right maxilla below the infraorbital foramen. There was no reactive bone, and no grossly visible dental pathology.

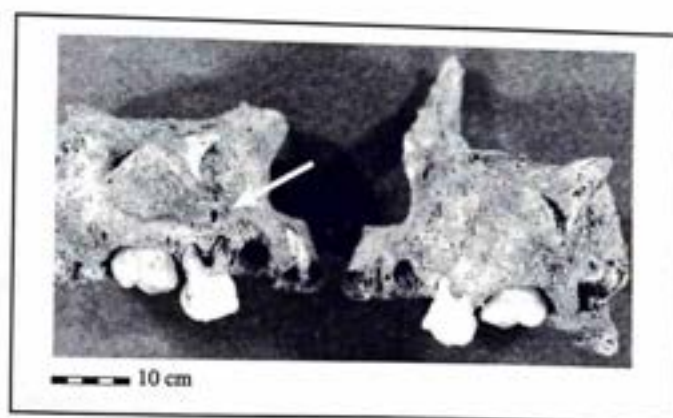


FIG. 50. Maxillae from burial 123 (2 years) with an active lesion on the left anterior surface above the first molar (arrow)

inflammation. There is no evidence of osteomyelitis in the form of changes in the marrow cavity, cloacae or involucra. The alternatives of secondary periostitis are considered later in this chapter, in the discussion in association with all the evidence from this individual.

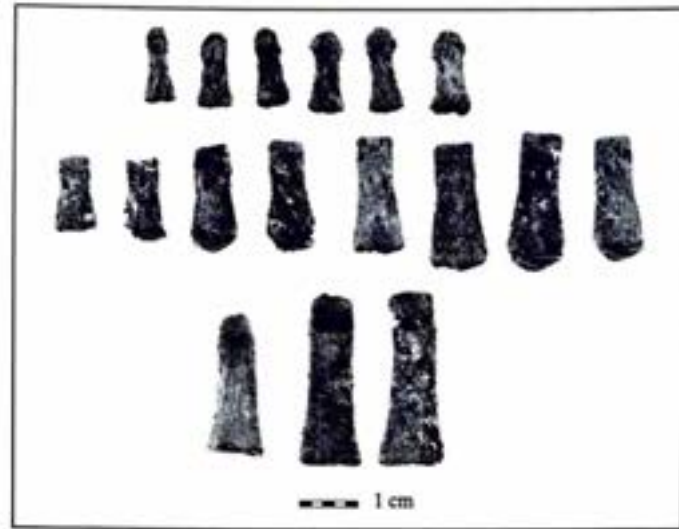


FIG. 51. Palmar view of hand phalanges from burial 37 (9 years) with enlarged nutrient foramina

Burial 67, a 35-year-old male, has a small irregular depression on the external surface of the right parietal, approximately 15 x 20 mm and 3 mm in depth. There is no reactive bone, and no lesion is visible on the inner table. The most likely basis for such an isolated lesion is trauma, with healing of damaged tissues.

On B21, an eight-year-old, several of the middle and distal hand phalanges and the fifth metatarsals have hypertrophied shafts, with deposition of bone on the subperiosteal surface, and gross porosity of the cortical bone, producing a 'lacy' appearance. Nutrient foramina in the shafts of the fourth and fifth metatarsals and in some hand phalanges are enlarged (Figs. 55–56A). The shafts of the clavicles and the spines of the scapulae also have porous bone deposition. This child has an active lesion on the nasal bones, with enlargement of the vascular foramina, porosity and irregular formation of bone on the subperiosteal surface (Fig. 56B). Conditions that need to be considered in differential diagnosis include osteomyelitis, treponematosi, leprosy, tuberculosis and chronic, severe anaemia. Osteomyelitis is unlikely to be responsible, as the gross cortical porosity of the hand and foot bones in the absence of sequestra and cloacae is not a characteristic of this condition (Silverman 1985). Early infections of the treponemal infection, yaws, can produce dactylitis (Hackett 1957). The pattern may be not unlike that seen in B21. Periostitis of the nasal bones and the nasal process of the maxilla can also occur in treponemal infection. Leprosy affecting the nasal area results in bone resorption rather than deposition, and the anterior nasal spine and hard palate are involved rather than the nasal bones themselves. Tuberculosis can affect the nasal area secondarily to long-standing infection of the soft tissues of the face, but as with leprosy, the disease results in bone resorption, not the porosity and periosteal reaction seen in this individual (Ortner and Putschar 1981). The extreme porosity of the cortex of some of the small bones of the hands and feet

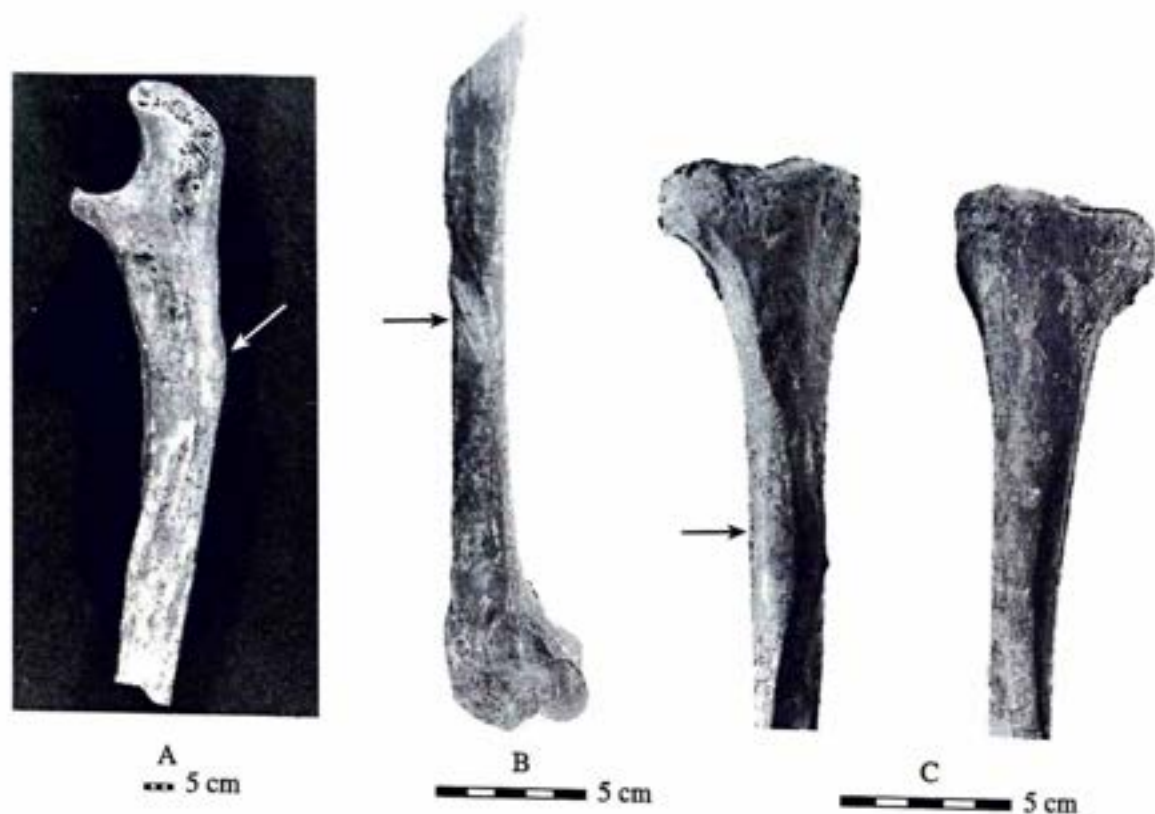


FIG. 52. Long bones with subperiosteal lesions. A: left ulna, burial 38. This has a localized hypertrophy of the cortex c. 25 mm in length, on the subcutaneous dorsal border distal to the trochlear notch of the left ulna (arrow). B: right humerus, burial 67. There is a cortical lesion on the lateral shaft (arrow). There is no evidence of active bone remodelling at the site. C: tibiae, burial 129. The right tibia has an area of localized hypertrophy on the anterior border (arrow). There is no evidence of active bone remodelling at the site

and the enlarged nutrient foramina are consistent with aspects of the bony responses described in classical clinical reports of children with chronic or severe anaemia (Caffey 1951, 1957) although the medullary cavity is not greatly enlarged.

On B88, a child of about one year, the cortices of the long bones are very porous, and there is extensive proliferation of subperiosteal reactive bone on the diaphyses of the limb bones and the clavicles. The proliferation extends in a radial arrangement, and is clearly distinct from the cortical bone. The metaphyses are not involved (Figs. 57-60). This pathology is non-specific and may have several alternative causes. These include infantile cortical hyperostosis, hypervitaminosis A, yaws, osteomyelitis and genetic anaemia. Infantile cortical hyperostosis, a poorly understood condition of 'obscure pathogenesis' (Silverman 1985:841), produces subperiosteal new bone forma-

TABLE 68: *The prevalence of fractures*

Burial	Sex	Age	Observation
61	Female	48	The left clavicle is at least 15 mm shorter than the right, (the right clavicle is slightly damaged, so accurate measurement is not possible) and the shaft has been remodelled with thickening and an increase in angularity (Fig 53A). A radiograph shows the trabeculae in the midshaft are of variable orientation, although the cortex is of uniform thickness along the shaft.
91	Male	45	The shaft of the left fifth metacarpal is bowed, with the concavity towards the palmar surface and a protuberance on the dorsal surface. The radiograph shows a line of remodelled bone extending diagonally across the medullary cavity. The position on the hand is again consistent with trauma. The involvement of the medullary cavity suggests that the bone had fractured but healed well.
110	Female	36	The left clavicle is shorter than the right, the midshaft is thickened and the angles of the lateral and medial curves of the shaft are increased. There is an exostosis on the inferior surface of the bone at the attachment of the <i>subclavius</i> muscle, and the margin on the medial surface between the attachments for the <i>pectoralis major</i> and <i>sternocleidomastoid</i> muscles is accentuated (Fig 53B). There is a small perforation on the posterior midshaft that may represent a remodelled cloaca. A radiograph shows sclerosis at the midshaft in the region of the cloaca, and marked porosity of the cortex at the remodelled muscle attachments.
142	Female	31	The shaft of the right fourth metacarpal has a thickening on the dorsal surface. The surface of the bone is smooth, with no periosteal reactive bone visible. The radiograph shows remodelling of both the dorsal and palmar cortices, including areas of thickening extending into the medullary cavity.

tion on the diaphyses of long bones (especially the tibia), the mandible, clavicle and occasionally the scapula and ribs. The distribution of the subperiosteal new bone can be variable (Jaffe 1972, Silverman 1985). The cortical new bone can become profuse, increasing the cortices to more than twice the original thickness. The metaphyses or epiphyses are not affected. The disease develops early in infancy, rarely later than five months of age and usually resolves within some months of diagnosis. It has not been recognised as a direct cause of fatality. A familial form of the condition

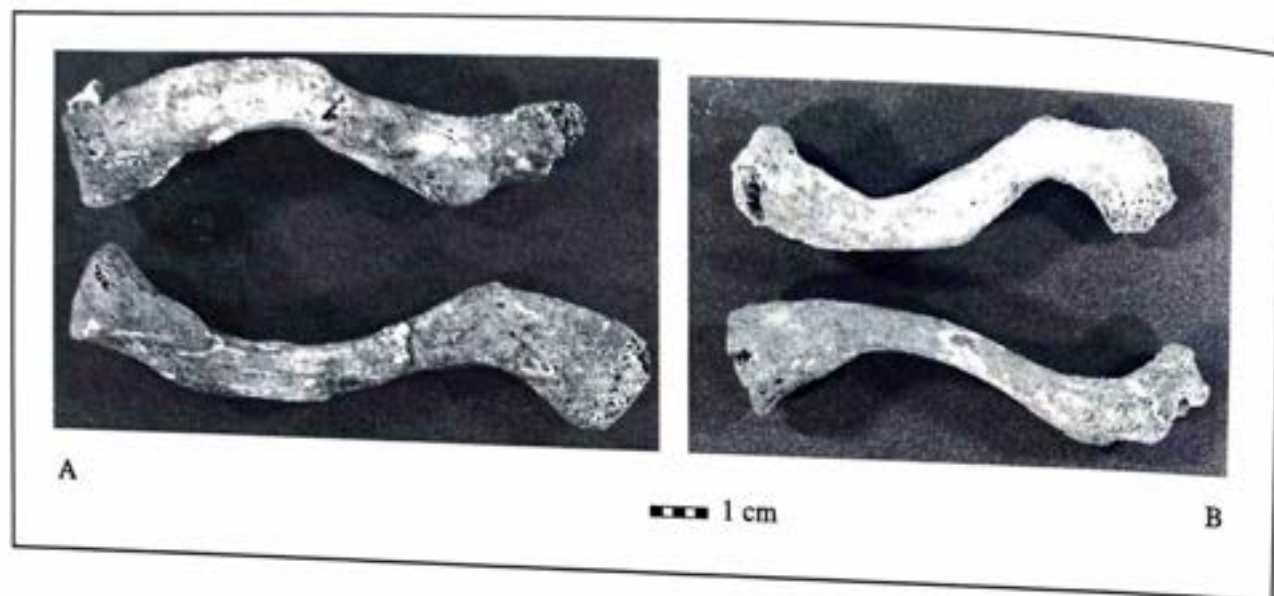


FIG. 53. A: clavicles, burial 61. The left clavicle (upper) is shorter than the right and the shaft has been remodelled with thickening and an increase in angularity. B: clavicles, burial 110. The left clavicle (upper) is shorter than the right, the midshaft has hypertrophied and the angles of the lateral and medial curves of the shaft have increased. The cloaca is not visible

has been recognised (MacLachlan *et al.* 1984, McKusick 1992). There is 'probably' also a sporadic form of unknown pathogenesis (MacLachlan *et al.* 1984). The condition was first recognised and recorded in the 1930s and 40s. Since then the number of cases recognised as inherited has increased and recently no 'sporadic' cases have been recorded (MacLachlan *et al.* 1984). The presence of the cortical hyperplasia in only one infant at Khok Phanom Di suggests that the familial form of the disease can be discounted. There is very little written about the sporadic form and despite an extensive search of the literature no estimate of its incidence or details of environmental factors triggering the condition have been found, other than a single case of prenatal iatrogenic origin (Pazzaglia *et al.* 1985).

Hypervitaminosis A can also induce the development of subperiosteal new bone, with the ulnae and metatarsals typically involved. The clavicles, tibiae and fibulae are also commonly affected, the femora, humeri, metacarpals and ribs less commonly and the mandible rarely. The metaphyses and epiphyses are not usually involved (Resnick and Niwayama 1981). Reported clinical cases involve overdoses fed to children by misguided parents. The liver of certain species of fish is high in vitamin A and occasionally hypervitaminosis is developed by eating fish liver (Higashi 1961). It has also been recorded in infants fed chicken livers (Silverman 1985). An unusual diet is clearly required to produce this condition. Yaws can also produce bony changes in its early stages which include diaphyseal periostitis, particularly in young children (Hackett 1976). Osteomyelitis can also produce periostitis (Jaffe 1972, Ortner and Putschar 1981, Silverman 1985). However, gross porosity is not a characteristic of this infection and there is no evidence on the skeleton of B88 of the cloa-

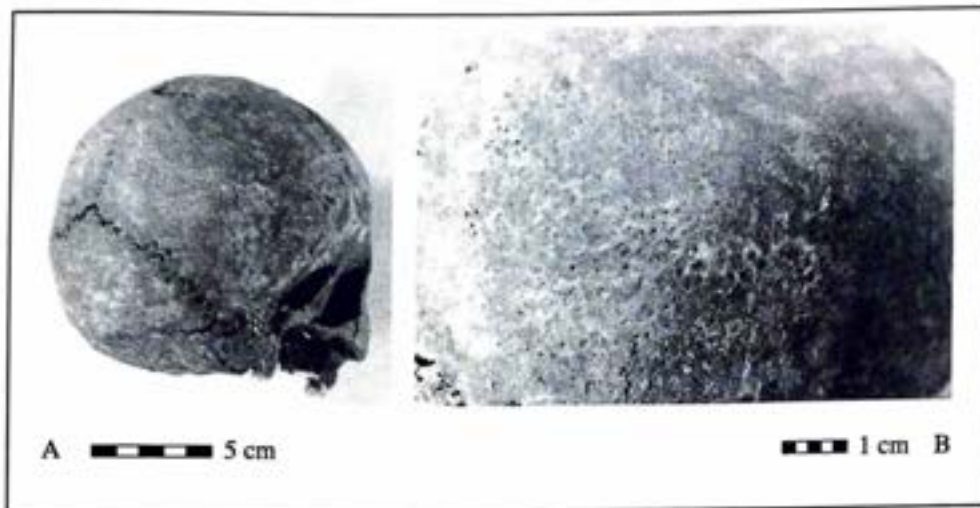


FIG. 54. The posterior parietal of burial 44, male, 18 years. A: showing an area of porosity. B: close up of the same parietal

cae, sequestra or involucra which are usual bony responses. The widespread distribution of lesions on the child is hardly compatible with survival in osteomyelitis. Subperiosteal new bone formation on the shafts of long bones, similar in appearance to the 'hair-on-end' arrangement of cranial subperiosteal bone in some cases of thalassaemia, has been described in Thai children with hereditary anaemia (Singcharoen 1989).

A young adult male (B24), has the right humerus 17 mm shorter than the left, and the muscle attachments, particularly on the deltoid crest, are strongly developed (Fig. 61A). The head and proximal end of the bone above the deltoid crest are angled medio-inferiorly. On an antero-posterior radiograph, the epiphyseal line is evident and also angled medio-inferiorly. There is no reactive bone visible on the periosteal surface, and no other pathology is evident on the radiograph (Fig. 61B). Differential diagnoses for this morphology include trauma, infection and anaemia. Fracture of the epiphyseal cartilage plate of the proximal humerus can occur during the growth period (Dameron and Reibel 1969). The long term skeletal response in untreated cases is not known but with non-surgical treatment involving immobilization the damage can result in shortening of the bone. The age at which the injury is sustained influences the results, but older children can suffer permanent angular deformity (Dameron and Reibel 1969). Infection can also result in permanent deformity, as osteomyelitis during infancy can spread to the cartilaginous growth plate with subsequent disturbances of growth (Resnick and Niwayama 1981). Both fracture and infection result in reactive bone growth. There is no evidence on B24 of reactive bone on the proximal humerus, although this does not exclude the possibility that trauma or infection occurred during early childhood, with subsequent complete remodelling. Premature fusion of the proximal epiphysis of the bone is relatively common in individuals suffering from thalassaemia (Currarino and Erlandson 1964; Exarchou *et*

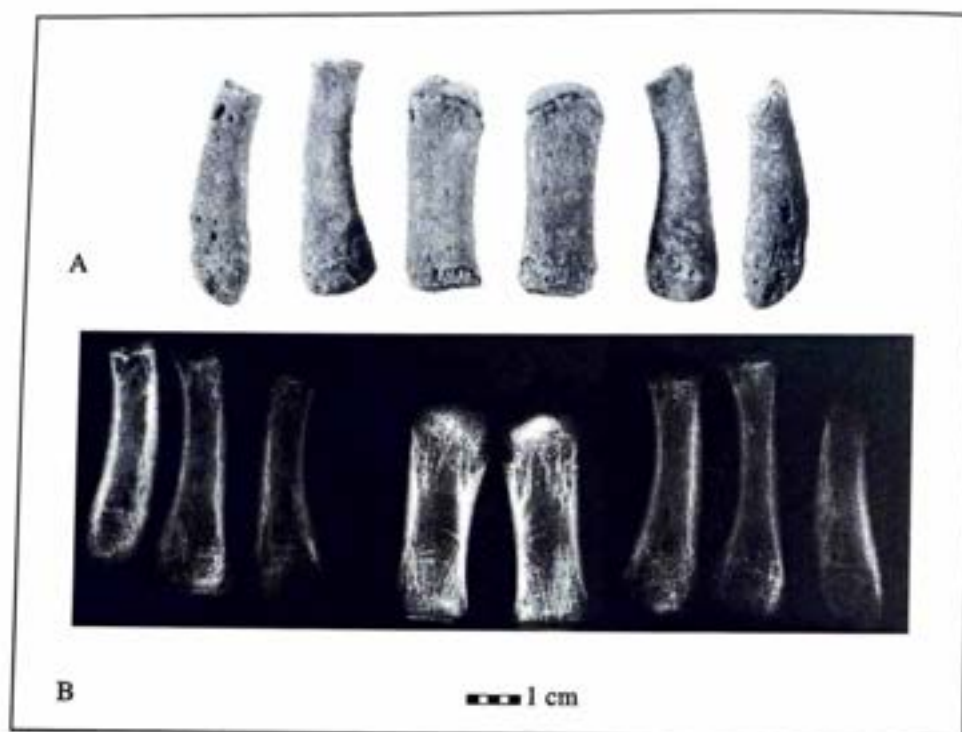


FIG. 55. A: dorsal views of the first, fourth and fifth metatarsals of burial 21 (8 years) The fifth metatarsals have hypertrophied shafts, with deposition of bone on the periosteum, and gross porosity of the cortical bone, producing a 'lacy' appearance. Nutrient foramina in these bones and the fourth metatarsals are enlarged. B: radiographs of the same metatarsals

al. 1984). Clinicians have reported up to 26 per cent of patients with homozygous ($n = 16/62$) (Exarchou *et al.* 1984) having deformities of the humerus resulting from premature fusion of the epiphysis. The severity of the anaemia is apparently not a factor in the development of this condition (Exarchou *et al.* 1984). A case from the prehistoric Middle East similar to B24 has been described by HersHKovitz *et al.* (1991).

Burial 94, a 31 year-old female, has reactive bone superficial to the cortex on the distal subperiosteal surfaces of the tibiae and fibulae. The bones are damaged, with the ends missing, and the exposed cortices are very thin, with marked porosity of the areas underlying the reactive bone. The periosteal reaction is apparent on the entire circumference of the tibiae, covering up to 3 cm length of the distal shafts (Fig. 62). There was no pathology elsewhere on this skeleton. Given this lack of evidence of systemic disease, the most probable diagnosis is tropical ulcer, which affects the distal leg, producing periostitis (Ennis *et al.* 1972). These are frequently of unknown aetiology and are described as 'non-specific' ulcers, although they can result from varicose veins or secondarily to trauma or sickle-cell anaemia. The bilateral lesions argue against trauma as an aetiology in this case

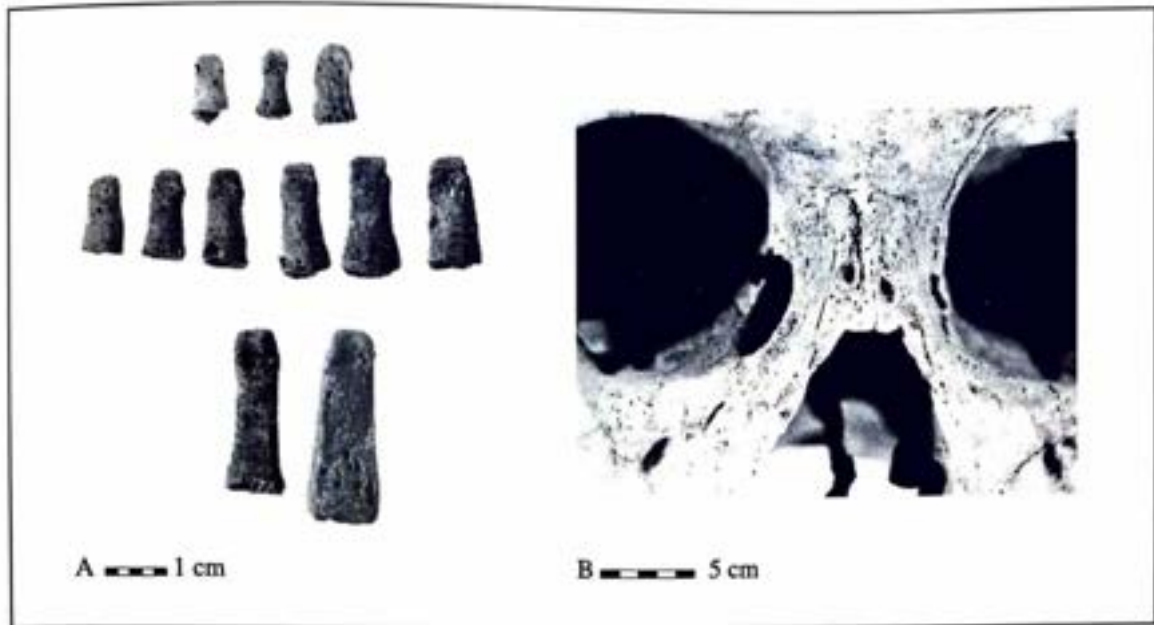


FIG. 56. A: palmar view of hand phalanges from burial 21. Several phalanges have very porous cortical bone. B: nasal bones from burial 21, with enlarged foramina and porous, reactive bone

and sickle-cell anaemia can be excluded, for reasons discussed further in the section on anaemia. Beyond these exclusions, the aetiology is unclear. There is no reason to believe that the infection was virulent as there is no evidence for osteomyelitis in the form of cloacae or sequestrae on the leg or foot bones.

Burial 45, a female aged 45 years, has bone elevations on the medial distal subperiosteal surfaces of both first metatarsals, near the articular surfaces. The joint surfaces appear to be unaffected. The area of bone is raised, with a roughened, undulating surface. On both metatarsals there are erosions, with those on the left foot larger than those on the right (Fig. 63). Possible explanations include the early stages of the development of gout, which can result in bone proliferation but more often erosions produced by tophi (deposits of urate crystals). These may occur at a distance from the joint and have a 'punched out' appearance (Resnick and Niwayama 1981). Gout commonly occurs at the first metatarso-phalangeal joint, but is not usually symmetrical and is more common in males than females. An alternative explanation is the development of infected lesions through repeated trauma to the overlying soft tissues, with the erosions representing cloacae draining the infection. It is difficult to imagine an activity that would produce symmetric lesions of this manner. There is no obvious explanation for the lesions.

In one of the earliest burials, B152 (male, aged 37 years) the diaphyseal cortices of the major limb bones are very thick, with almost no medullary cavity in some areas (Fig. 64A). The external appearance of all bones is normal and the metaphyses, epiphyses and cancellous bone are not af-

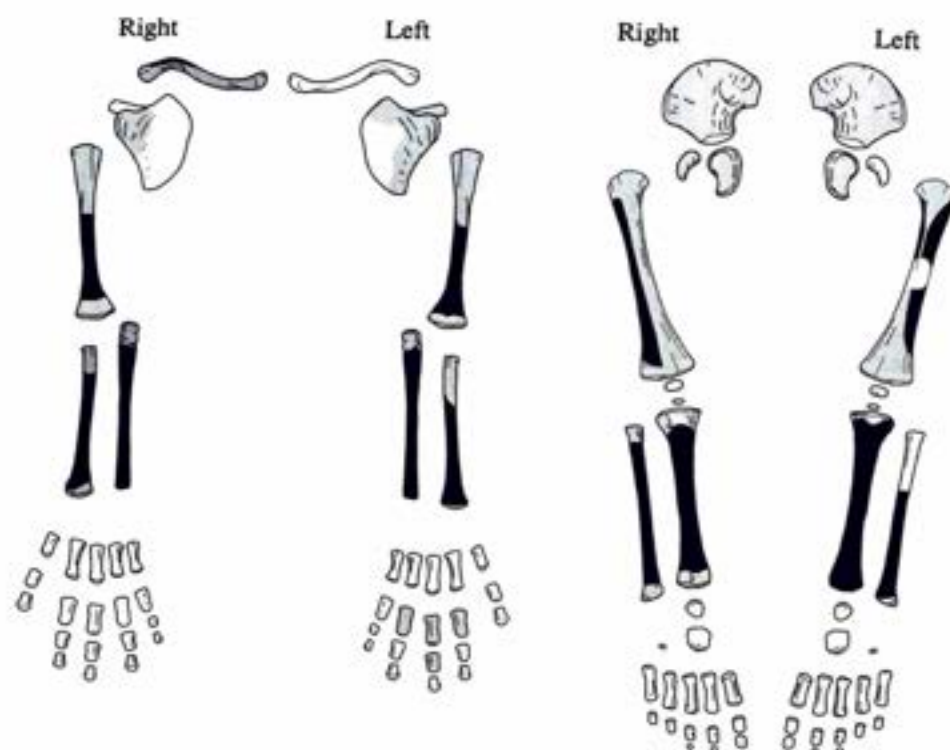


FIG. 57. Upper and lower limbs of burial 88, showing the skeletal elements present (light shading) and distribution of reactive periosteal bone (dark shading)

ected. In a broken section of the distal fibula (Fig. 64B) there is evidence of osteoblastic activity in the form of a build-up of bone of about 0.5 mm thickness with the appearance of circumferential lamellar bone within the medullary cavity. The bone has a dense, sclerotic appearance and is very heavy, although most of the skeletons at this depth in the site are very heavy because of remineralization. Mineral deposits have coated the bone surfaces, including the endosteal surface, and infiltrated the Haversian system. Differential diagnoses include a variety of dysplasias and cranio-tubular hyperostoses including Van Buchem disease, sclerosteosis, craniodiaphyseal dysplasia, and autosomal dominant osteosclerosis (Eastman and Bixler 1977, Gelman 1977). These congenital conditions are very rare and there seems to be considerable confusion in the clinical literature as to their precise characteristics. The endosteal hypertrophy of the long bones of B152 is very similar to the illustrations of a case of Van Buchem disease (Eastman and Bixler 1977), with the exception of the involvement of the cranial base and mandible in the clinical case. All these dysplasias appear to affect the cranium or other bones in addition to diaphyses, or to affect the external dimensions of the bones. In the absence of any cranial defects and given the normal external appearance of the bones, none of them appear to be the cause of the condition in B152. Paget's disease also results

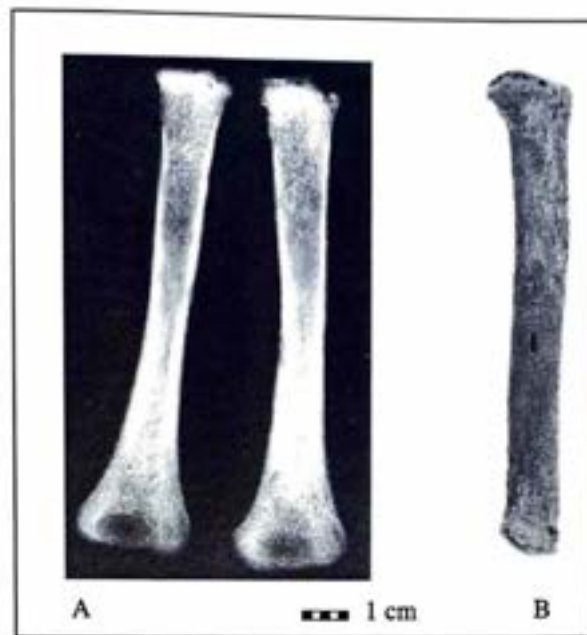


FIG. 58. A: antero-posterior radiograph of burial 88 humeri, showing the deposition of reactive subperiosteal bone. B: medial view of the left humerus of burial 88. The distal half of the diaphysis is hypertrophied with deposits of porous bone on the periosteal surface. The nutrient foramen is enlarged

in bony overgrowth, but again affects the external dimensions of bones. Endosteal hypertrophy of the femoral shafts and other long bones, with sclerosis and thickening of the cortex and obliteration of the medullary cavity has been described in cases of sickle-cell thalassaemia (Reynolds *et al.* 1973). This is a result of ischaemia following infarction, and is therefore characteristic of sickle-cell anaemia and not of anaemia caused by other haemoglobinopathies.

General pathology - discussion

This section considers the skeletal pathology diagnosis in the Khok Phanom Di population and discusses the diseases which may be represented, both in individuals and the population. In addition to considering differential diagnoses based on the evidence of skeletal pathology, prevalence rates are taken into account. These are considered in relation to reported rates in other skeletal populations and in the clinical literature. Unfortunately, the comparison of the range of diseases present and their prevalence among skeletal populations is difficult. Not only is there the potential for inter-observer variation in the subjective assessment of skeletal pathology, but there is no standardization in the methods of summarising and reporting data. Where the prevalence of a skeletal pathology is reported simply as an overall figure without consideration of the age or sex of the individuals, or of the pattern within each individual, the data cannot be directly compared except in very broad terms. Both clinical and anthropological publications, until recently, have concentrated on extreme cases

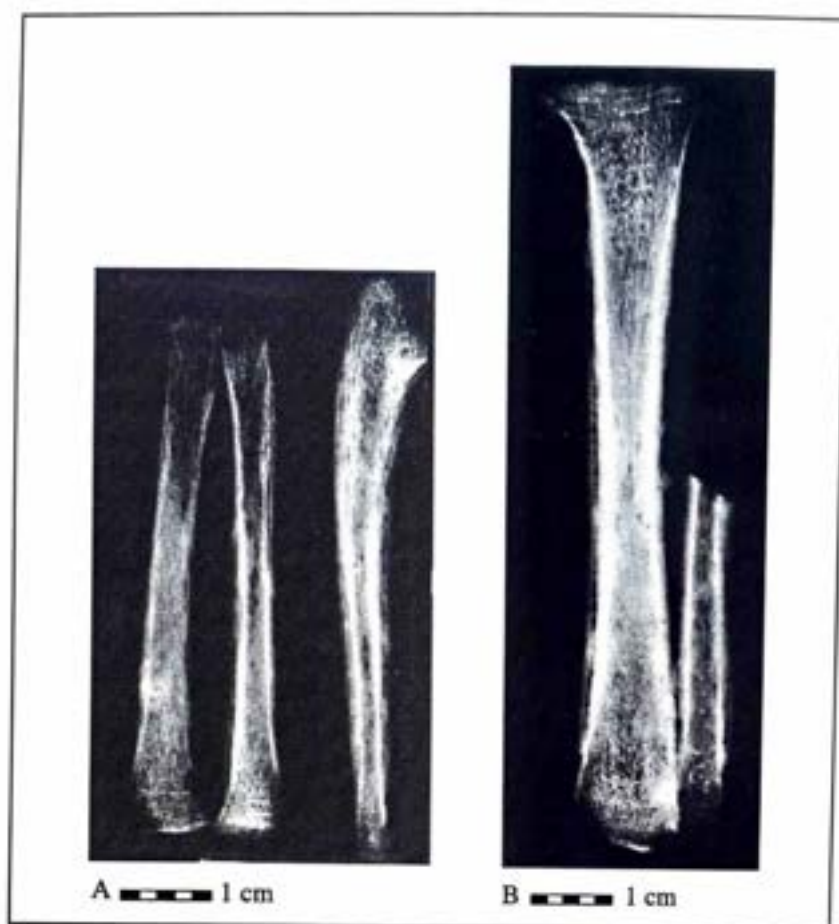


FIG. 59. Radiographs of the radii, right ulna, tibiae and part of the fibula of burial 88, showing the periosteal deposits and porotic cortex

of skeletal pathology, making it even more difficult to compare the prevalence of the less spectacular remodelling. There are very few publications from South East Asian populations. Accordingly, the assessment of pathology at Khok Phanom Di relies primarily on intrasite comparison, with consideration of data from other populations only where the comparability is unequivocal, and necessary to establish the 'normal' range of any skeletal characteristic.

The lesions which occur in more than one individual include cribra orbitalia. As has already been noted, this is a response to anaemia. In 1969, Sangvichien *et al.* described thick cranial vaults in the people at the prehistoric site of Ban Kao in Central Thailand. They identified these as evidence of anaemia caused by β -thalassaemia/HbE disease. This is a combination of two of the genetic haemoglobin polymorphisms, of which the sickle-cell gene is best known. These balanced polymorphisms are believed to persist through a survival advantage conferred on carriers in malarial

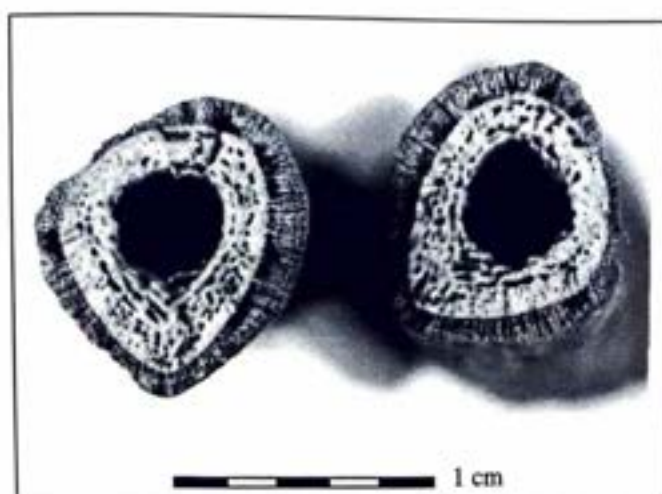


FIG. 60. Cross-sections of the mid-shaft of the left tibia of burial 88. The cortex of the bone is very porous and there is proliferation of radial bone on the periosteal surface. The colour of the additional bone is the result of differential absorption of ochre colouring from the burial matrix

areas. In subsequent skeletal reports for sites in North East Thailand, Pietrusewsky (1974a, 1988b) suggested that a large proportion of all adults had pathologically thickened crania. He attributed this to anaemia, probably again reflecting thalassaemia. The presence of the genes for thalassaemia or one of the abnormal haemoglobins in these prehistoric populations has implications for the explanation of their adaptation to the environment. Firstly, it is an indicator that the population was exposed to the malarial parasite, which has a major impact on the health of a population. Secondly, it is an example of genetic adaptation to environment and as such is an aspect of human biology which is significant enough to contribute to the explanation, beyond cultural and geographic factors and resource distribution, for the demography of a population. The presence of anaemia in the Khok Phanom Di population and its possible indication that the people carried one or more of these genetic diseases has been given special attention in the investigation of skeletal pathology. The evidence for anaemia in the population, the theory of the skeletal response to the condition and the wider implications of the diseases are discussed in the following section in some detail. Other pathology affecting a number of individuals reflects infection of the periodontal tissues. Dental pathology is discussed in detail in chapter VI.

Evidence of healed injuries in the form of fractures and localized subperiosteal lesions on subcutaneous surfaces of bones also affect a number of individuals. Their presence in the population indicates that those who suffered the trauma were sufficiently healthy to recover from the tissue damage and any ensuing infection. Eight adults were affected, four with fractures and four with lesions reflecting soft tissue injury, comprising 11 per cent of the adults. This does not necessarily include all cases of skeletal trauma suffered by the adults, as the small bones of the hands and feet were not all present in most skeletons. Also excluded would be any fractures suffered at the time of



FIG. 61. A: posterior view of humeri of burial 24 (male, 24 years). The right humerus is 17 mm shorter than the left (left 290 mm, right 273 mm), and the muscle attachments, particularly on the deltoid crest, are strongly developed. The head and proximal end of the bone above the deltoid crest are angled medio-inferiorly. B: antero-posterior radiograph of the humeri of burial 24. The epiphyseal line is visible and is angled medio-inferiorly

death, as it is not possible to distinguish unremodelled fractures from post-mortem breakage, unless the latter is very recent. Pathology other than joint degeneration on the appendicular skeletons reported from other prehistoric Thai populations is minimal. At Ban Na Di, there was one fractured metacarpal, and one individual with a lesion on the anterior tibia suggestive of soft tissue infection (Houghton and Wiriayomp 1984). The bones involved and the frequency of lesions is similar to that at Khok Phanom Di, and probably indicative of the type and frequency of accidental injuries suffered by physically active populations.

The remaining cases are of individuals with diverse single or multiple lesions. Burial 44 has periostitis on the skull, B94 on the tibia and fibula. Burial 21 has lesions on the hands and nose,

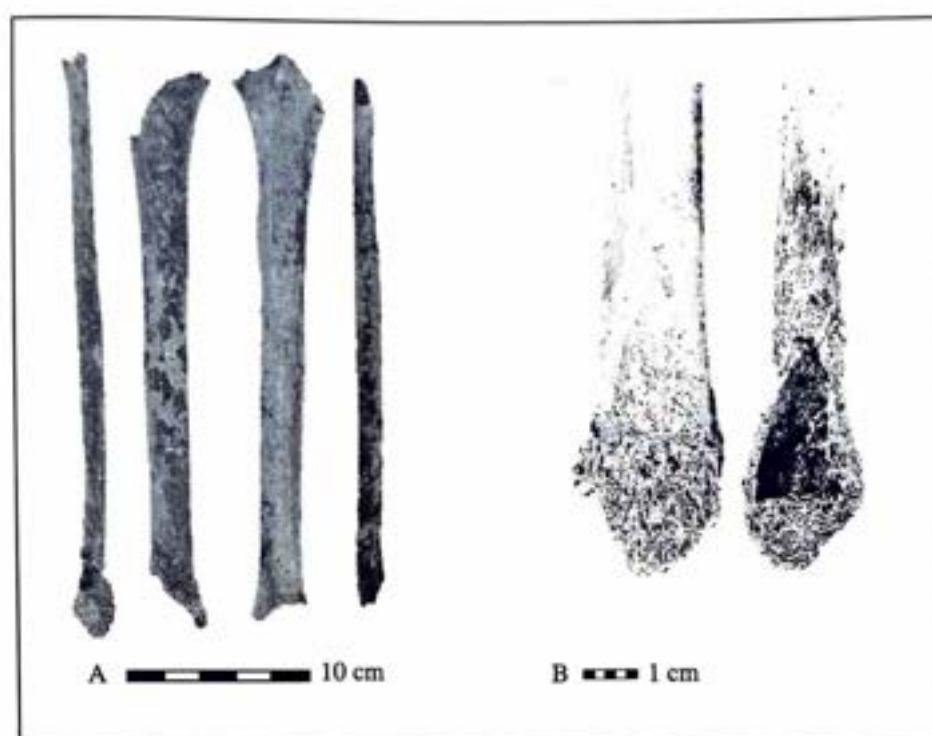


FIG. 62. A: the tibiae and fibulae of burial 94, illustrating the reactive bone on the periosteal surface of the left tibia and fibula. B: close-up of the distal left tibia and fibula of burial 94, showing the reactive bone on the periosteal surface

B88 on multiple bones. Burial 24 has a mis-shapen humerus and B152 thickening and sclerosis of the long bone shafts. When making a diagnosis from lesions which are not pathognomonic, as most of these are, other factors such as frequency and distribution of the disease in the population need to be taken into consideration.

In the diagnosis of the subperiosteal hyperostosis of B88, the absence of other examples in the population is relevant. It is unlikely that a single case of either the familial form of infantile cortical hyperostosis or hypervitaminosis A would exist in a population this size. The enigmatic sporadic form of infantile cortical hyperostosis cannot be entirely discounted. Yaws is a possible explanation for the lesions in both B21 and B88. It is a disease easily transmitted within the home and its surroundings and is common among those engaged in agricultural work, such as rice-growing (Bruce-Chwatt 1978). In living populations, before the advent of antibiotics, seropositivity in adults in endemic areas was as high as 80 per cent (Hackett 1947). Clinical estimates of the prevalence of bony changes in individuals with yaws suggest about 1–5 per cent will be affected (Ortner and Putschar 1981, Rothschild and Heathcote 1993). The rate can be expected to be higher in skeletal populations, where lesions are identifiable at an earlier stage than from radiographs of living indi-

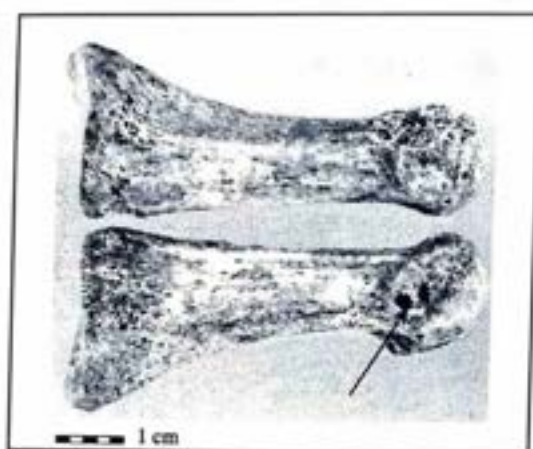


FIG. 63. Medial view of first metatarsals of burial 45. Both bones have elevations on the medial distal subperiosteal surfaces, near the articular surfaces. The joint surfaces appear to be unaffected. The elevated area of bone has a roughened, undulating surface, with erosions. These are larger on the left foot than on the right (arrow)

viduals. Reports indicate that the rate in skeletal populations ranges from 20 per cent in individuals over the age of 10 years (Rothschild and Heathcote 1993) to 80 per cent (Houghton 1996). The complete absence of any relevant bone changes, either active or inactive, in the population from Khok Phanom Di aged 10 years or more ($n=74$) argues against treponemal infection as an explanation for the lesions on burials 21 and 88.

As has already been noted, the prevalence of cribra orbitalia suggests that anaemia was suffered by a proportion of the subadult population. The discussion of the other skeletal pathologies of burials 21, 24 and 88 offers several alternative diagnoses in each case but a consistent factor is that anaemia is one of the alternatives. Accounting for all the lesions without considering anaemia would require the suggestion that a variety of infections were present in the population but that each affected an uncharacteristically small proportion of the people.

ANAEMIA

Anaemia is a condition which is very common in tropical populations (Manson-Bahr and Apted 1982). It is a disorder of the blood, characterised by a deficiency in the quality or quantity of red blood cells (erythrocytes) resulting from excessive blood loss, ineffective erythropoiesis (production of the cells) or haemolysis (premature destruction of erythrocytes) (Dacie 1985). Red cell producing (haemopoietic) bone marrow activity increases to compensate for the deficiency of erythrocytes, hypertrophying as it does so. The skeletal response is secondary to this marrow hypertrophy (Moseley 1974, Weatherall and Clegg 1981, Dacie 1985). There are numerous aetiologies of anaemia, including dietary deficiencies and parasitic infections (Manson-Bahr and Apted 1982, Da-

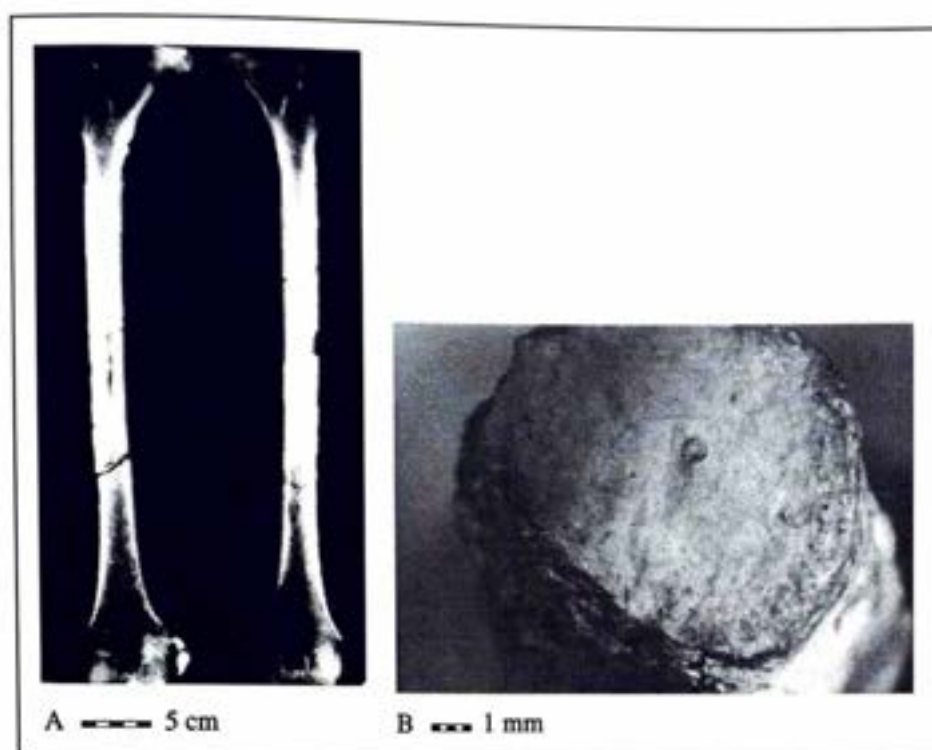


FIG. 64. A: radiographs of femora of burial 152, showing extreme thickness of the cortex of the shaft of unknown aetiology. B: section of the distal fibula of burial 152, showing the extreme thickness of the cortex with narrowed medullary cavity. There is a build-up of circumferential lamellar bone of about 0.5 mm thickness on the endosteal surface

cie 1985) and the genetic anaemias such as sickle-cell anaemia and thalassaemia. The presence of some of these have significant connotations for the explanation of the general health of a community.

In infants, haemopoietic marrow is present in the long bones and the ribs, skull, sternum and vertebrae. Any part of the skeleton, and the limbs in particular, may be affected by the hypertrophy of the marrow. Beginning at about the age of four years, haemopoietic marrow is progressively replaced by fatty marrow in the appendicular skeleton. By adulthood, haemopoietic marrow is normally present only in the axial skeleton (Williams *et al.* 1980). The individual skeletal response can therefore change over time as the marrow regresses from the extremities towards the axial skeleton (Caffey 1951, 1957, Aksoy *et al.* 1966, Weatherall and Clegg 1981, Dacie 1985). In adults, active skeletal response would appear on the cranium, although in some of the thalassaemia syndromes active erythropoiesis may continue to be stimulated in other bones (Weatherall and Clegg 1981). The age of the individual has further significance in identifying anaemia in that not only is the site of skeletal response likely to change over time, but the degree of response will reduce as the plasticity of the bone decreases with age (Lanzkowsky 1968). Skeletal evidence in an adult is an indication

that the individual had suffered in childhood, rather than that they were necessarily anaemic at the time of death.

Modern clinical diagnosis of the presence and aetiology of anaemia concentrates on physiological symptoms and blood biochemistry, as these are not only diagnostic but also of more immediate concern in managing the disorder. Although there are multiple causes of the condition, skeletal changes have been clinically reported only in individuals with inherited disorders of haemoglobin and acquired chronic iron deficiency anaemia (Stuart-Macadam 1992). Most cases of anaemia of these aetiologies are mild and would not produce any skeletal response, and even in more severe cases bony changes are not necessarily of clinical concern compared with other symptoms. Clinical observations of those cases with skeletal evidence of anaemia show that the basic response is similar regardless of the initiating disease, although there are some aspects of the skeletal reaction which are specific. The degree of skeletal response does not necessarily correlate with the degree of anaemia, with significant variation in the site and extent of remodelling even among individuals with the same type or degree of anaemia (Caffey 1951, 1957, Moseley 1971). There are some secondary skeletal reactions such as bone infarctions which are specific to particular genetic anaemias (Moseley 1974). Except where these secondary reactions have occurred, identification of the aetiology of the anaemia is difficult on the basis of skeletal pathology alone.

Many of the reports of the clinical findings of skeletal evidence of anaemia concern cases of homozygous β -thalassaemia, as this produces some of the most distinctive skeletal changes of any of the anaemias, with features evident both radiographically and on gross observation (e.g. Caffey 1951, 1957). There are also case descriptions of skeletal changes in β -thalassaemia trait carriers (Sfikakis and Stamatoyannopoulos 1963), thalassaemia-Haemoglobin E disease (Chernoff *et al.* 1956), α -thalassaemia (Na-Nakorn *et al.* 1965, Wasi *et al.* 1969) and iron deficiency anaemia (Lie-Injo 1958, Aksoy *et al.* 1966, Lanzkowsky 1968). Reports of radiological evidence of skeletal changes in response to malaria were made in Italy in the late 1930s (Chini *et al.* 1938, Chini 1939), but more recent unequivocal clinical reports have not been found. This suggests that anaemia associated with malarial infection infrequently affects the skeleton.

Bony evidence of anaemia in the cranium is most common in the anterior cranial fossa, where the orbital plate of the frontal bone forms the orbital roof. The orbital plate may hypertrophy to accommodate the expansion of the marrow within its normally closely apposed tables. The effect on the structure is that the subperiosteal surface of the inferior table becomes firstly porous and secondly develops radiating trabeculated or cribrotic bone growth (Hengen 1971, Ortner and Putschar 1981, Stuart-Macadam 1989). The only clinical evidence is thickening of the roof of the orbit visible on radiographs (Caffey 1937, Stuart-Macadam 1987a).

A similar response is produced in the cranial vault in the form of grossly visible external porosity, thinning of the external table, expansion of the diploe and thickening of the trabeculae with reduction in the number and radial arrangement of those remaining (Ortner and Putschar 1981, Stuart-Macadam 1987a, 1992). In extreme cases the development of radial trabeculae with an orientation perpendicular to the cranial vault forms a radiographic appearance described as 'hair-on-end' (Baty *et al.* 1932, Caffey 1937, Moseley 1971, Stuart-Macadam 1987a). In these cases the outer table is virtually destroyed, and in skeletal material, the appearance of the outer vault is one of extreme porosity with deposition of periosteal bone, in patterns described as 'labyrinth-like' (Ortner and

Putschar 1981:262) or 'like coral' (Angel 1967:379). The frontal, parietal and occipital bone above the superior nuchal line can all be involved (Baker 1964).

The vault changes are commonly described both in clinical cases of genetic and iron-deficiency anaemia (Lie-Injo 1958, Lanzkowsky 1968, Moseley 1971, Weatherall and Clegg 1981) and in skeletal studies (Angel 1967, Ortner and Putschar 1981). The early stages of these vault changes are given less emphasis in clinical reports than in skeletal reports. The extreme condition, with absence of the outer table, is not typical as it is present in only 5-20 per cent of clinical cases (Fernbach 1984, Silverman 1985, Scutellari *et al.* 1989). Generalised osteoporosis is much more common, although definitely less spectacular and much more difficult to differentiate from the normal appearance of the cranial vault on radiographs.

From as early as the nineteenth century, reports of skeletal samples have included observations of porosity, cortical thinning and diploic expansion of the cranial vault or the orbital plates (Angel 1967, Stuart-Macadam 1985). The aetiology and relationship of these conditions has prompted considerable research and debate in the anthropological literature over the last hundred years, and they have been reported from skeletal collections widespread both geographically and chronologically (e.g. Zaino 1964, Angel 1966, 1967, 1971, Hengen 1971, El-Najjar *et al.* 1975, Lallo *et al.* 1977). The hypothesis that anaemia, either genetic or acquired, is responsible is now widely accepted (Stuart-Macadam 1989), although the underlying cause of the anaemia has not always been clear (Ortner and Putschar 1981). The relationship of the evidence from skeletal samples with radiographic evidence from clinical studies was not systematically studied until the 1980s (Stuart-Macadam 1987a, 1987b, 1989).

Various terms have been used to identify the lesions, with 'porotic hyperostosis' (Angel 1966) for the cranial vault and 'cribra orbitalia' (Welcker 1888: cited in Angel 1967) for the orbital plates being now most commonly used. These terms are not generally used in clinical descriptions of the cranial response to anaemia, although they are now recognised by clinicians as describing the gross cranial evidence of conditions identified in radiographs of living individuals (Weatherall and Clegg 1981). The concentration on these cranial lesions in early anthropological skeletal reports is probably a reflection of both the preferential collection of crania and the concentration of reactive bone in the cranium in adults. There are numerous descriptions of the skeletal changes of porotic hyperostosis and cribra orbitalia in skeletal studies (e.g. Angel 1967, El-Najjar *et al.* 1975, Stuart-Macadam 1985).

In a study on sickle-cell anaemia patients, Reynolds (1962) found that in patients with anaemia the ratio of diploic to compact bone in the cranial vault ranged from 1:2.3-1:7.3, with a mean of 1:4.4, whereas in none of a series of normal controls did the ratio of diploic to cortical bone in the cranial vault exceed 1:2.3. On the basis of this, Stuart-Macadam (1987a) measured the ratios on radiographs of the frontal bone in two skeletal groups, one Romano-British and one nineteenth century East Asian, and found significantly higher ratios in those individuals with other cranial evidence of porotic hyperostosis than in those without lesions.

Other cranial changes reported include enlarged vascular impressions on the inner table (Lawson *et al.* 1984) and 'onion-skin' layering of the vault apparent on radiographs (Orzincolo *et al.* 1989). Internally, development of the frontal sinuses and the mastoid air cells may also be retarded (Caffey 1951, Weatherall and Clegg 1981, Fernbach 1984, Stuart-Macadam 1987b).

A further major skeletal reaction in the skull described in clinical reports of thalassaemia are the facial changes resulting from hypertrophy of the marrow of the maxilla and zygomatic bones. The effects of this are prominence of the malar eminences, depression of the bridge of the nose, maxillary protrusion, widening of the interorbital bones (hypertelorism) and lack of formation of the maxillary sinuses (Baty *et al.* 1932, Caffey 1957, Baker 1964). Secondary effects involve the dentition with the anterior teeth widely spaced and protruding, with overbite and malocclusion (Caffey 1957, Kaplan *et al.* 1964). The appearance of these individuals is described as 'mongoloid-facies' in young children and 'rodent-facies' in older children (Caffey 1957). Because of the distinctive appearance of these individuals in Caucasian populations, this skeletal effect of thalassaemia is commonly described in clinical reports (Baty *et al.* 1932, Caffey 1951, Caffey 1957, Baker 1964, Moseley 1971) but no skeletal reports describing it have been found. As the description 'mongoloid facies' implies, the facial changes would not be expected to be so obvious in Oriental populations as in Caucasians, as they represent morphological features which are normal in the Far East. Clinical reports from Thai populations are equivocal on the distinctiveness of thalassaemic facies compared with the normal Thai physiognomy. Chernoff *et al.* (1956) found no obvious thalassaemic facies in 30 patients (children and adults) with severe thalassaemia-HbE disease, although they noted:

"...a number of individuals had very suggestive changes. Nevertheless, the facial appearance of our patients resembled in many ways the Mongoloid facies described in instances of thalassemia from the Mediterranean area, where oriental features do not normally complicate the picture." (Chernoff *et al.* 1956:466)

In a study of 14 Thai patients with thalassaemia-HbE disease or homozygous β -thalassaemia (13 children, one adult) Flatz *et al.* found:

"All patients had the typical appearance of patients with thalassaemia major: pallor, facial changes (hypertelorism and prominent zygomata) and protruding abdomen. There was no difficulty in recognizing the supposedly 'mongoloid' facial changes in the mongoloid Thai population." (Flatz *et al.* 1965:216)

It is not clear how distinctive the facial changes could be expected to be in the population from Khok Phanom Di.

The mandible is not involved in gross morphological changes, but there are changes in trabeculation and general osteoporosis (Baker 1964).

Identification of the aetiology of lesions seen on skeletal samples is improved greatly when a clinical approach is taken as far as is possible and all bones are included, not just the skull. Postcranial changes generally reflect the same response to marrow hyperplasia as in the skull, with expansion of the medullary cavity in long bones accompanied by reduction in distribution and coarsening of trabeculae, and extreme thinning and porosity of cortices (Caffey 1951, Baker 1964, Weatherall and Clegg 1981). In infants and young children, this is most commonly described in the metacarpals of the hands, although it has also been reported in the major limb bones (Baty *et al.* 1932, Caffey 1957, Baker 1964). The marrow hypertrophy is accompanied by increased vascularity, resulting in

the enlarged nutrient foramina in the phalanges (Middlemiss and Raper 1966, Fink *et al.* 1984, Lawson *et al.* 1984). Overall skeletal growth and maturation can also be retarded in severely anaemic individuals (Weatherall and Clegg 1981, Laor *et al.* 1982). The only description of long bone changes in skeletal material is that of Angel (1966, 1971), who describes long bones from infants from pre-historic sites in the Mediterranean with an unremodelled "... 'inner shell' of bone attached to the cortex by a few trabeculae." (Angel 1966:760).

Reaction in the bones of the axial skeleton is not restricted to children but can occur at all ages. Vertebrae and ribs can develop the same coarsening of the trabecular pattern evident elsewhere in the skeleton, with the trabeculae in the vertebrae aligning themselves vertically (Baker 1964). In adults, the vertebrae can collapse (Baker 1964), and Caffey (1951) describes multiple compressed vertebrae in a 15-year-old. Baker (1964) also describes 'swelling' of the ribs adjacent to the spine in older patients.

Anaemia - methods

As the site and severity of skeletal responses to anaemia varies with age, features other than the relevant pathology such as cribra orbitalia already described in children and adults, differ to some extent. In incomplete or damaged adult skulls where the internal structure of the bones of the vault was visible, the thickness of the bones was noted, and where possible, the ratio of diploic to cortical bone was measured. The diploe were checked for any unusual structure, and the outer table for porosity or deposition of subperiosteal bone. Complete adult crania were radiographed and the thickness of the vault, the internal structure of the tables, the formation of the frontal sinuses and the form of the impressions for meningeal vessels were noted. Vault thicknesses were recorded at bregma, the highest point of the cranium perpendicular to the Frankfort Horizontal Plane (vertex), and on the frontal bone, midway between nasion and bregma, outside the limit of the frontal sinus. Dial calipers graduated to 0.05 mm were used, and the measurements were adjusted for radiographic enlargement using a scale included on the images. There was some difficulty in selecting points for measurement, as there is no standard method of recording cranial thickness and therefore not all data from studies of cranial thickness can be used for comparison in establishing a normal range of thicknesses (Ishida and Dodo 1990). Leeuwenberg (1990) established that the most commonly used points in studies based on radiographs are bregma, lambda and vertex, and used these points in her initial study in order to have data which were comparable with the widest possible range of other populations. Lambda has been omitted from this study because of the variability of the degree of bone development in the occipital (Webb 1990), and has been replaced by the measurement on the frontal bone. The frontal bone has been included because it is often affected in cases of pathological thickening of the vault. The thickness of the orbital plates was measured on lateral cephalographs of adults and children, using the methods of Stuart-Macadam (1987a) and correcting for radiographic enlargement.

Maxillary protrusion was measured at the sagittal midline from the lateral radiographs. The measurement was based on the method used by Sorathesn (1988) as this study of the craniofacial norm in modern Thai provides the only data suitable for use as a standard. The measurement is used in establishing the degree of dentofacial deformities in candidates for surgery (Sorathesn 1988). The measurement (SNA) is of the angle from the midpoint of Sella (S) to Nasion (N) to Subspinale, the

most posterior point on the curve between the anterior nasal spine and the crest of the maxillary alveolar process (A) (Connor and Moshiri 1985:120).

The lateral development of the maxilla and malar bones was assessed by comparing the relationship of bi-jugal width (Measurement 43 (1), Martin and Saller 1957) with the width between the infraorbital foramina. The bi-jugal width was used as the measure of lateral size as it includes both the maxilla and malar, but the width on its own is not useful, as it will vary depending on cranial size. The central cranium on the sagittal plane is relatively stable as it is constrained by neural development (Houghton and Kean 1987). The width between the infraorbital foramina therefore provides a 'control' from which the relative transverse development of the midface can be measured. The degree of development of the maxillary sinuses was checked on anterior-posterior cephalographs. This was recorded subjectively, as there are no norms for the highly variable sinus size and shape. The only postcranial feature recorded in adults was the enlargement of nutrient foramina in metacarpals, metatarsals and phalanges.

The same observations were made on the crania of children as for adults, with consideration of the degree of development of the thickness and internal structure of the tables of the cranial vault and frontal sinuses as would be appropriate for the age of the child. The maxillae, zygomatic bones, and sphenoid were checked for any evidence of thickening or failure of pneumatization of the paranasal sinuses. The facial profile was observed for maxillary protrusion and the dentition checked for spacing of the maxillary teeth and dental occlusion. Complete crania were radiographed and the same features observed as for adults. The degree of maxillary protrusion as indicated by the angle SNA was measured where possible. Additional measurements of the facial profile were based on the method used by Yen (1973). This is a study of the facial profile of Chinese rather than Thai or other Southeast Asian boys, but is the closest data found which provides some idea of the norm at different ages during growth. The measurements used are illustrated in figure 65 (Yen 1973:302). The angles of the nasal bone, naso-maxillary, subnasal and alveolar were used. Transverse facial widths were measured as for adults.

Long bones were checked for evidence of cortical thinning and medullary expansion, either through gross observation or from radiographs. The size of nutrient foramina on metacarpals, metatarsals and phalanges were checked.

Anaemia - results

The surfaces of many of the cranial vaults were damaged postmortem or heavily encrusted with calcareous deposits. The proportion on which pathology affecting the external table could be confidently assessed was small and almost entirely from the later mortuary phases. As most of the children were buried in the later phases, the surfaces of their crania were relatively well preserved. The only individual of any age with changes on the external table was B44, already described.

Cephalographs were taken of 18 female skulls and 11 male skulls. In virtually all cases the density of the bone made it impossible to obtain images with the internal structure of the tables visible. For comparative purposes, the thicknesses of the vaults were recorded, as listed in table 87. Table 69 summarises the data.

The male skulls are significantly thicker than the female skulls at bregma and vertex but not at the frontal. The male skulls increase in thickness from front to back, whereas the female skulls are

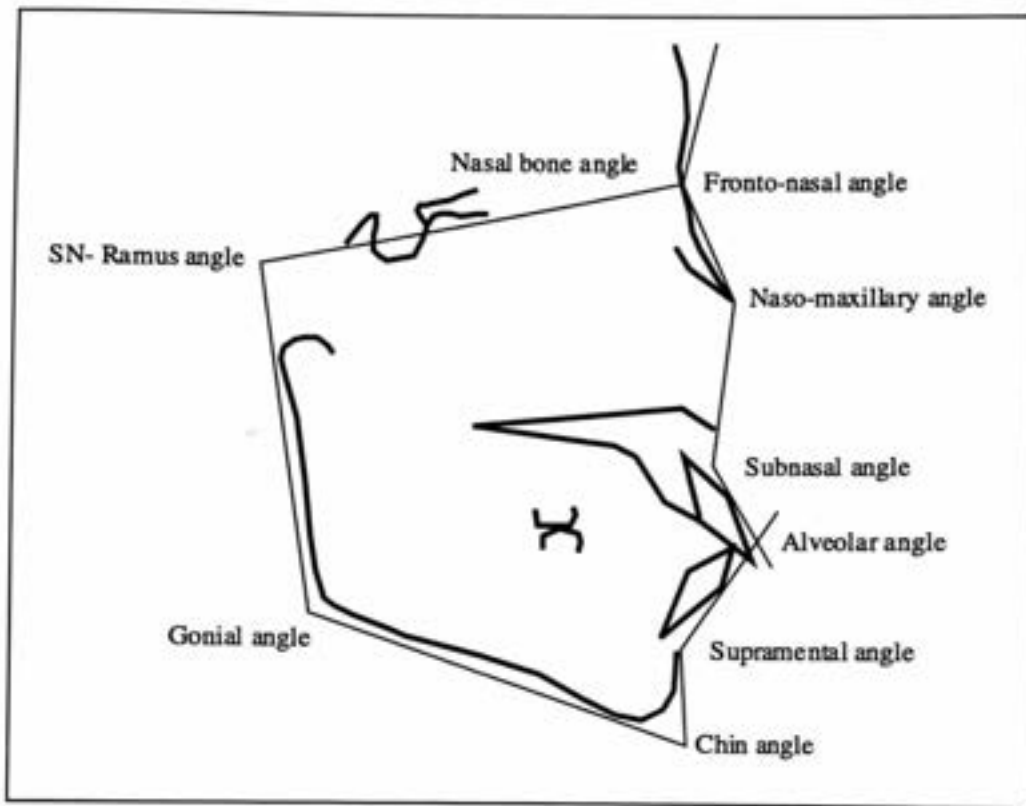


FIG. 65. Facial angles used in assessing the degree of alveolar protrusion. Angles used were nasal bone, nasomaxillary, subnasal and alveolar. After Yen 1973:302

thicker at the frontal. There is no statistically significant relationship between age and mean thickness in either sex. Figure 66 illustrates the cephalographs of two of the individuals with relatively thick cranial vaults.

There are two females with skulls which are markedly thicker than the other individuals, and in one of these, B77, the thickness is most extreme at the frontal bone, exceeding the next thickest measurement at that point by 3.1 mm (33 per cent). In the other female, B79, the thickest point is at vertex, and the measurement at the frontal bone is within the range in other females. Two males (burials 38, 42) have markedly thicker measurements at vertex and bregma, but the frontal bone thickness is again within the range of other individuals.

The cranial vaults of fifteen adults, ten females and five males, were damaged in such a way that a section of the parietal bone was exposed and able to be measured. These measurements had to be taken at the most accessible point on the parietal, and are therefore not necessarily directly comparable with each other as the thickness of both the vault and the internal structures varies in different areas. Nevertheless, the ratios give some indication of whether thicker vaults are a result

TABLE 69: *Cranial vault thickness means and standard deviations for both sexes. Measurements are from lateral cephalographs. Vertex = the highest point of the calvarium, measured perpendicular to a line drawn between porion and orbitale. Frontal = midway between bregma and nasion (mm)*

	Frontal	Bregma	Vertex
Females			
Mean	7.68	6.91	7.47
Standard deviation	1.65	1.47	1.30
Males			
Mean	7.45	8.67	9.00
Standard deviation	1.09	1.88	1.29
Males vs females	ns	t=2.8, df=27 p<.01	t=3.1, df=27 p<.01

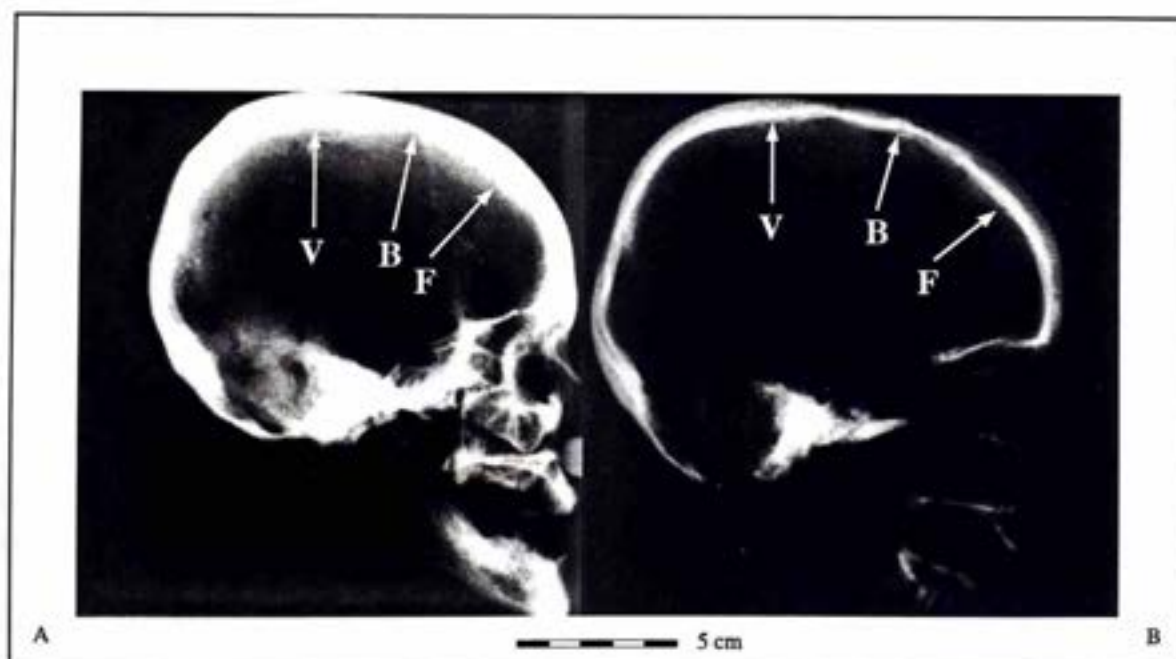


FIG. 66. A: cephalograph of burial 18 (female, 42 years) with vault thickness measurements within the upper range from Khok Phanom Di. F=frontal 9.24 mm, B=bregma 7.32 mm V= vertex 7.41 mm. B: cephalograph of burial 4 (female, 26 years) with vault thickness measurements within the upper range from Khok Phanom Di. F=frontal 8.78 mm, B=bregma 5.40 mm V= vertex 7.87 mm

of hypertrophy of the diploe and thinning of the outer table, or are overall thicker than average with internal structure of normal proportions. The thickness of the bone, and the ratio of cortical bone to diploe for these individuals are listed in table 88. In nine of ten females in the sample the vault thicknesses ranged from 6.5–9.7 mm, with a mean of 8.4 mm (s.d. 1.6) and in all males ($n=5$) the range was 6.3–9.5 mm, with a mean of 7.5 mm (s.d. 1.3). The ratio of diploe to cortical bone was less than 2.5:1 in all except one female. The exceptional individual was an older female (B56) with a parietal thickness of 11.7 mm and a ratio of diploe:cortical bone of 3.6:1 (Fig. 67A). This measurement was taken on the right parietal, slightly anterior to the parietal boss.

The condition of the diploe was difficult to assess, as in many cases the calcareous deposit which coated the outer surface of the cranium had infiltrated the interstices of the diploe and coated the diploic spaces, giving a false impression of thickening of the individual trabeculae (Fig. 67B). On the basis of the subjective observation of exposed diploe, none appeared to have an unusual structure in the form of thickened trabeculae or intertrabecular spacing.

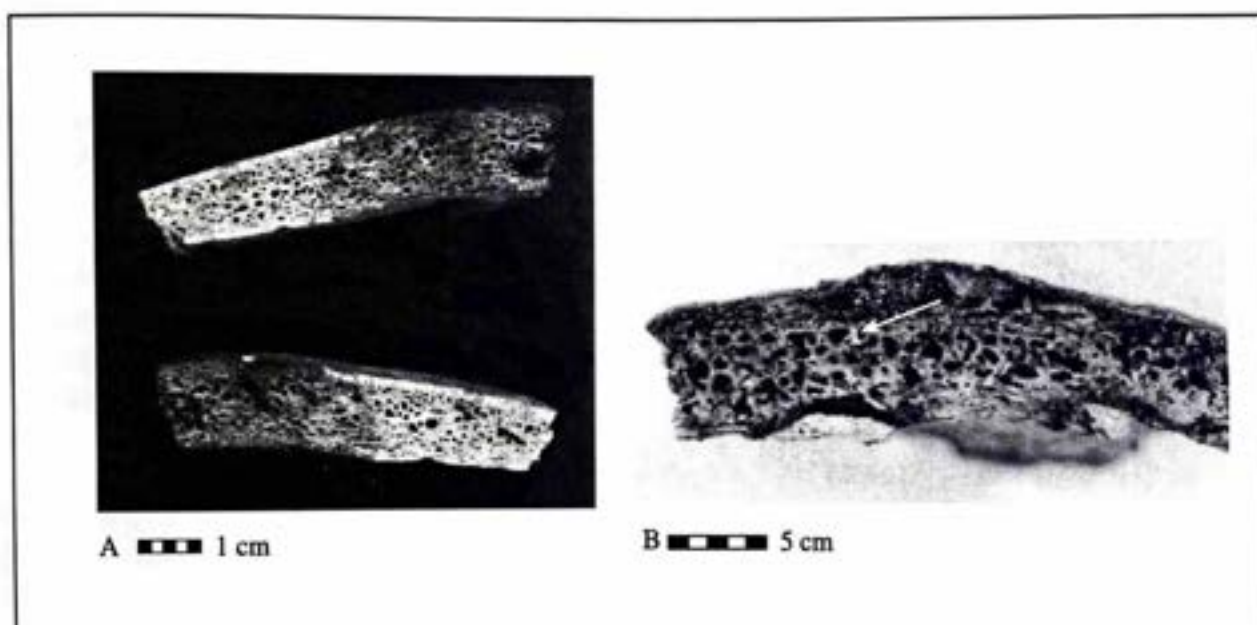


FIG. 67. A: cross-section of vault of burial 56 showing hypertrophy of the diploe and thinning and porosity of the outer table. The discolouration on the bone occurred post-mortem. B: cross-section of a cranial vault with the diploic trabeculae coated with a calcareous deposit. The arrow indicates an example of a trabeculum with a clear coating which represents the deposit

As the thickness of the calvarium can vary with factors such as age and ethnic group (Ethier 1971), it is not necessarily representative of pathology. It is considered here briefly in order to establish whether or not the calvarial thicknesses of the prehistoric Thai are within the range recorded from *Homo sapiens*. The difficulty of establishing a range of 'normal' values for cranial vault thicknesses is partly a result of the differences in measuring technique mentioned earlier, with the prob-

lem exacerbated by variations between measurements taken from cephalographs and those taken directly from the bone. Despite correction for radiographic enlargement, Brown (1987) found that cephalographs gave reduced measurements when compared with direct measurements of the bone. Vault thicknesses measured at the midline can also be affected by local anatomical features of the endocranium such as sutures and grooves for vessels (Brown 1987).

Some data on cranial vault thicknesses are listed in table 70, to give an indication of the relative thickness of the Khok Phanom Di crania as measured from cephalographs. As noted earlier, the measurements on the parietal bones of damaged skulls were not made at standard points and are not therefore directly comparable with other samples. The measurements quoted from other prehistoric sites in Thailand are apparently only of those individuals deemed to have 'thickened' vaults, but the maximum measurements at the parietal all appear to be about 11 mm, about the same as the maximum at Khok Phanom Di. Ivanhoe (1979) lists thicknesses at mid-frontal and bregma for a variety of individuals and groups of different time-periods from various areas in the northern hemisphere. Neanderthals include individuals with thicknesses up to 11 mm at both mid frontal and bregma, and later Neolithic and Copper Age individuals with thicknesses up to 9–10 mm, although the majority are around 5–7 mm. The pattern of thicker crania in prehistoric groups compared with modern peoples is consistent with the trend to thinner cranial vaults over time, noted in Australian Aborigines by Brown (1987), in Jomon and modern Japanese by Ishida and Dodo (1990), and Near Eastern populations by Smith *et al.* (1985). The reduction in cranial thickness is associated with the general temporal reduction in skeletal robusticity (Brown 1987).

There is no clear pattern of age-effect on thickness. Different studies give conflicting evidence on variations in thickness with age (Todd 1924, Smith *et al.* 1985, Ishida and Dodo 1990) and sex (Adeloye *et al.* 1975, Brown *et al.* 1979, Smith *et al.* 1985, Ishida and Dodo 1990). Statistically significant differences often depend on the site of measurement of the skull. Females tend to have slightly thicker crania at the midfrontal, and males at bregma and vertex. In most populations, the thickness of male skulls tends to exceed that of females, but with differences rarely statistically significant (Brown 1987). Brown (1987) suggests that larger skulls tend to have slightly thicker vaults, but with considerable variation. There is no correlation between stature and vault thickness either in the Khok Phanom Di population or in the Pleistocene Aborigines studied by Brown (1987).

Sangvichien *et al.* (1969:33) described the thickness of the flat bones of the skull of seven individuals from Ban Kao as 'extreme'. They recorded parietal bone thicknesses of 7–11 mm, with the 'thickening' being caused by expansion of the diploic tissue, which was also of much coarser texture than normal. They note that:

"Such thickness and coarseness of the diploic tissue occur most commonly in thalassemia diseases, although they may be observed (rarely and not to the same extent) in chronic severe iron deficiency anemia and in other congenital hemolytic anemias."
(Sangvichien *et al.* 1969:33)

On the basis of the prevalence of a variety of thalassaemia syndromes (inherited haemoglobinopathies) in Thailand today, they ascribe the condition to β -thalassaemia-Haemoglobin E disease in the case of one individual with a parietal thickness of 11 mm. They suggest that both HbE and β -thalassaemia must have been present in the inhabitants of Thailand for at least 3000 years and

TABLE 70: *Thickness of the cranial vault (mm), measurements from cephalographs or directly from crania in some skeletal samples. Frontal: on the midline on the sagittal plane, midway between bregma and nasion. Vertex: the highest point of the calvarium, measured perpendicularly to a line drawn between porion and orbitale*

Females	Date (years B.P.)	n	Frontal		Bregma		Vertex	
			Mean	s.d.	Mean	s.d.	Mean	s.d.
Skeletal								
1. Khok Phanom Di	4000–3500	18	7.68	1.65	6.91	1.47	7.47	1.30
2. Australian Aborigine	Pleistocene	8	8.40	1.73	8.40	1.07	7.40	0.50
	Recent	52	7.50	1.35	7.80	1.39	7.70	1.35
3. N.Z Maori	Recent							
- North Island		6	-	-	5.57	1.08	5.97	1.09
- South Island		5	-	-	7.14	0.24	7.16	0.43
- Chatham Islands		7	-	-	6.67	0.98	6.90	0.50
4. Japanese - Jomon	6000–2000	18	-	-	7.90	3.51v	-	-
5. Japanese	Modern	47	-	-	6.00	1.43v	-	-
6. European	Modern	200	4.10	-	4.92	-	-	-
Living								
8. Australian Aborigine		31	-	-	7.60	0.90	7.10	0.90
Males								
Skeletal								
1. Khok Phanom Di	4000–3500	11	7.45	1.09	8.67	1.88	9.00	1.29
2. Australian Aborigine	Pleistocene	20	10.40	2.41	10.80	1.93	9.20	1.68
	"	4	8.70	1.64	10.10	1.44	9.70	3.04
	Recent	47	7.80	1.58	8.90	1.50	8.70	1.44
3. N.Z Maori	Recent							
- North Island		10	-	-	6.40	1.14	6.33	0.94
- South Island		10	-	-	7.34	0.86	7.10	1.21
- Chatham Islands		12	-	-	6.95	1.45	6.58	0.51
4. Japanese - Jomon	6000–2000	26	-	-	8.80	1.80v	-	-
5. Japanese	Modern	105	-	-	6.30	1.59v	-	-
6. Chinese	Modern	49	4.62	-	6.42	-	-	-
7. European	Modern	200	4.89	-	5.65	-	-	-
Living								
8. Australian Aborigine		28	-	-	7.30	0.80	7.20	0.80

v = variance. Data sources: 1. This study, 2. Brown 1987, 3. Leeuwenberg 1990, 4, 5. Ishida and Dodo 1990, 6, 7. Ivanhoe 1979, 8. Brown *et al.* 1979

have persisted to the present because of some selective advantage such as improved immunity to the malarial parasites (1969:34). In the single individual from the site of Sai Yok which is geographically close but chronologically earlier than Ban Kao, Jacob (1969:52) notes that the cranial bone is "quite thick for *Homo sapiens*", but qualifies this by stating that the diploe is not pathological and is intact. In the plate illustrating a cross-section of the parietal bone the scale suggests a thickness of c. 9 mm (Jacob 1969:41). Both the inner and outer tables appear to be very thin, although the outer surface may have been abraded postmortem.

Pietrusewsky reports 'thickening of the diploe' or 'porotic hyperostosis' in 14 of 23 adult skulls of both sexes at Non Nok Tha (1974a:23). The skulls had average thicknesses of 8–11 mm in the parietal bones and 8–10 mm in the frontal bones, but he notes that there were no 'irregularities' of the outer table. Again at Non Pa Kluay, a majority of adults (5/7) had thickened cranial vaults, but with no external table thinning or porosity (Pietrusewsky 1988b). Wiriyaromp (1984a), and Houghton and Wiriyaromp (1984) recorded no cranial vault pathology at Ban Na Di.

The mean thicknesses of the cranial vaults of the Khok Phanom Di population at bregma are within the range of the means of the other prehistoric groups listed. The health status of the other prehistoric groups is not known but it cannot be assumed that a relatively thick vault is pathological in the absence of other evidence for disease. Despite this conclusion for the Khok Phanom Di adults, there are individuals within the group with other pathology which, in combination with thicker calvaria, suggest disease. All evidence for each individual is discussed later in this chapter.

Cephalographs were taken of intact crania of five children aged between five and fourteen years at death. The internal structure of the vault was visible on four individuals. The thickness of the vault and the diploe and cortical bone are listed in table 89, with the ratio of diploe to cortical bone (inner and outer tables combined). The overall cranial thickness varies, with B11 having the thickest vault, within the range of the adult vaults. Despite the thickness, the ratio of diploe to cortical bone is not high. At mid-frontal, the ratio ranges from 0.68:1.00 to 1.21:1.00 in the four individuals.

On those children where the vault was damaged and the internal structure exposed, the diploe appear of normal thickness and structure and the outer table appears intact. Thicknesses measured at midfrontal and on the parietal bones are listed in table 90. As with the adults, there is no control over the point of measurement on the parietal. The only measurement which is relatively thick is that of the parietal bone of B145.

The thicknesses of cranial vaults in children measured from radiographs can be compared with longitudinal data collected from a small sample ($n=32$) of healthy white American children (Roche 1953). Thicknesses at midfrontal in this sample during the ages of nine to twelve years range from 4.3–4.8 mm, at bregma detailed data are not given but 4.0 mm is a minimum, and at vertex the range is 4.7–5.2 mm. Of the four children in this age group from Khok Phanom Di, B37 had a skull thickness slightly outside this range at midfrontal, and B11 had a thicker vault at all points. However, none of the crania had diploe/cortical bone ratios which indicated that the thickness of the vault was the result of diploic expansion. Rather the thickness appears to reflect either an inherited form at the upper end of the normal range in humans, or a biomechanical effect.

The prevalence of cribra orbitalia at Khok Phanom Di was presented earlier in this chapter but the significance is considered further here, in the context of skeletal evidence for anaemia. Cribra orbitalia has been relatively commonly reported in prehistoric skeletal populations, but the possibility

of interobserver variance in the classification of the bony evidence reduces the value of comparison of prevalence among populations. The proportion of the adult Khok Phanom Di population with cribra orbitalia of all grades is low at 5 per cent compared with that in a wide variety of eleven populations (total sample size = 6948) summarised by Stuart-Macadam (1985:396), where the reported proportions range from 6–45 per cent, and with a series of nine diverse Pacific populations reported by Suzuki (1987) with a smaller range of 8–15 per cent with cribra orbitalia. Hengen (1971) lists a series of five small samples from tropical and subtropical countries with an overall mean of 50 per cent of adults with cribra orbitalia of all grades, with 26 per cent having lesions of grade 2 or higher. Whether the differences are due to interobserver variation, however, is not known.

A higher proportion of the children than of adults has cribra orbitalia. Those who survived beyond infancy are particularly affected. The grade in most individuals is greater than for adults. That more children have a higher grade of cribra orbitalia than adults is a common finding in anthropological studies, as listed by Stuart-Macadam (1985:396). In this selection, the proportion of children with lesions is significantly higher than the proportion of adults with lesions in all eleven populations. This may indicate that individuals with severe anaemia do not survive beyond childhood, or may reflect the greater susceptibility of the bone to remodelling during the growth period. Stuart-Macadam (1985) suggests that the lesions in adults are the remodelled legacy of childhood anaemia, with the orbit unlikely to respond to anaemia acquired in adulthood.

The proportion of children with cribra orbitalia at Khok Phanom Di is higher than that in any of the eleven populations listed by Stuart-Macadam (1985), except a group from a leprosy hospital cemetery in Denmark (Møller-Christensen 1953). This suggests that anaemia was an important element in childhood morbidity at Khok Phanom Di, whether or not it contributed to mortality.

The thickness of the orbital plates of the Khok Phanom Di children did not reach more than 1 mm, and although no standards for the thickness of this structure in children in general have been found, this is well below the 3 mm thicknesses found by Stuart-Macadam (1987a) to be more prevalent in children with cribra orbitalia than in children without the evidence of anaemia. Few of the incomplete adult skulls had upper orbital plates (the orbital plate of the frontal bone) which were intact and able to be measured for thickness. The single individual on which the plate was intact and where the plate was thickened with cancellous bone was B92. The thickness at the centre of the plate was 3.8 mm. In the only other skeletal report to consider the thickness of the orbital plates, Stuart-Macadam (1987a) found thicknesses of 3 mm or higher only in individuals with other orbital lesions in two series. The single individual at Khok Phanom Di with a thickened orbit roof (B92) had light cribra orbitalia.

The formation of frontal sinuses was observed on antero-posterior cephalographs of adults. Thirteen per cent were completely absent (4 of 29 crania radiographed) and 6 per cent unilaterally absent (2 of 29). Two of the individuals without sinuses at Khok Phanom Di had retained metopic sutures, an inherited trait which is often found together with the lack of formation of frontal sinuses (Sedwick 1934). In adults, the frontal sinus is completely absent in 5 per cent and unilaterally absent in 1 per cent, according to Schuller (1943), although it is not clear whether this frequency is of those of genetic origin or includes the consequences of environmental disturbance of the development of the sinus.

Three infants (burials 101, 121, 150; aged 15–30 months) have cranio-facial bones which are thicker than those in other infants in the sample and in crania of normal infants in the collection in the Anatomy Museum at the University of Otago. The antero-lateral sections of the frontal bones and the zygomatic bones have hypertrophied. This is particularly evident in the antero-posterior dimension of the zygomatic bones, with rounded edges on the orbital rim and inferior margins (Fig. 68). The individuals and the affected bones are listed in table 71.

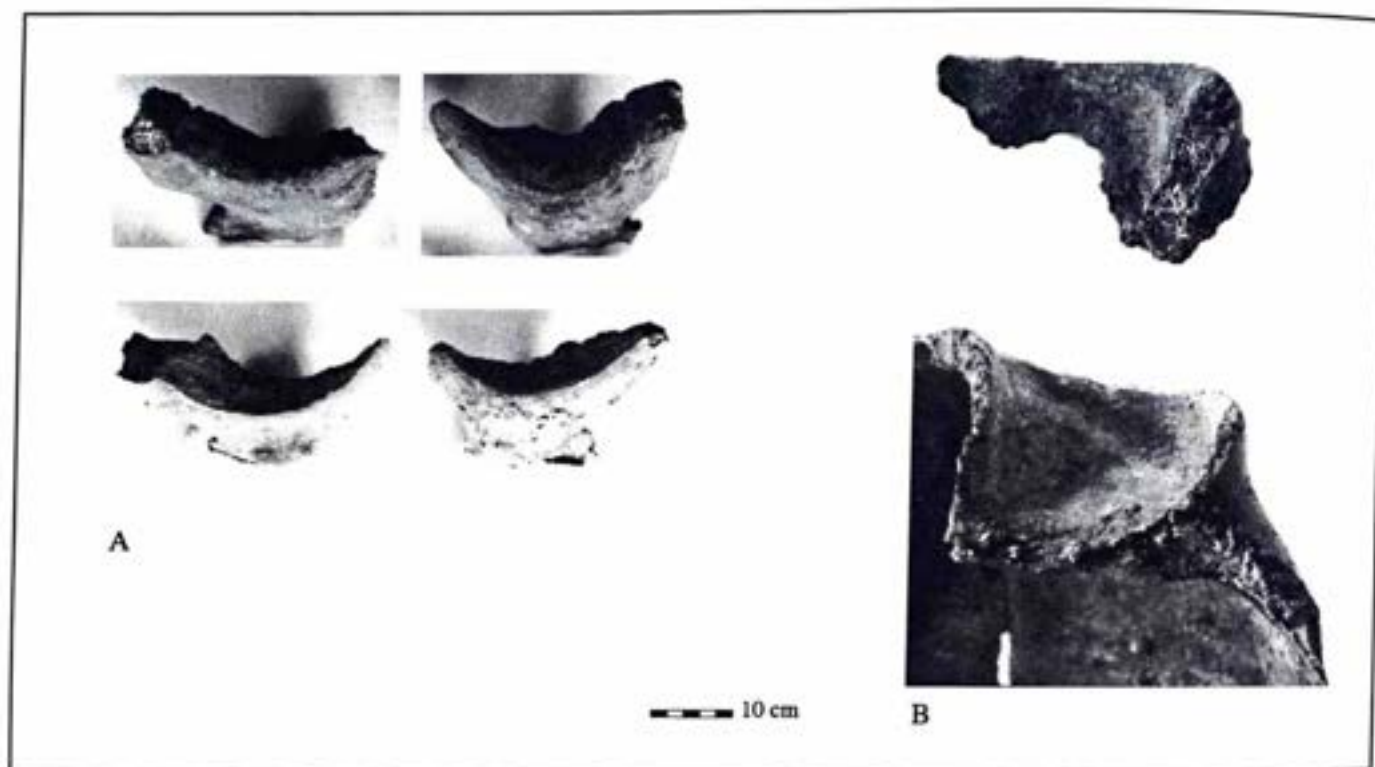


FIG. 68. A: antero-superior view of infant zygomatic bones. The two upper bones (burials 101, 121) showing hypertrophy and rounded contours compared with the more angular outline of the two normal bones from infants of the same age from Khok Phanom Di (lower). B: inferior aspect of antero-lateral frontal bone of infant burial 121 (upper) and normal infant of the same age (lower). The bone from burial 121 illustrates hypertrophy at the zygomatico-frontal suture compared with the normal bone. Porosity indicating cribra orbitalia is also visible in the orbital plates

The hypertrophy of the facial bones of these infants is believed to be evidence of the skeletal response to marrow hypertrophy reflecting anaemia. There is no reactive bone or other evidence of local infection. No other diseases are known to produce the same skeletal response.

Measurements of the facial angles taken from cephalographs of children are listed in table 91. In all four children, the central maxillary incisors protrude anteriorly beyond the mandible, and in three children (burials 11, 17, 21) the lateral incisors also protrude. Despite this, these teeth do not appear widely spaced as is reported in some clinical cases of maxillary protrusion (Caffey 1951),

TABLE 71: *Hypertrophy of cranial bones in children*

Burial	Age (years)	Observation
101	1.50	Hypertrophy of the zygomatic bone
121	1.25	Hypertrophy of the greater wing of the sphenoid, the zygomatic bone, the frontal process of the maxilla and the frontal bone, posterior to the zygomatic process
150	2.50	Hypertrophy of the zygomatic bone

and the anterior teeth of B37 are actually crowded. On the basis of a subjective assessment, seven of the nine children with intact faces appear to have maxillary protrusion, although it appears to be a reflection of the morphology of the alveolar process rather than hypertrophy of the maxilla. Those which are prognathic are burials 11, 12.1, 17, 21, 32, 37 and 145. Burials 1 and 6 are not affected. Figure 69 illustrates the cranium of B37.

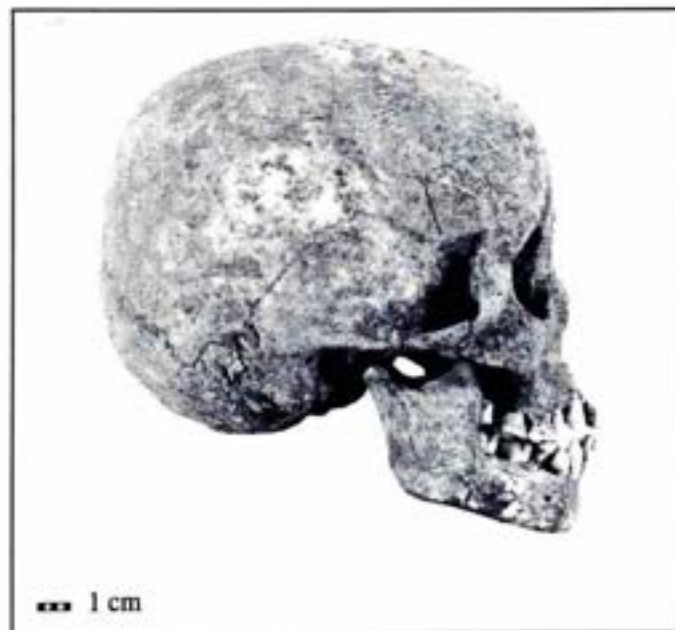


FIG. 69. Cranium with alveolar protrusion and crowding of the anterior teeth

The measurements of bi-jugal and bi-infraorbital foramina widths in children are listed in table 92. The ratio of bi-jugal to bi-infraorbital width is similar in children to that in adults. Two

individuals, burials 1 and 17, have relatively wide lateral growth of the midface. B37 has a wide face, with the bi-infraorbital foramina width within the range of adult widths.

TABLE 72: *Facial profiles in Khok Phanom Di children and Chinese boys (Yen 1973, mean age 11.8 years)*

Angle	Khok Phanom Di			Chinese		
	n	Mean	Range	n	Mean	Range
Nasal Bone	1	87	-	50	100	87-114
Naso-maxillary	1	162	-	50	148	125-168
Subnasal	1	133	-	50	142	123-157
Alveolar	3	112	103-120	50	129	107-150

No data on the degree of maxillary protrusion in modern Thai children have been located, but Yen (1973) has published data for Chinese boys from Taiwan (Table 72). Yen found that the Chinese facial profile is straighter than that of Caucasians, except in the area of the mouth where both the alveolar bone and the incisors have a "marked labial inclination" (1973:302). The profiles of the small sample of Khok Phanom Di children have the same characteristics, but to a greater degree. The angles of the upper face are within the range recorded on the Chinese but at the extreme of 'flatness', and the alveolar angle shows a greater mean alveolar protrusion than the Chinese.

The measurements of the degree of maxillary protrusion in the facial skeleton of adults are listed in table 93. Two females, burials 60 and 64, and two males, burials 24 and 30, have maxillae with marked protrusion. In a high proportion of adults the maxillary incisors have been ablated so it is not possible to judge whether there was dental malocclusion. In those adults with intact dentition, the lateral radiographs show that the anterior dentition on both the maxilla and mandible protrude but this appears to be a reflection of alveolar prognathism rather than hypertrophy of the maxilla itself. No adults have the malocclusion of incisors evident in the children.

Maxillary protrusion as indicated by the angle sella-nasion-subspinale in the Khok Phanom Di population is similar to that in healthy modern Thai, as reported by Sorathesn (1988). Table 73 lists the summary statistics. The differences between the Khok Phanom Di and the modern Thai means are not statistically significant.

The measurements of the degree of lateral hypertrophy of the mid-facial skeleton (bi-jugal and bi-infraorbital foramen widths) in adults are listed in table 94 and summarised in table 74. The extent to which the maxilla and zygomatic bone have developed laterally is indicated by the ratio of the bi-jugal width to the width of the neural template as indicated by the bi-infraorbital foramina width. In all individuals the bi-jugal width is over twice the bi-infraorbital width. Extreme development of the lateral midface in one female (B4) and three males (burials 24, 30 and 38) is indicated by the ratio of the two width measurements.

Sorathesn (1988:199) mentions the 'characteristic bimaxillary protrusion' in Thai compared with Caucasians. As there are no data available for the bi-infraorbital width in other populations, it

TABLE 73: *Mean maxillary protrusion as indicated by the angle sella-nasion-subspinale (SNA) measured from cephalographs*

Females	n	Mean	Standard deviation
Khok Phanom Di	13	84.08	4.44
Modern Thai ¹	50	85.02	3.73
Males			
Khok Phanom Di	9	85.22	4.49
Modern Thai ¹	50	83.02	11.24

¹ Sorathesn 1988

TABLE 74: *Bi-jugal and bi-infraorbital widths used in the assessment of lateral hypertrophy of the maxilla and zygomatic bones (mm)*

	Females (n=18)			Males (n=11)		
	Mean	s.d.	Range	Mean	s.d.	Range
Bi-jugal	119.4	4.1	113.5–125.8	123.6	7.5	109.8–131.9
Bi-infraorbital	56.2	2.5	51.0–61.8	57.2	3.9	51.5–61.7
Ratio	1:2.12		1:2.03–1:2.27	1:2.16		1:2.03–1:2.49

Bi-jugal = Martin's measurement 43(1) (Martin and Saller 1957) Bi-infraorbital = width between the infraorbital foramina

is not possible to draw any comparison of the relative degree of lateral facial development at Khok Phanom Di. However, Pietrusewsky (1988b) gives bi-jugal widths for samples from Non Pa Kluay. These are listed in table 75.

Both sexes in the Khok Phanom Di population have relatively wide faces compared with their North East Thai contemporaries. The difference between Khok Phanom Di and the small sample from North East Thailand is statistically significant only in the females ($p < 0.008$).

In no cases at Khok Phanom Di had the maxillary air sinuses failed to form. Of the sample of 29 antero-posterior radiographs, four adults, three females (burials 4, 35 and 73) and one male (B24), have maxillary sinuses which are relatively small compared with the rest of the group. Four children have faces sufficiently intact for the condition of the paranasal sinuses to be visible on cephalographs. The subjective assessment of the development of the sinuses is listed in table 76. The maxillary sinuses are unclear in the radiographs but in three of the four cases, the frontal sinus was very small.

The frontal sinus grows steadily from birth until the ninth or tenth year and more rapidly after that until about 20 years (Maresh 1940). Given that this is a very general statement, the relatively

TABLE 75: *Mean bi-jugal widths from prehistoric South East Asian populations*

Females	n	Mean	Standard deviation	Range
Khok Phanom Di	18	119.4	4.1	113.5–125.8
Non Pa Kluay	2	115.0	4.2	112.0–118.0
Males				
Khok Phanom Di	11	123.6	7.5	109.8–131.9
Non Pa Kluay	2	116.5	6.4	112.0–121.0

Martin and Saller (1957) measurement 43(1) in mm. Data sources: Khok Phanom Di - This study
Non Pa Kluay - Pietrusewsky 1988b

TABLE 76: *Formation of air sinuses in children*

Burial	Age	Formation of cranial air sinuses
11	12	The maxillary sinuses are not clear on the radiographs as they are filled with dirt but gross observation shows at least some development. The frontal sinus has developed on the right only and is small. The mastoid air cells have developed
17	12	All sinuses have developed.
21	8	The maxillary sinuses are unclear, the frontal sinus is small, and the mastoid air cells are undeveloped.
37	9	The maxillary sinuses are unclear, the frontal sinuses are very small, and the mastoid air cells have developed

small frontal sinuses on the Khok Phanom Di children are consistent with their pre-pubertal age rather than suggesting that the sinuses have failed to form.

Anaemia - discussion

This section draws together the results of the investigation of skeletal characteristics which may be indicative of anaemia and attempts to identify individuals with the condition. The evidence of the orbital plates (cribra orbitalia) suggests that three-quarters of those who died in childhood had suffered or were suffering from anaemia. As no data on the 'normal' range of variation of thickness of the cranial vault or of the ratio of diploe to cortical bone in children has been found in the literature, measurements of vault thickness or ratios are not diagnostic. On the basis of qualitative assessment, none of the children with cribra orbitalia had porotic hyperostosis of the cranial vault. Stuart-

Macadam (1992) notes the possibility that the two areas of the skull can respond independently, and that lesions in the orbit commonly occur without the vault being affected. Cribra orbitalia may be one of the first, and often the only, lesion indicative of anaemia to appear in the skeleton (Stuart-Macadam 1989). The investigation into the evidence for anaemia in the adult population at Khok Phanom Di shows that the thickness of the adult cranial vaults alone cannot be assumed to reflect a response to anaemia. There was only one individual (B56), with a ratio of diploe to cortical bone approaching the mean of 1:4.4 found in the sickle-cell anaemics recorded by Reynolds (1962: cited in Stuart-Macadam 1987a:513). The remainder are within the range of ratios in normal individuals in Reynold's study. Although there is a lack of control of the site of the measurement, the normal internal structure of the vault is consistent with the suggestion that the thickness of the vault in the Khok Phanom Di people is not necessarily a response to marrow hypertrophy. In common with other prehistoric groups, it may have a structural or functional explanation.

The skeletal evidence nevertheless suggests that the aetiology of at least some of the cases of anaemia at Khok Phanom Di appear to have been genetic anaemia. This includes the infants with hypertrophy of the facial bones. Hypertrophied facial bones "...almost certainly establish the diagnosis of thalassaemia major." (Moseley 1971:698). The Khok Phanom Di infants with hypertrophy of the facial bones have no evidence of cribra orbitalia or porotic hyperostosis of the vault. Again, the age at death may be the relevant factor in the nature of the skeletal response. Hypertrophy of the facial bones was not evident in older children at Khok Phanom Di, although several, including those with cribra orbitalia, had wide, flat faces with protrusive maxillae. In the absence of readily accessible detailed data on the range of normal dimensions of the juvenile Southeast Asian face, these features could not be accepted as conclusive evidence that this morphology represented a departure from the normal.

The data collected from each individual in the investigation of anaemia is considered in conjunction with non-specific indicators of health and with skeletal pathology which is not specific to anaemia but which may be a consequence of the anaemia or of a primary disease. The diagnosis of anaemia in an individual takes into account health characteristics not specific to anaemia, but diagnostic in the presence of one or more specific indications. Such factors include cortical thickness and age at death.

Infants with evidence of anaemia are burials 88, 101, 105, 121 and 150. In the first four, the evidence is cranial (Table 77). In some cases there is also evidence of growth disruption or enlarged medullary cavities and thinned cortical bone in the diaphyses (Fig. 70), which are all potentially associated with episodic or chronic ill health. Although the poor health cannot be specifically attributed to severe anaemia or one of the underlying diseases, it is not inconsistent with such a diagnosis.

The skull of B88 is damaged, but there is no evidence of cribra orbitalia, hypertrophy of the facial skeleton or diploic expansion of the cranial vault on the fragments which are present. The diagnosis of anaemia is based on the condition of the long bones, which is very similar to that described in a case report of an eight-year-old Thai child with β -thalassaemia major (Singcharoen 1989). The child was still suffering from 'severe anaemia and frequent episodes of congestive heart failure' despite treatment (Singcharoen 1989:168). These conditions would have been fatal at an early age in a prehistoric child. The modern Thai child had the diploic widening and classical 'hair-on-end' ra-

TABLE 77: *Individuals with skeletal pathology suggestive of anaemia*

Burial No.	Sex	Age	Evidence of anaemia and general health
21	Child	c. 8 years	Anaemia: moderate cribra orbitalia ; extreme cortical porosity and thinning, hand phalanges and fifth metatarsals
24	Male	c. 25 years	Anaemia: malformation of proximal humerus. General Health: short stature
56	Female	c. 45 years	Anaemia: Cranial vault with expanded diploe and thin tables. General Health: very short stature, thin cortices on long bones
88	Infant	9 months	Anaemia: porotic cortical bone, radial periosteal bone proliferation on long bones. General Health: dental enamel hypoplasia
101	Infant	15 months	Anaemia: hypertrophy of cranio-facial bones (especially anterior frontal bone)
105	Infant	3 months	Anaemia: hypertrophy of cranio-facial bones (especially anterior frontal bone)
121	Infant	15 months	Anaemia: hypertrophy of cranio-facial bones including maxilla, zygomatic, temporal, greater wing of sphenoid, frontal (antero-lateral). Moderate cribra orbitalia . General Health: dental enamel hypoplasia, osteoporosis
150	Infant	30 months	Anaemia: hypertrophy of cranio-facial bones: frontal, zygomatic. General Health: dental enamel hypoplasia, osteoporosis

diographic appearance of the skull, but the apparent absence of this condition in B88 may have been a reflection of the younger age at death of the child.

There is an infant aged less than one year, B99, with minor cranio-facial hypertrophy similar to that described in burials 101, 105, 121, and 150. This may be the early development of a response to anaemia but there is no other skeletal support for the diagnosis. Another infant, B33 (three years), has enlarged nutrient foramina on the hand phalanges. This infant also had evidence of multiple episodes of growth disruption, but the skull was too damaged for assessment of the condition of the facial or cranial bone. There was no evidence of ill-health specifically attributable to anaemia in the remaining eight infants who died between the ages of six months and five years, although in most cases the condition of the skeletons precluded the recording of such evidence. Neonates (n=47) do not show any evidence of anaemia.

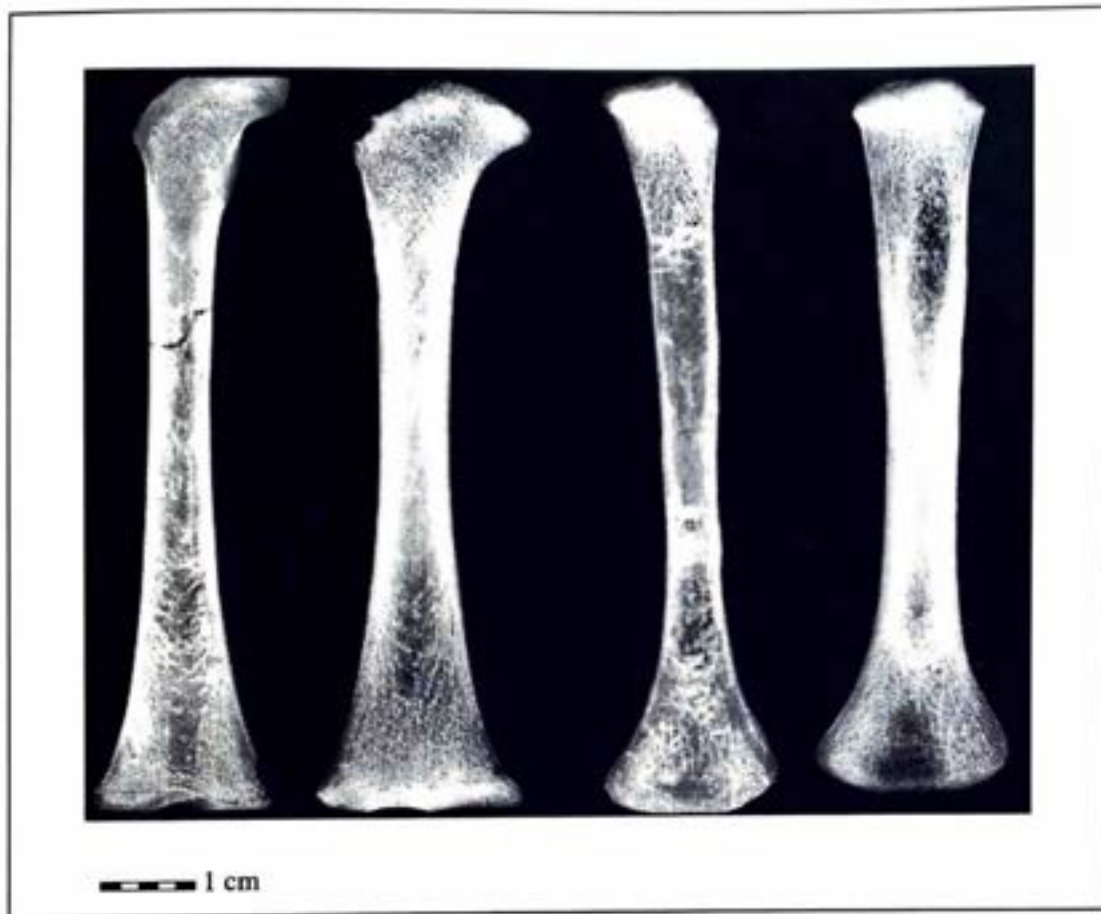


FIG. 70. Antero-posterior radiograph of infant humeri and femora from Khok Phanom Di. The bones on the left in each case (femur burial 150; humerus burial 121) illustrate thinned cortices and widened medullary cavities compared with the normal bones on the right from an infant of the same age

Of the children, B21 had severe cribra orbitalia in addition to the extreme porosity of the small bones of the hands and feet and enlarged nutrient foramina. Apart from this individual, five other children had cribra orbitalia. Three (burials 12.1, 32, 37) also had enlarged nutrient foramina in the phalanges but no other unequivocal pathology. All children had evidence of growth disruption and B12.1 of growth retardation.

The skeletal evidence in individuals surviving beyond the critical periods of infancy and childhood can be expected to be less distinctive than in those where the condition proved fatal early in life, as has been previously discussed. The disease must have been relatively mild for the individual to have survived. Of the 68 adults, there are two (burials 56 and 24) with multiple skeletal characteristics possibly indicative of anaemia. The one female, B56, is the only adult with demonstrated cranial vault thickening attributable to diploic expansion. She also has possible maxillary hypertro-

phy. She was a very tiny woman, by far the shortest in the population (141 cm, population mean 154 cm S.D. 4.5 cm), with evidence of multiple episodes of growth disruption. This suggests ill health in childhood, but despite that she was fertile and had lived to a good age. She suffered from osteoporosis, but whether that reflects age-related involution or poor development during early life in response to ill-health is not possible to discern. Although a confident diagnosis of anaemia cannot be made, the possibility cannot be discounted.

Burial 24 does not have vault changes other than absence of the frontal sinuses. There was no cribra orbitalia or maxillary hypertrophy, although the maxillary sinuses are relatively small. He was of very short stature (estimated stature 154 mm, population male mean 162 mm S.D. 5 mm), but the cortical bone and the muscle attachments were well developed. The small size of the sinuses may be a reflection of small body size. He had unusual development of the right humerus almost identical to the effect of premature fusion of the proximal epiphyses of humeri described in thalassaemics as previously discussed. This individual survived into his mid-twenties, and appears to have led an active life despite the deformity of one arm. The good development of the cortical bone suggests a healthy life, and appears to be at variance with the shortness of his stature.

Burial 44 has a thickened cranial vault, and is the only individual with associated porosity of the outer table, although this is not symmetrical. He also had no frontal sinuses, but this is likely to be associated with retention of the metopic suture (Schuller 1943). Metopism is generally believed to be a genetic trait, but Reimann *et al.* (1978, cited in Stuart-Macadam 1985:396), found an association between iron-deficiency in childhood and retention of the metopic suture. In a British skeletal collection, Stuart-Macadam (1985) found that the prevalence of metopism was significantly higher in individuals with cranial evidence of anaemia than in other individuals. This suggests a possible association of both the metopism and absence of frontal sinuses in B44 with anaemia. He had no cribra orbitalia or other morphological or metrical changes in the face indicative of anaemia. Although the vault changes, combined with thin cortical bone and his early death, do not exclude anaemia as an explanation for his condition, he is not included as an anaemic individual because of the range of other possible diagnoses.

Several other adults had a variety of individual or minor characteristics possibly attributable to anaemia. Three young adults, two males (burials 92 and 103) and one female (B60) had cribra orbitalia, but otherwise minimal craniofacial evidence of response to anaemia. The males were both short, although B103 had very good cortical bone. Both males had vertebral degeneration, which is unusual for young individuals, although the cortical indices do not indicate premature osteoporosis as an explanation. The females had evidence of multiple growth disruption but, again, good cortical bone. Other adults with equivocal characteristics which may indicate anaemia include B107, a female who had enlarged phalangeal nutrient foramina and enlarged vascular impressions in the skull. Lawson *et al.* (1984) describe both these conditions in thalassaemic patients. This individual was also gracile and osteoporotic, but had survived into her forties. Burial 40 (female) also had enlarged vascular impressions and a slightly thickened cranial vault. Burial 4 (female) has craniofacial changes, including possible hypertrophy of the maxilla. There was no cribra orbitalia. She was short, but had good cortical bone, was fertile and had survived into her mid twenties. Burial 18 had a thickened frontal bone and the frontal sinus was unilateral. She was short, and had severe degeneration of the vertebral column. Burials 38, 77, 83, 147, and 154 (three females, two males)

also had some possible indications of anaemia but these were in isolation or of a minimal degree. Burial 94 has no specific indication of anaemia but has reactive bone on the distal tibia which is evidence of periostitis. This may have been a response to leg ulcers, which is a common condition in tropical countries, including among thalassaemics.

Burial 28, tentatively estimated to be male, is an enigma. This individual was short, with extreme osteoporosis, a thicker than average cranial vault, and sexually ambiguous pelvic morphology. Failure or delay of puberty has been recorded in homozygous β -thalassaemics (Weatherall and Clegg 1981), and this may account for lack of development of a clear sexual dimorphism of the pelvis. All the teeth had been lost from the maxilla, but the iliac crests were not fully fused. There was slight evidence of growth disruption. The osteoporosis and tooth loss suggest that this was one of the older individuals in the group, but the unfused epiphyses are not consistent with this assumption. The persistence of epiphyseal lines long after the expected time of fusion is common in thalassaemics (Baker 1964), raising the possibility that the estimated age of 21 years is too young. It is conceivable that this individual was anaemic, but all characteristics are generalised and may be symptomatic of an unidentified systemic illness.

Of the individual cases described above, all include thalassaemia as a differential diagnosis, despite aspects of non-conformity with standard clinical descriptions. A difficulty with using the evidence described and illustrated in clinical reports as a basis for making a diagnosis from dry bones is that clinical reports tend to describe the more extreme cases, where the evidence is radiographically visible. As a consequence of this, many textbooks describe these extreme skeletal reactions as if they should be expected in any individual suffering from a disease. An example is the already-mentioned tendency to describe the 'hair-on-end' appearance on radiographs of skulls as common in thalassaemics. Many of the clinical reports of cases in older children and young adults, particularly in recent years, are of individuals with severe anaemia who have been treated with blood transfusions. Without treatment, many children and infants with a severe manifestation of the disease would have died at an earlier age. Cases of mild anaemia are not of the same clinical interest. The bony changes in the reported cases are not necessarily directly comparable with prehistoric evidence from individuals of a similar age. It is also clear from the clinical literature that skeletal responses to chronic anaemia, and to thalassaemia in particular, are many and varied and to expect to see all, or even many, of them in a particular population is unrealistic. For example, although there were no children with the 'classic' lesion of porotic hyperostosis, there are reports of clinical cases of genetic anaemia in Asia where the cranium was less affected than the post-cranial skeleton (Lie-Injo 1958, Nagaratnam 1989). The absence of cranial vault lesions does not negate the diagnosis.

In addition to the individuals described above, others, including infants, had skeletal conditions such as osteoporosis which are included among the characteristics described in thalassaemics. However, given the multitude of bases for the development of this condition, these individuals have been excluded from the discussion. It is not prudent to assume every case of pathology in the population to be indicative of thalassaemia. Conversely, that does not exclude the possibility that some or all of these infants were thalassaemic and that the bony response had simply not developed to the point where distinctive changes were evident. It may be that the cause of death in children in these circumstances is not necessarily the anaemia but any one of the infections, such as intesti-

nal parasites to which children in tropical climates could have been exposed. Anaemic children are particularly susceptible to infection (Weatherall and Clegg 1981), and therefore the extent of bony changes consequent on the anaemia are not necessarily correlated with age at death.

Conclusions which can be drawn on the prevalence of anaemia on the basis of the skeletal changes described must be recognised to reflect a minimum. Given the potential for anaemia to develop from a variety of causes, only the individuals with skeletal evidence specific to genetic anaemia, can be included in the calculation of prevalence of the condition in the population. In the Khok Phanom Di sample, ignoring the new-born infants ($n=47$), the minimum proportion of individuals with evidence of genetic anaemia is listed and the evidence summarised in table 77. This gives a total of eight out of 107 individuals. Using the Hardy-Weinberg formula, this would give a minimum estimate of homozygotes for thalassaemia in the population of .065, with up to .44 either hetero- or homozygotes. In Thailand today, at least 40 per cent of the population carries one of the traits. In a relatively homogeneous population such as Khok Phanom Di, the level may be higher than that at any one time.

JOINT REMODELLING

Two of the many factors involved in the functioning of healthy synovial joints are the maintenance of stability and the equitable distribution of load across the joint surface (Bullough 1981). The contributing morphological and physiological features are the geometry of the articular surface and the strength, resilience and elasticity of the articular cartilage and the subchondral bone. Joints are constantly remodelled during life to maintain stability and load distribution in compensation for changes in structure. This remodelling can take place without changes in the articular cartilage (Sokoloff and Hough 1985). The formation of osteophytes on the knee without structural changes leading to osteoarthritis has been documented by Hernberg and Nilsson (1973). The size of the osteophytes increased with age, and more rapidly in those individuals where arthritis was present. Ortner and Putschar (1981) also note the possibility of lipping without osteoarthritic degeneration, but the differentiation of non-pathological osteophytes from those formed as a consequence of the disease in dry bone is not simple.

Arthritis (arthrosis) and phytosis

Although bony remodelling of joints can occur without pathology, progression of the condition results in osteoarthritis in synovial joints or osteophytosis at the fibrocartilaginous joints between vertebrae (Ortner and Putschar 1981). 'Osteoarthritis' is the most widely used clinical term for synovial joint degeneration, although this term is a misnomer as it implies that the condition is necessarily inflammatory. 'Osteoarthrosis' is a more correct description, but is not as widely used. Osteophyte formation on the non-synovial joints of vertebral bodies as a consequence of intervertebral disc degeneration and protrusion is not arthritic in the strict sense, but represents the equivalent condition to that of arthritis (Steinbock 1976, Ortner and Putschar 1981) and is included here. Other, less common, forms of joint disease include unequivocal pathologies such as rheumatoid arthritis, septic arthritis and ankylosing spondylitis.

The degeneration of joints has received a lot of medical attention because of its universality. As there is a clear association with increasing age, the increasing longevity of many populations has resulted in greater awareness of the effects (Kean 1986). The clinical entity and evidence from skeletal collections are not directly comparable, however, as clinical concern is largely with the symptoms of pain, stiffness and immobility. The radiological evidence of the disease is loss of joint space, subchondral sclerosis, cyst formation and the presence of osteophytes (Fergusson 1987). Only 30 per cent of radiographically detectable joint degeneration produces clinical symptoms, with little overt effect on health and no disability in the form of pain or reduced mobility until the degeneration is severe (Masi and Medsger 1989). Non-symptomatic, non-pathological remodelling of the bone in joints with age and biomechanical stress as can be identified from skeletal material has received little attention in living populations, apart from autopsy studies (Moskowitz 1989) and studies such as that of Hernberg and Nilsson (1973) on the relationship of osteophytes in the knee joint and subsequent osteoarthritis.

The aetiology and pathogenesis of osteoarthritis has been widely studied and debated (Fergusson 1987, Sokoloff and Hough 1985). Figure 71 illustrates the wide range of aetiologies which produce the same ultimate bone response, and the complexity of the problem of identifying the causes in any individual. Although degeneration of the cartilage is most commonly cited as the initial lesion, microfractures and 'stiffening' of the subchondral bone have also been suggested as a source of stress on the cartilage (Radin 1976).

Although age itself has an effect on the structure and biochemical composition of joints which is at variance with osteoarthritic changes, "...the overall effect of ageing is to reduce the cartilage's ability to withstand fatigue failure." (Fergusson 1987:440). In modern, western, populations, alteration of the joint with age is widely accepted as a principal aetiological factor in osteoarthritis (Ortner and Putschar 1981, Masi and Medsger 1989). The interpretation of patterns of remodelling requires consideration of all factors involved (Rogers and Waldron 1989). In skeletal studies, biomechanical stress resulting from physical activities has been cited as the principal aetiological factor (Merbs 1983, Goodman *et al.* 1984, Rathbun 1984).

Recent emphasis on sports medicine and occupational health has stimulated interest in the effects on the joints of physical activity, and although the primary focus has been on trauma, there has been some research on the long-term effects of strenuous and repetitive activity. The results of these studies have often been contradictory (Sokoloff and Hough 1985, Masi and Medsger 1989). Most data on occupational associations are based on occupation groups rather than individuals, and do not take into account the exact nature of the physical actions involved, or other aetiological factors such as the age of the individuals (Hadler 1977, Masi and Medsger 1989). Waldron (1989), in a study of eighteenth and nineteenth century skeletons from individuals with known occupations, found that age was a greater aetiological factor than manual activity in osteoarthritis in that population.

Studies on skeletal groups which have considered overall joint degeneration as evidence of variations in physical activity, particularly in relation to the transition from hunting and gathering to agriculture, include a number of papers in Cohen and Armelagos (1984). Merbs (1983) has considered the patterns of joint degeneration in more detail and related them to activities in a Canadian Inuit population. Tainter (1980) has carried the analysis one step further and considered the social

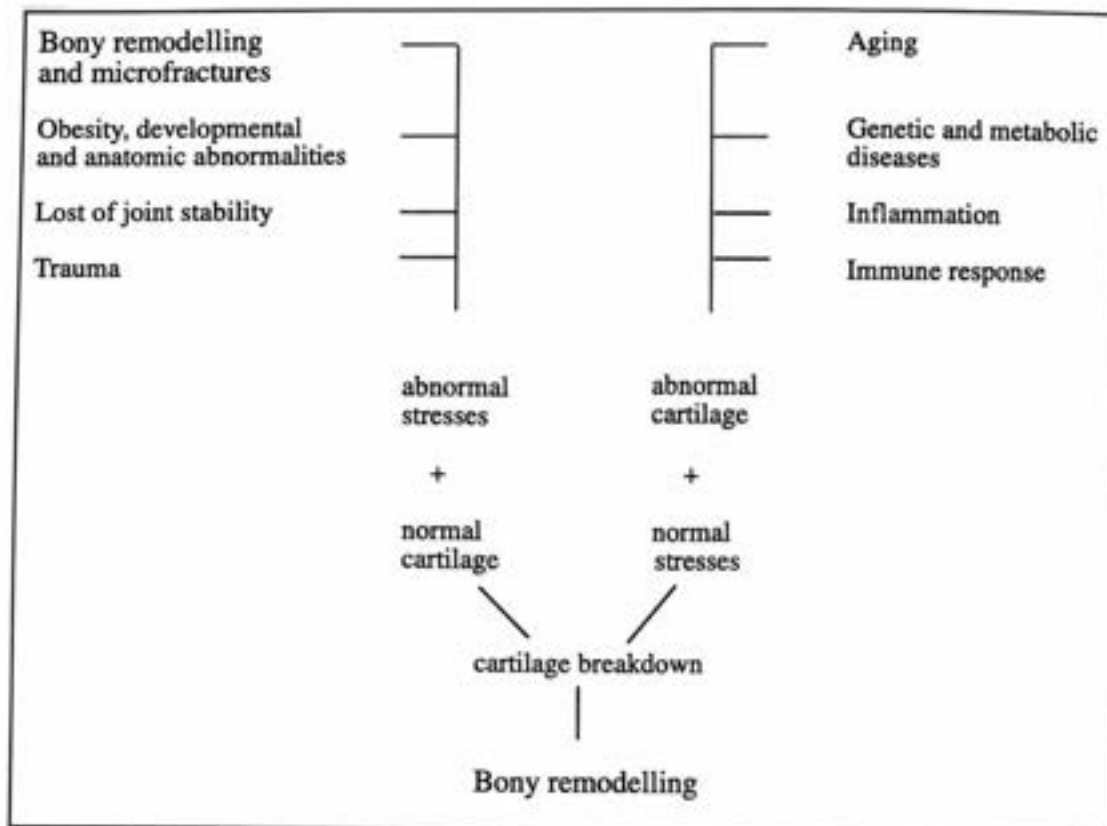


FIG. 71. Aetiology of osteoarthritis (after Howell 1989:1596)

implications of variations in patterns. Ortner (1968) in a detailed study of the elbow in two skeletal groups, offers a model for approaching the analysis of joint degeneration in relation to biomechanical stress.

Gross evidence of bony remodelling in synovial joints, including the articular facets of the vertebrae, is in three principal forms:

- Formation of osteophytes or 'lipping' at the margins of the articular cartilage.
- Production of sclerotic subchondral bone on articular surfaces through osteoblastic activity on joint surfaces in response to cartilage destruction. Mechanical attrition and eburnation of these surfaces occurs in severe cases.
- Rarefaction of the articular surface, particularly of the femoral head and acetabulum, as a result of the formation of subchondral 'pseudocysts' in response to the intrusion of synovial fluid through the articular cortex under pressure. Pitting of the surface may also occur in the absence of cysts.

The pattern of bone alteration varies from joint to joint, because of differing joint construction and degrees of weight-bearing (Steinbock 1976, Ortner and Putschar 1981).

Gross evidence of osteophytosis of the vertebral bodies (also known as spondylitis deformans) is in the form of exostoses, or lipping, arising from the junction of the body and the longitudinal ligaments of the annulus fibrosis on the superior and inferior margins of the vertebral bodies (Rothman and Simeone 1975). They form in response to the raising of the periosteum from the vertebral margins by the protrusion of the annulus fibrosus following narrowing of the disc tissue. This can be caused by vertical prolapse or desiccation. These may appear on any section of the spine, often on adjacent vertebrae, and in their most extreme form can fuse vertebrae together (Steinbock 1976, Ortner and Putschar 1981). Spondylitis deformans is very common in skeletal remains but as the osteophytes themselves are usually clinically asymptomatic the effect of the condition was not necessarily as marked as might be expected from the appearance of the vertebrae. The clinical symptom which can be associated with the osteophytes is radiating pain resulting from protrusion of the bone into the intervertebral foramina and compression of the spinal nerve root. The same condition can arise through the protrusion or extrusion of the disc tissue into the foramina (Jaffe 1972).

Recording of degenerative joint changes

All joints were examined for the presence of the principal bone reactions (lipping, articular surface porosity, and eburnation) associated with joint degeneration. This included osteophytes on vertebral bodies. The observations were recorded with the following grades:-

0. No changes observed.
1. Minimal lipping (osteophyte formation), without porosity.
2. Moderate lipping and/ or porosity.
3. Severe lipping, eburnation and/or porosity.
4. Fusion of osteophytes/joint fusion and immobilization.

Figures 72–73 illustrate examples of joint surfaces with various grades of degeneration. The initial recording was based on individual articular surfaces. The data were then combined as joints for the assessment of the prevalence and degree of degeneration. To avoid misdiagnosing those cases where articular surfaces were missing or damaged, each joint was included in the analysis only where there were data available from surfaces on at least two bones, or where there was at least one surface with bony changes graded '2' or more, and therefore unequivocally pathological. The articular surfaces which were included in each joint were as follows:

Shoulder - Acromio-clavicular, glenoid fossa, humeral head.

Elbow - Distal humerus (capitulum, trochlea), radial head, ulnar trochlear notch.

Wrist - Distal radius, distal ulna, carpals.

Hand - Metacarpals, phalanges.

Hip - Acetabulum, femoral head.

Knee - Distal femur, patella, proximal tibia.

Ankle - Distal tibia, distal fibula, talus, calcaneum.

Foot - Tarsals (other than talus and calcaneum), metatarsals, phalanges.

Vertebrae -

Synovial (apophyseal) - Inferior articular process with superior articular process of adjacent vertebra.

Fibrocartilaginous - Inferior rim of body with superior rim of adjacent body.

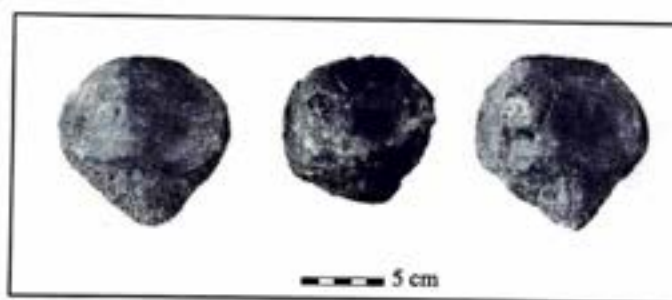


FIG. 72. Articular surfaces of patellae with degeneration of (left) grade 1 (minor lipping), (centre) grade 2 (moderate lipping, minor porosity) (right) grade 3 (minor lipping, severe porosity)

The analysis considers the data in two categories, the first of which includes all grades ('re-modelled') and the second only those joints with changes of grades two and three, which are less equivocally pathological. The data have been classified in this way with the aim of identifying the patterns of joint use both within and among individuals in addition to identifying those individuals with joints which were potentially painful or disabling. In the vertebrae, degeneration of the discs may also result in herniation of the disc and pressure erosion of the articular surface of the vertebral body. This results in the formation of porous depressions (Schmorl's nodes) in the articular surface (Ortner and Putschar 1981). These were recorded where they could be clearly differentiated from postmortem damage to the surface. Figure 74 illustrates examples of Schmorl's nodes on thoracic vertebrae.

In addition to evidence of joint degeneration, variation in the vertical height of the vertebrae was found to be sufficiently common in the population to merit systematic recording. This most often



FIG. 73. Thoracic vertebrae with osteophytosis of the articular surfaces. Top vertebra - superior grade 2, inferior grade 3; second vertebra - superior and inferior grade 1; third and fourth vertebrae - ungraded

took the form of the compression of the anterior vertebral bodies. The effect is primarily anterior because the posterior is supported by the neural arch, the synovial joints and strong musculature. This is noted by Parfitt and Duncan (1975) as the most common vertebral deformity. It can ultimately result in anterior collapse of the vertebral body. The detailed observations were restricted to the lower thoracic (T7-12) and lumbar regions of the spine, as no evidence was found in the upper thoracic spine. The degree of anterior compression was classified subjectively from gross observation on the following scale (Fig. 75):

1. Minimal.
2. Moderate.
3. Severe, with or without gross evidence of fracture of the vertebral body.

Joint degeneration - axial skeleton

As the principal joints of the vertebral column, the apophyseal joints of the vertebral arches and the fibrocartilaginous joints between the vertebral bodies are considered first. The analysis includes at least one joint from all adults except three of the youngest males. The details for each individual are not listed because of the amount of data involved. The data are summarised for each sex by age at death in table 78 and by region of the spine in table 79.

TABLE 78: *Evidence of degeneration of the apophyseal joints (articular processes) and fibrocartilaginous joints (vertebral bodies) of the vertebrae of adults*

Articular processes	<30 years		30+ years		Total	
	Individuals	Joints	Individuals	Joints	Individuals	Joints
All adults						
Observed	28	743	37	999	65	1742
Remodelled	16	64	32	214	48	278
Pathological	5	15	21	96	26	111
Estimated prevalences (%)*						
Pathological	2(1-6)		10(6-14)			
Females						
Observed	15	356	21	580	36	936
Remodelled	7	19	19	101	26	120
Pathological	1	2	13	45	14	47
Estimated prevalences (%)*						
Remodelled	6(3-11)		17(11-24)			
Males						
Observed	13	387	16	419	29	806
Remodelled	9	45	13	113	22	158
Pathological	4	13	8	51	12	64
Estimated prevalences (%)*						
Remodelled	11(7-18)		28(20-37)			
Vertebral bodies						
	<30 years		30+ years		Total	
	Individuals	Joints	Individuals	Joints	Individuals	Joints
All adults						
Observed	28	530	37	643	65	1173
Remodelled	18	92	36	238	54	330
Pathological	9	23	24	113	33	136
Estimated prevalences (%)*						
Pathological	5(2-10)		20(15-27)			
Females						
Observed	15	293	21	360	36	653
Remodelled	8	25	21	128	29	153
Pathological	2	4	14	59	16	63
Estimated prevalences (%)*						
Remodelled	9(4-18)		36(16-61)			
Males						
Observed	13	237	16	283	29	520
Remodelled	10	67	15	110	25	177
Pathological	7	19	10	54	17	73
Estimated prevalences (%)*						
Remodelled	28(18-41)		39(28-51)			

*Estimated using a logistic regression model. Figures in brackets represent 95 per cent confidence intervals, adjusted for over-dispersion in the model

'Remodelled' data include all grades of degeneration. 'Pathological' data include degeneration of grade two or higher

TABLE 79: *Degeneration of the articular processes and vertebral bodies of adults by region of the spine, based on joints with all grades of degeneration*

	Cervical			Thoracic						Lumbar		
				upper			lower					
Articular processes	L	R	All	L	R	All	L	R	All	L	R	All
All adults												
Observed	293	289	582	210	197	407	212	212	424	167	162	329
Affected	58	46	104	22	35	57	30	31	61	29	27	56
Females												
Observed	165	156	321	115	112	227	113	105	218	88	82	170
Affected	21	21	42	10	17	27	14	9	23	14	14	28
Estimated prevalences (%)*												
under 30 years	6(2-15)			3(1- 15)			9(3-22)			8(2-23)		
30+ years	21(12-33)			17(9-30)			12(6-25)			17(8-32)		
Males												
Observed	128	133	261	95	85	180	99	107	206	79	80	159
Affected	37	25	62	12	18	30	16	22	38	15	13	28
Estimated prevalences (%)*												
under 30 years	10(4-24)			5(1-24)			15(6-33)			13(4-35)		
30+ years	33(2-93)			27(1-91)			21(1-88)			27(1-91)		
Vertebral bodies	Cervical			Thoracic						Lumbar		
All adults				upper			lower					
Observed	306			281			310			276		
Affected	58			46			106			120		
Females												
Observed	181			152			167			153		
Affected	28			16			43			66		
Estimated prevalences (%)*												
under 30 years	7(2-18)			7(2-18)			8(3-19)			13(6-27)		
30+ years	21(13-32)			14(7-27)			42(30-55)			66(53-77)		
Males												
Observed	125			129			143			123		
Affected	30			30			63			54		
Estimated prevalences (%)*												
under 30 years	19(9-37)			18(8-36)			37(22-56)			37(21- 57)		
30+ years	28(14-47)			27(14-47)			50(33-67)			50(32- 68)		
*Estimated prevalence based on the prevalence of the disease in the population.												

*Estimated using a logistic regression model. Figures in brackets represent 95% confidence intervals, adjusted for over-dispersion in the model

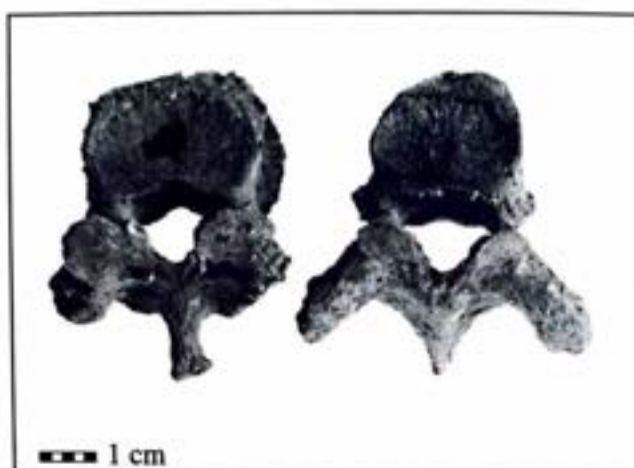


FIG. 74. Thoracic vertebrae with depressions on the centre of the endplates of the bodies (Schmorl's nodes) resulting from pressure erosion following herniation of the disc. The vertebra on the left also has some post-mortem damage of the edges of the depression

The prevalence and degree of degeneration was greater on the vertebral bodies than the articular processes in both sexes at all regions of the spine. As joint degeneration was used in the age estimations of some older individuals, the relationship with age cannot be used as an indicator of the absolute rate of degeneration. Comparison between the sexes is valid, however, and as there were statistically significant effects for age the data are reported for two age groups. Males developed degeneration in both types of joints earlier than females. On the articular processes, this sex difference was also evident in older individuals. On the vertebral bodies, degeneration progressed relatively faster with increasing age in females than in males so that sex differences were not significant in older individuals. Nevertheless, the proportion of remodelled joints classified as pathological (grade 2 or greater) was similar in both sexes at all ages in both types of joints.

Degeneration of the articular processes was more prevalent in the lower spine than the upper in young individuals. The pattern changed in older individuals, with the cervical, upper thoracic and lumbar vertebrae affected to a similar degree. Despite the greater prevalence of degeneration of the articular processes in all regions of the spine in males of all ages than in females, the regions of the spine affected were similar in both sexes at all ages. There were no significant differences between the left and right sides in either sex in any region. Degeneration of the vertebral bodies was more prevalent in males than in females at all ages in all regions, with the exception of the lumbar region in older individuals where degeneration was more common in females. In young females, degeneration was more common in the lumbar region than elsewhere in the spine. In older females both the lumbar and lower thoracic regions were affected more than the upper spine. In males of all ages the lower thoracic vertebrae and lumbar spine were more commonly affected than the upper spine.



FIG. 75. Lumbar vertebrae with anterior compression of the body of grade 3 (top vertebra), grade 2 (second vertebra), grade 1 (third vertebra). The lowest vertebra is ungraded. These vertebrae are from different individuals

Osteoarthritic changes on the costo-vertebral joints, both on the vertebral bodies and the transverse processes, were also recorded. The degeneration included both porosity of the joint surface and lipping of the margins (Table 80).

TABLE 80: *Prevalence of degenerative remodelling of the costo-vertebral joints of the vertebral bodies and transverse processes of adults by sex and age at death*

	n	Upper Thoracic		Lower Thoracic	
		Right	Left	Right	Left
Females					
<30 years	2	1	0	2	2
30+ years	9	5	5	6	7
All	11	6	5	8	9
Males					
<30 years	6	1	1	6	6
30+ years	8	5	5	6	7
All	14	6	6	12	13

The degeneration of the costal joints started earlier in males than in females, particularly in the lower thoracic region, but beyond this the patterns were similar in both sexes. There was more degeneration in the lower thoracic than the upper, but no differences between the sides. The joints most commonly involved were those of the first and last thoracic vertebrae, and the recorded prevalence in the upper thoracic relates almost exclusively to the first costo-vertebral joint. In the lower thoracic the degeneration is more widespread and involves all joints in some cases. In one older male (B115), two costal cartilages had ossified and fused to the eighth and ninth thoracic vertebrae.

In addition to the development of osteophytoses on the vertebral bodies, porosity of the superior and inferior surfaces was observed in many individuals in the cervical vertebrae. The porosity was not necessarily associated with phytosis. It appeared on the antero-superior surface of the bone in younger individuals, but in older individuals the entire superior and inferior surfaces of the bodies became involved. Table 81 summarises the prevalence.

TABLE 81: *Prevalence of porosity in the cervical vertebrae of adults by sex and age at death*

Age	Females		Males	
	n*	%*	n	%
<30 years	2	13	5	36
30+ years	11	52	10	62
All	13	36	15	50

*Number and proportion of individuals with at least one cervical vertebral body with porosity

Both sexes were affected, with the porosity appearing earlier in males than in females. Some degeneration was evident in all vertebrae from C3 to C7, although the joints most commonly affected were those on the fourth and fifth cervical vertebrae. The body of the seventh vertebra was affected in only one female.

Schmorl's nodes were recorded on nine individuals, three females and six males. The depressions ranged in size from a few millimetres to about one centimetre in diameter, and varied in shape from circular to longitudinal. All individuals were aged 30 years and over at death except one of the males. This individual also had the most vertebrae involved, with lesions appearing in the bodies of all between T6 and L2. In two males, the bodies of L2 and L3 were affected, but in the remainder of the cases the lesions were on the lower thoracic vertebrae and involved a single joint, with two adjacent vertebrae affected.

Eleven adults (nine males, two females) with patterns of spinal degeneration which are at variance with that observed in the majority of individuals or are more extreme in degree are listed in table 82.

To summarise, spinal degeneration was more prevalent in younger males than younger females. Beyond the age of 30 years the degree and prevalence became similar in both sexes except degeneration of the articular processes which were affected more in males than females. Comparing the

degeneration by region, the lower spine was more affected in individuals of both sexes at all ages than the upper spine, although this regional difference was significantly greater with increasing age in the vertebral bodies in females than in males. Degeneration was relatively greater in the upper spine in the older males than in the older females.

Degenerative change of the temporomandibular joint is common (Table 83). Both males and females were affected by lipping of the articular surface, but porosity of the articular surface was evident only in females 35 years and over at death. A greater proportion of individuals are affected in the older females than the younger age group, but neither the age difference nor the sex difference is statistically significant at the 5 per cent level. Changes on the posterior surface of the condyle were found in 14/44 (31 per cent) adults. These changes were in the form of varying degrees of porosity and depression, either along the margin of the articular surface or inferior to the articular surface. There were no statistically significant differences in prevalence by age or sex. Two examples are illustrated in figure 76. This appears to represent remodelling in response to the forcing of the condyle against the tympanic plate of the temporal bone during mastication. These cases have been excluded from the analysis as the remodelling is external to the articular surface and does not represent arthritis. Three of these individuals also had degenerative changes of the articular surface.

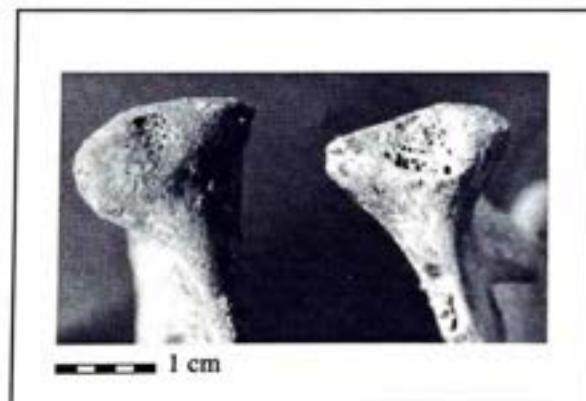


FIG. 76. Two examples of degeneration of the mandibular condyle in the form of porosity of the posterior surface of the condyle

Pathology on the post-cranial axial skeleton other than the vertebrae principally involves lipping and porosity of the area of attachment of the first costal cartilages to the manubrium, and porosity of the clavicular articular facets. This was observed on nine individuals, all males. All were affected bilaterally, but the lipping was predominant on the right in four individuals and on the left in three.

Joint degeneration - appendicular skeleton

Observations were made of the condition of at least one appendicular joint of 65 of the 68 individuals aged 15 years and over at death. The prevalence of degenerative joint changes in the limbs is summarised in tables 84 and 85.

The method of analysis was the same as that used for the vertebral joints, and there is the same restriction on the validity of the comparison of the degree of degeneration with age. There were no significant sex differences in the rate or degree of degeneration, either overall or in any joint. The joints most commonly affected were the shoulders, hands, knees, ankles and feet. The hips were least affected. This pattern was established in young individuals and continued with increasing age, although the differential rate of degeneration among joints decreased to some extent in older individuals. There were no significant differences in degeneration between left and right sides in any joint.

Individuals with multiple joint involvement are listed in table 86. All these individuals had degeneration in all or almost all major limb joints. Those joints with degeneration of grade two or more are listed in the table.

Joint degeneration - discussion

Comparison of degeneration by sex shows that the prevalence of joint degeneration was higher in males than females at an early age in the spine. Males were also affected at an earlier age than females in other forms of degeneration in the spine. Sex differences in the rate or degree of remodelling in older individuals were less obvious, although males were still generally more affected than females. Females had relatively more degeneration in the lower vertebrae than the upper with increasing age, where the pattern of spinal degeneration in males was more uniform with the upper vertebrae involved as well as the lower. In the appendicular joints, there were no statistically significant sex differences in either age group. The shoulders, hands, knees and feet were most affected and the hips least in both age groups. There were no significant differences in prevalence between left and right sides in either the spine or limbs.

The prevalence of joints with degeneration of grade two or more, classed as pathological, did not differ significantly between the sexes in either spine or limbs. Almost two-thirds of adults over thirty (24/37) had at least one spinal joint affected and almost 90 per cent (31/36) had at least one major limb joint affected. Seven individuals (one female, six males) had multiple joints with moderate-severe degeneration in the spine and 10 individuals (three females, seven males) in the major limb joints. Three males (burials 93, 115 and 132) had both spine and limb joints affected.

There are no published comparative data for appendicular joint degeneration from other prehistoric Thai sites. As a broad indication of the rate and frequency of degeneration at Khok Phanom Di relative to other populations, the data are compared with samples of modern American Whites and Blacks, protohistoric Inuit, and prehistoric American Indians from Pecos Pueblo, New Mexico reported by Jurmain (1977). These samples totalled almost 800 individuals but when distributed by ethnic group, sex and age at death each subsample was small. Nevertheless, the study provides standardised data for a selection of geographically separated groups with different genetic backgrounds and/or living in different environmental conditions. The data show that Inuit had the earliest onset

and most severe involvement of all joints, that Blacks were more frequently affected than Whites in all joints except the hip, and that degeneration in Pueblo Indians was uniformly low. The data given by Jurmain are for moderate-severe degeneration and are compared with the data for degeneration of grade two or more at Khok Phanom Di.

In females, the onset and progression of joint degeneration up to the age of 40 at Khok Phanom Di was generally slower than other groups except Pueblo Indians. This was particularly evident in the hips, which were almost unaffected in the females at Khok Phanom Di. After the age of 40, the comparative rates of degeneration between Khok Phanom Di and the other populations reversed. Progression was relatively rapid at Khok Phanom Di so that frequencies reached equivalent levels to those in Inuit and Blacks. In the males, joint degeneration at Khok Phanom Di followed a pattern of onset and frequency similar to that of the Inuit. Exceptions were at the elbow, where onset was slower but frequencies similar after the age of thirty, and the knee where frequencies were higher at an earlier age at Khok Phanom Di.

A brief comparison of frequencies of joint degeneration at Khok Phanom Di with the Inuit data from Merbs (1983) shows that degeneration in all joints at Khok Phanom Di was greater than in the Inuit except in the elbows of both sexes and the hips of males. Merbs does not provide age-related data.

Degeneration in the major limb joints of adults at Khok Phanom Di appears to have been relatively frequent and severe in comparison with other geographically and chronologically widely-spaced groups (Jurmain 1977, Merbs 1983). In females and older males, spinal degeneration progressed at a similar rate to other populations, with half of individuals affected by the thirties and 100 per cent beyond the age of 40. Both these statements are qualified by the uncertainty of the demographic structure of the comparative samples, by interobserver variation in assessing degeneration and by the variation introduced by differential preservation.

TABLE 82: *Individuals with multiple or unusual patterns of vertebral degeneration*

B18, Female, aged 42y. Cervical spine - has extreme phytosis and arthritis, particularly on the left. Thoracic spine - there is fracture and collapse of bodies of T10 and T12, with Schmorl's nodes and osteophytes. T11 has retained its shape. There is degeneration of the costal facets. Lumbar spine - T12 and L1 have fused, L1 has Schmorl's nodes and osteophytosis and has compressed to form a 'codfish' shape (Ortner and Putschar 1981)

B28, Male, aged 21y Lumbar spine - the vertebral bodies have collapsed and are very porotic, with extreme phytosis. L5 has spondylolysis. There is no degeneration in other regions of the spine

B42, Male, 32y. Cervical and upper thoracic spine - has slight arthritis, and porosity of the cervical bodies. Lower thoracic - T11 has collapsed towards the R, with associated phytosis. The L costal facet of T12 is raised and enlarged. Lumbar - L5 has porosity on posterior superior body, and has almost fused to the sacrum

B87, Female, 24y. Lumbar - there is extreme phytosis on L4 and anterior porosity of L5. L3 and L4 have spondylolysis

B92, Male, 18y. Lower thoracic - the bodies are anteriorly compressed, with enlarged vascular foramina on the right anterior. L4 and L5 have extreme porosity on the anterior superior surface

B93, Male, 42y. There is moderate-severe degeneration recorded on all regions of the spine, including the costal articular facets, and Schmorl's Nodes on upper thoracic and lumbar vertebrae. There is particularly severe phytosis and porosity on L3-L5, and L5 is compressed posteriorly. The spinous process of L4 appears to have been articulating with L5. L5 has spondylolysis

B103, Male, 26y. Lower thoracic spine - there are asymmetrical articular facets and laminae T11, T12 and L1. The spine was bent to the right in the ground. Lumbar spine - L5 was probably partially fused to S1

B115, Male, 38y. There is evidence of severe degeneration on the entire spine, including phytosis, arthritis of articular processes and costal articular facets, and porosity and compression of vertebral bodies. This is particularly evident on the right. A number of the costal cartilages have ossified, and this has resulted in the ribs fusing to the vertebral bodies of T8 and 9 on the right

B129, Male, 27y. Cervical and upper thoracic - there is slight-moderate arthritis, phytosis, and porosity of the cervical bodies. Lower thoracic - there is moderate arthritis, phytosis, porosity and Schmorl's Nodes. The bodies of T8 and 9 are fused on the right. Lumbar - there is severe arthritis and moderate phytosis and porosity of bodies, particularly L3

B131, Male, 25y. Cervical and upper thoracic - have slight-moderate arthritis, phytosis and porosity of the cervical bodies. Lower thoracic - there is extreme arthritis of articular processes and costal articular facets and extreme phytosis on T11 and T12, with virtual fusion of bodies on the right. Lumbar - there is slight-moderate phytosis, arthritis and porosity of bodies

B132, Male, 34y. There is moderate-severe degeneration recorded on all regions of the spine. There is asymmetrical spondylolysis of L5, with separation at the pars interarticularis on the left and at the pedicle on the right

TABLE 83: *Prevalence of degenerative changes of the temporomandibular joint by sex and age at death*

	All adults			Females			Males		
	N	n ¹	% ²	N	n ¹	% ²	N	n ¹	% ²
<30 years	18	3	17	8	1	13	10	2	20
30+ years	29	11	38	18	9	50	11	2	18
Total	47	14	30	26	10	38	21	4	19

¹The number of individuals with degeneration of the TMJ on at least one side,²Proportion of individuals with degeneration of the TMJ on at least one sideTABLE 84: *Evidence of remodelling and pathological degeneration of all major joints of the appendicular skeleton in adults of both sexes*

	<30 years		30+ years		Total	
	Individuals	Joints	Individuals	Joints	Individuals	Joints
All adults						
Observed	29	323	36	385	65	708
Remodelled	18	61	35	234	53	295
Pathological	8	16	31	92	39	108
Females						
Observed	15	170	21	216	36	386
Remodelled	8	22	21	129	29	151
Pathological	2	5	20	51	22	56
Males						
Observed	14	153	15	169	29	322
Remodelled	10	39	14	105	24	144
Pathological	6	11	11	41	17	52
Estimated prevalences (%)*						
Both sexes						
Remodelled	19(12-29)		60(50-70)			
Pathological	6(2-14)		35(26-44)			

*Estimated using a logistic regression model. Figures in brackets represent 95 per cent confidence intervals, adjusted for over-dispersion in the model

TABLE 85: *Evidence of degeneration of the individual major appendicular joints of adults of both sexes. Data represent all grades of degeneration for left (L) and right (R) sides. The number of individuals affected is also included (Indiv.)*

	Shoulder			Elbow			Wrist			Hand
	L	R	Indiv.	L	R	Indiv.	L	R	Indiv.	Indiv.
Upper limb										
All adults										
Observed	51	53	56	56	61	63	40	46	54	53
Affected	26	30	32	17	24	27	13	16	21	23
Females										
Observed	29	28	31	30	33	34	21	26	30	27
Affected	12	14	15	8	14	15	4	8	11	11
Males										
Observed	22	25	25	26	28	29	19	20	24	26
Affected	14	16	17	9	10	12	9	8	10	12
Estimated prevalences (%)*										
<30 years	28(19-38)			13(8-19)			12(7-19)			24(14-38)
30+ years	75(64-83)			53(45-60)			50(42-58)			71(60-80)
Lower limb										
	Hip			Knee			Ankle			Foot
	L	R	Indiv.	L	R	Indiv.	L	R	Indiv.	Indiv.
All adults										
Observed	38	44	51	53	54	57	51	56	57	52
Affected	7	10	12	26	28	32	22	23	26	30
Females										
Observed	18	23	26	31	30	32	29	32	33	29
Affected	4	6	7	17	16	18	11	12	13	14
Males										
Observed	20	21	25	22	24	25	22	24	24	23
Affected	3	4	5	9	12	14	11	11	13	16
Estimated prevalences (%)*										
<30 years	6(4-12)			25(17-35)			19(12-27)			34(21-49)
30+ years	34(27-42)			72(61-80)			63(54-72)			80(67-88)

*Estimated using a logistic regression model. Figures in brackets represent 95 per cent confidence intervals, adjusted for over-dispersion in the model

TABLE 86: *Individuals with polyarticular arthritis of the limbs*

Burial	Sex	Age	Joints with pathological degeneration
18	Female	42	All margins of the distal articular surface of the right femur have extreme lipping, and the articular surface has raised, porous areas. On the right tibia, the medial plateau is extremely porous on the posterior edge, and the margins of the articular surfaces are lipped. The patella is not present. The pattern of remodelling is that of severe osteoarthritis, but far exceeds the degree of joint degeneration apparent on the left knee
27	Female	41	Shoulders, elbows, hands and knees
45	Female	45	Both first metatarsals have reactive bone on the lateral distal surfaces, including cloacae on the left. Degenerative disease of the joints of the left leg is greater than on the right
61	Female	48	Shoulders, hands, knees, ankles and feet. Degeneration extreme in knees and ankles
67	Male	35	Left shoulder, right wrist, right knee, ankles and feet
72	Male	32	Right shoulder, left wrist and knees
74	Male	39	Shoulders and left knee
79	Female	47	Hips and knees
91	Male	45	Right shoulder, hands, left knee and feet. Half of joints not able to be scored, including left shoulder and right knee. Degeneration extreme in hands
93	Male	42	Wrists, hands and knees
115	Male	38	Left wrist, hands and left knee
132	Male	34	Right shoulder and left ankle

NON-SKELETAL EVIDENCE FOR DISEASE

Burial 56, a female aged 45 years, was found with a compacted mass of organic material in the pelvic basin. This contained rice fragments, fish bone and scales, (Greenwood 1991). The mass probably represents bowel or stomach contents. This was unique at Khok Phanom Di which suggests that the material was altered in some way from normal gut contents. It may have survived because it was compacted. This individual had suffered from multiple episodes of growth disruption and was the shortest female in the group. She has some characteristics which suggests that she may have been anaemic. The coincidence of this pattern of health and the food residue suggests that there was some relationship, but as she had been fertile and lived to the age of 45 years it is difficult to identify a chronic condition which would account for all the evidence. The mass may have been the result of an intestinal obstruction such as carcinoma, which would have been the cause of death.

TABLE 87: *Adult cranial vault thickness measurements from lateral cephalographs (mm) adjusted for radiographic enlargement. Vertex = the highest point of the calvarium, measured perpendicular to a line drawn between porion and orbitale. Frontal = midway between bregma and nasion*

Burial	Frontal	Bregma	Vertex	Burial	Frontal	Bregma	Vertex	Burial	Frontal	Bregma	Vertex
Females											
4	8.78	5.40	7.87	13	7.23	6.77	6.04	18	9.24	7.32	7.41
35	7.05	6.04	6.41	45	5.58	5.86	6.95	47	7.69	5.95	5.67
58	4.94	5.49	7.05	61	8.14	6.95	8.60	64	6.41	5.58	5.76
73	7.32	6.86	8.42	77	12.26	11.16	10.16	79	8.60	9.15	9.97
83	7.78	6.41	6.31	87	8.69	8.69	7.69	94	5.95	7.32	6.86
102	6.68	6.86	7.50	107	7.23	6.68	7.14	109	8.60	5.95	8.69
Males											
24	6.41	7.50	7.96	28	8.05	7.78	8.23	29	5.40	6.95	8.33
30	6.13	5.58	7.59	38	7.50	10.43	11.35	42	8.60	12.08	10.89
57	8.33	9.15	9.15	67	8.33	10.06	9.24	72	7.59	9.15	9.24
74	7.05	7.05	7.32	93	8.60	9.61	9.70				

TABLE 88: *Adult cranial vault thickness and ratio of diploic to cortical bone measured on the parietal bones of individuals with damaged crania. The cortical bone includes both inner and outer tables of the vault (mm)*

Burial	VT	R C/D	Burial	VT	R C/D	Burial	VT	R C/D
Females								
15	6.50	1:1.50	19	9.70	1:1.66	27	7.00	1:2.04
31	9.45	1:1.66	40	9.10	1:2.50	56	11.70	1:3.59
60	7.00	1:1.19	113	7.60	1:1.81	122	8.20	1:1.56
154	7.60	1:2.45						
Males								
92	6.60	1:1.64	100	6.30	1:1.86	129	8.00	1:1.35
131	7.30	1:1.09	147	9.50	1:2.17			

VT: Vault thickness, R C/D: Ratio cortical bone/diploe

TABLE 89: *Cranial vault thickness, thickness of diploe and cortex (inner and outer tables combined) and ratio of diploe to cortical bone (measured at mid-frontal) in children. Measurements in mm, from lateral cephalographs, adjusted for radiographic enlargement*

Burial	Age	Vault thickness			Diploe	Cortical bone Midfrontal	Ratio cortical bone/diploe
		Frontal	Bregma	Vertex			
1	9	4.2	-	-	2.3	1.9	1:1.21
37	9	5.2	4.4	5.2	2.1	3.1	1:0.68
11	12	6.9	5.9	8.4	3.2	3.7	1:0.86
17	12	4.4	5.1	5.2	1.8	2.5	1:0.72

TABLE 90: *Cranial vault thickness on the midfrontal and parietal bones of children with damaged crania. The cortical bone includes both inner and outer tables of the vault. Measurements are in mm*

Burial	Age	MF	P	Burial	Age	MF	P	Burial	Age	MF	P
6	9	-	4.5	7	1.75	c. 3.0	3.4	12.1	12	-	4.5
20	12	-	5.4	21	8	3.2	4.4	32	11	-	c. 2.7
88	0.75	c. 3.0	2.0	96	4	c. 3.0	c. 1.5	121	1.25	c. 3.0	2.3
101	1.50	c. 3.0	2.0	137	5	-	4.9*	145	8	c. 3.5	7.2

MF: Midfrontal, P: Parietal*; bregma

TABLE 91: *Facial profile angles measured from lateral cephalographs of children. SNA = angle sella-nasion-subspinale. The other angles are illustrated in figure 51*

Burial	Age	Nasal bone	Naso-maxillary	Subnasal	Alveolar
21	8	-	-	-	120
37	9	87	-	-	113
11	12	-	-	-	-
17	12	-	162	133	103

TABLE 92: *Bi-jugal and bi-infraorbital widths used in the assessment of lateral hypertrophy of the maxilla and zygomatic bones in children. (mm) Bi-jugal - Martin's 43(1) (Martin and Saller, 1957) Bi-infraorbital - width between the infraorbital foramina*

Burial	Age	Bi-jugal width	Bi-infraorbital width	Ratio
21	8	98.0	46.1	1:2.13
37	9	111.9	53.3	1:2.10
1	9	97.9	44.0	1:2.23
11	12	107.8	50.5	1:2.13
17	12	109.5	47.9	1:2.29

TABLE 93: *Facial profile angles of adults measured from lateral radiographs. SNA = angle sella-nasion- subspinale*

Burial	SNA(°)	Burial	SNA(°)	Burial	SNA(°)	Burial	SNA(°)
Females							
4	84	18	77	45	86	58	84
61	94	64	88	73	87	77	83
79	82	83	87	87	82	109	80
110	79						
Males							
24	90	30	93	38	86	42	87
44	82	67	84	72	85	74	78
93	82						

TABLE 94: *Bi-jugal and bi-infraorbital widths used in the assessment of lateral hypertrophy of the maxilla and zygomatic bones. (mm) Bi-jugal = Martin's measurement 43(1) (Martin and Saller 1957) Bi-infraorbital = width between the infraorbital foramina*

Burial	Bi-J	Bi-I	Ratio	Burial	Bi-J	Bi-I	Ratio	Burial	Bi-J	Bi-I	Ratio
Females											
4	115.80	51.00	1:2.27	18	115.10	55.10	1:2.08	35	121.10	55.70	1:2.17
45	115.00	52.50	1:2.19	47	113.50	54.85	1:2.06	56	119.50	57.40	1:2.08
58	122.70	58.50	1:2.09	61	118.20	57.20	1:2.06	64	117.20	57.00	1:2.05
73	119.30	58.40	1:2.04	77	125.75	61.75	1:2.03	79	120.40	56.40	1:2.13
83	118.75	55.70	1:2.13	87	125.30	59.10	1:2.12	94	116.40	54.90	1:2.12
109	121.70	55.90	1:2.17	110	114.40	53.50	1:2.13	142	120.00	56.60	1:2.12
Males											
24	122.65	54.65	1:2.24	28	112.65	53.20	1:2.11	30	129.60	57.30	1:2.26
38	131.90	52.80	1:2.49	42	124.95	60.80	1:2.05	44	116.00	56.00	1:2.07
67	128.70	61.70	1:2.08	72	129.35	59.20	1:2.18	74	128.00	63.00	1:2.03
92	109.80	51.50	1:2.13	93	126.40	59.00	1:2.14				

Bi-J: Bi-jugal, Bi-I: Bi-infraorbital

VI. DENTAL PATHOLOGY

PATHOLOGY of the dentition tends to receive more attention than the rest of the individual in skeletal analyses because the teeth are usually relatively well preserved, each tooth forms a unit on which observations can be made, and there is likely to be less ambiguity about the nature of lesions. The dentition has often been reported separately from, or to the exclusion of, the rest of the skeleton, and particularly in older reports has tended to be the basis for most of the observations reported. It is recorded separately here primarily because of the different physical and biochemical structure of teeth and bones, although the synergistic interaction of pathology of the dentition and those of the rest of the individual, and the possibility of dental response to systemic problems (or vice versa) are taken into consideration in the interpretation of observations.

The conditions recorded as dental pathology will include not only those specifically affecting the teeth, but also those of the supporting alveolar bone, as the structures are intimately connected both physically and physiologically. The overall condition of the dentition will generally reflect the association between pathologies in both the teeth and the alveolar bone, and studies using only one or two indicators cannot provide a complete picture of the condition of the dental complex (Hall *et al.* 1986). Dental pathology is nevertheless unique in that it reflects not only health but also, when considered together with patterns of non-pathological wear on the teeth, directly reflects the composition of the diet. The focus in this chapter will be on health, and diet is discussed in chapter VII.

As has already been noted, because of the highly mineralized composition of teeth (enamel is 95 per cent inorganic material (Einspahr and Bugg 1980)), dental reactions to environmental or biomechanical stress are limited to destruction of the tooth. Apart from limited remineralization of demineralized areas of enamel in the right environment, such as in the presence of fluoride, the dentition does not have the ability to remodel itself in response to damage. The possible sources of damage of the tooth structure are caries, wear (both through chewing food and using teeth as tools), and traumatic damage. Wear of a tooth does not necessarily represent pathology. Only in those cases where the destruction of the crown is excessive and the pulp chamber is exposed, providing a channel for the entry of infection into the supporting tissues, is there a condition created which constitutes unequivocal pathology. Defects formed on a tooth during the period of development (enamel hypoplasia) have already been discussed in the context of evidence of growth disruption as this reflects a secondary response to systemic environmental stress rather than a local reaction to disease of the tooth.

Apart from damage to the structure, the only other possible response of the teeth to environmental or mechanical stress is the loss of the tooth. This can result from pathologies which have affected the supporting tissues, including the alveolar bone, or as a consequence of continued eruption of the tooth in maintaining occlusion in the event of extreme wear of the crown. Loss can also be unrelated to the condition of the tooth, as a result of trauma, or cultural practices involving tooth extraction. The bony support of the dentition is subject to the range of conditions affecting all bone,

but the most common remodelling of alveolar bone is in response to inflammation of the periodontal tissues. This results in increased porosity of the alveolar bone, formation of pockets around the tooth roots, and resorption of the alveolar crest. Infection originating from exposure of the dental pulp through wear or caries results in inflammation of the tissues around the apex of the root. This is reflected in the formation of periapical cavities in the bone. In life, these would have been occupied by relatively benign granulomata, dental cysts or, with more virulent infections, abscesses. One other condition affecting teeth which can often be diagnosed from archaeological remains is the presence of calculus. Apart from very unusual cases of the recovery of kidney, gall, or bladder stones or of calcified deposits in the circulatory system, this is the only example of recoverable evidence of bodily response to external conditions other than the evidence of alteration to bone or teeth. Each of these pathological conditions affecting the dentition is initially separately assessed. The overall pattern of dental pathology and the interrelationship among the conditions is drawn together in the discussion at the end of the chapter.

DENTAL CENSUS

For the dental census, recordings were made in the following categories:

- Tooth present and undamaged, either *in situ* or loose.
- Tooth present, damaged but with all or part of the crown remaining. The conditions of preservation were such that the enamel of many of the teeth was brittle, and easily 'flaked' off the underlying dentine.
- Tooth lost post-mortem, socket remaining, with or without root fragments *in situ*.
- Tooth lost ante-mortem, with the socket and the alveolar crest either partially or completely remodelled. This category was divided into two subcategories:
 - tooth lost from pathological causes.
 - tooth deliberately ablated.
- Tooth congenitally absent.

The classification of missing teeth as lost ante-mortem or congenitally absent is not straightforward. The dentition can respond two ways in cases of congenital tooth absence, or anodontia. Either the deciduous dentition is retained, filling the spaces normally occupied by the missing permanent teeth, or the teeth which do exist erupt mesially of their normal position, leaving no space for the missing teeth. The spacing of the teeth was examined for this characteristic. Radiographs were taken where possible in order to identify and eliminate cases of impaction. There were distinct, mainly symmetrical, patterns of missing anterior teeth in individuals of all ages which suggested that the deliberate removal of teeth was practiced.

The identification of the cause of ante-mortem loss where the patterns were not symmetrical or where many teeth had been lost was also not straightforward. Where there was any doubt about

the cause of the loss, the teeth were classified as lost through pathological causes. Details of the methods and results of the identification of deliberately ablated teeth are reported in Tayles (1996) and discussed in chapter VIII.

As has already been noted, dental conditions are one of the more common aspects of skeletal biology included in analyses of human remains, although at the time of recording these data, there were no universal standards for recording and reporting. In particular, many reports concentrate on teeth as the basic unit of analysis without regard to the interrelationships of pathologies (and morphologies) within individual dentitions, and without recognition of the relevance of the observations to the overall health of the individual.

The adult dental census data are summarised by tooth type in table 95. Because of the size of the data base, individual data are not listed for any of the dental pathology in this chapter. Data from 95 per cent of all possible permanent tooth positions (including existing teeth, empty sockets and alveolar bone of teeth lost ante-mortem) were recorded. In 43 of the 67 adults (64 per cent) all 32 tooth positions were accounted for. The mean number of tooth positions recorded for all individuals was 30.6.

The census includes all individuals except B22, who was found without a head. A single individual (B28) had the entire mandible missing. All other adults had some teeth in each dental arcade. Fifty-five per cent of teeth were present and intact and 24 per cent were damaged, lost from their sockets post-mortem, not fully erupted, or congenitally absent. The balance of 16 per cent were lost ante-mortem, either through deliberate ablation or pathological causes. The patterns of ante-mortem loss through pathological causes is reported more fully later in this chapter.

TABLE 95: *Summary of adult dental census*

n=67 individuals	M*3		M1, M2		Premolars		Canines		Incisors		All Teeth	
	n	%	n	%	n	%	n	%	n	%	n	%
Teeth present	132	49	357	67	361	67	162	60	176	33	1188	55
Lost or damaged pm	43	16	105	19	120	22	82	31	140	26	490	23
Lost am, pathological	44	16	55	10	37	8	12	4	12	2	160	8
Lost am, ablation	-	-	-	-	-	-	4	2	173	33	177	8
Unerupted or agenesis	28	11	0	0	2	0	0	0	2	0	32	1
Total Recorded	247	92	517	96	520	97	260	97	503	94	2047	95
No record possible	21	8	19	4	16	3	8	3	33	6	97	5
Total potential	268	100	536	100	536	100	268	100	536	100	2144	100

*M: molar

CARIES

Dental caries is fundamentally a microbial disease which affects the calcified tissues of teeth, beginning first with a localized dissolution of the inorganic structures of a given tooth surface by acids of bacterial origin, and leading to a disintegration of the organic matrix. (Legler and Menaker 1980:212)

Carbohydrates form the basis for plaque formation and bacterial action in the production of organic acids, and diet is, therefore, a primary factor in caries susceptibility. Other factors are of genetic origin, such as saliva composition and flow rate, characteristics of the immune system, the morphology of teeth (pit and fissure patterns), the contacts between teeth, the physical and chemical nature of the surface enamel (Legler and Menaker 1980) and the composition of oral micro-organisms (Morhart and Fitzgerald 1980). There is a demonstrated relationship between the presence of enamel hypoplastic defects and the prevalence of dental caries, particularly in the deciduous dentition (Sweeney *et al.* 1971, Jelliffe and Jelliffe 1971, Infante and Gillespie 1977, Alfano 1980). The presence of trace elements in the diet, particularly fluoride, can affect the surface enamel and inhibit caries formation. Cultural practices involving oral hygiene and the removal of bacterial plaque are also a factor. Despite the importance of carbohydrate consumption in the caries process, the relationship between diet and caries prevalence is not straightforward.

Of all the skeletal responses to pathology, caries is often the least affected by post-mortem damage. The prevalence is, therefore, relatively easily recorded and is widely reported in skeletal series. Despite its complex aetiology, caries are believed to provide evidence of the response in human health to such fundamental changes in subsistence methods as the transition from hunting and gathering to agriculture (Turner 1979).

The disease can progress from a small lesion to ultimate destruction of the crown of the tooth, although alteration of the conditions of the oral environment can arrest the development of caries. The progression of carious destruction from a small initial lesion can be quite rapid, with exposure of the pulp chamber and creation of a route for infection to enter the supporting tissues through the root canal. Ultimately, the crown may be completely destroyed and the tooth lost. The teeth most commonly affected by caries are the posterior teeth with complex crown morphologies, which provide pits and fissures for colonisation by bacteria and are less exposed to cleansing. Caries also develop at the points of contact between teeth, and on surfaces of roots exposed by recession of the gingiva in older individuals, although any tooth surface is susceptible. A particularly distinctive form of caries associated with hypoplastic defects of the enamel of the deciduous teeth is described as 'Cauque lesions' (Alfano 1980), 'bar decay', 'poor man's caries' (Jelliffe and Jelliffe 1971) or 'circular caries' (Cook and Buikstra 1979). The caries form along the lines of defective enamel, particularly the incisors, and can encircle the entire tooth.

All teeth, both deciduous and permanent, were examined for caries. The types of teeth which are carious and the position of caries on the tooth are both relevant in determining the interrelationships between caries, other dental pathologies, and tooth wear, and the possibility of dietary influence on these characteristics. The work of Moore and Corbett (1971, 1973) set a standard for recording caries in skeletal remains. Their scheme for recording caries position on the tooth, which was slightly modified by Powell (1985), forms the basis for the recording in the Khok Phanom Di dentitions, with some further modifications as follows:

- Pit and fissure, located in the molar buccal and lingual pits and grooves.
- Interproximal, located at the point of contact between two adjacent teeth.
- Smooth surface, located on smooth buccal or lingual surfaces.
- Cervical, located at the cemento-enamel junction.
- Root, located on the root below the cemento-enamel junction.
- Occlusal, located on the occlusal fissures of premolars and molars.
- Massive, involving the crown of the tooth to an extent that obscures the site of the original caries.

Only those cavities which were undoubtedly carious were recorded, as the post-mortem damage to the teeth in some cases produced lesions which were equivocal. The number of teeth lost, both ante- and post-mortem, the fragile condition of the teeth in many cases, and the difficulty in removing the post-mortem deposits of calcareous material which often filled what may have been carious cavities, created some difficulty in identifying lesions precisely. For these reasons, the data collected are regarded as recording minimum frequencies of caries.

TABLE 96: *Prevalence of caries in adult dentitions, showing the number and proportion of individuals with caries and the number and proportion of teeth which were carious*

	N	Individuals with caries		Teeth			Means (s.e.) per individual ¹			
		n	%	present	carious		n teeth		present	carious
All adults	67	39	58	1282	139	11	20.4	1.2	3.3	0.4
Females	36	24	67	657	96	15	19.2	1.7	4.0	0.6
Males	31	15	48	625	43	7	22.3	1.8	2.9	1.7
Sex differences		ns ²		p=0.0001 ³			ns		ns ⁴	

¹The mean and standard error in numbers of teeth present and carious in those individuals with caries.

² $\chi^2=2.29$, $p=0.13$. ³ $\chi^2=19.81$. ⁴ $p=0.077$

The prevalence of caries in the adult dentitions is summarised in table 96. More than half (58 per cent) of all adults had at least one carious tooth and 11 per cent of all intact teeth were carious. The rate of caries formation in relation to age will not be considered in detail because dental pathology was taken into consideration in the assessment of age at death of some older individuals. Despite this, there were some sex differences in the pattern of caries formation which it is valid to consider. Although the proportion of teeth which were carious was significantly higher in females than in males, the mean number of caries per individual was not significantly different. Figure 77 illustrates

the number of carious teeth per individual against age for both sexes and shows that the male mean in particular is influenced by a few individuals. Further statistical analysis of caries numbers in relation to age and sex took into account the number of extant teeth per individual, using a Poisson regression model modified to adjust for the wide variation in individual patterns. From this analysis it appears that females have a higher caries rate than men, in addition to the expected increase in the number of caries with age in both sexes. Figure 77 also shows that two of the three females who died before the age of 20 had caries, compared with none of the four males.

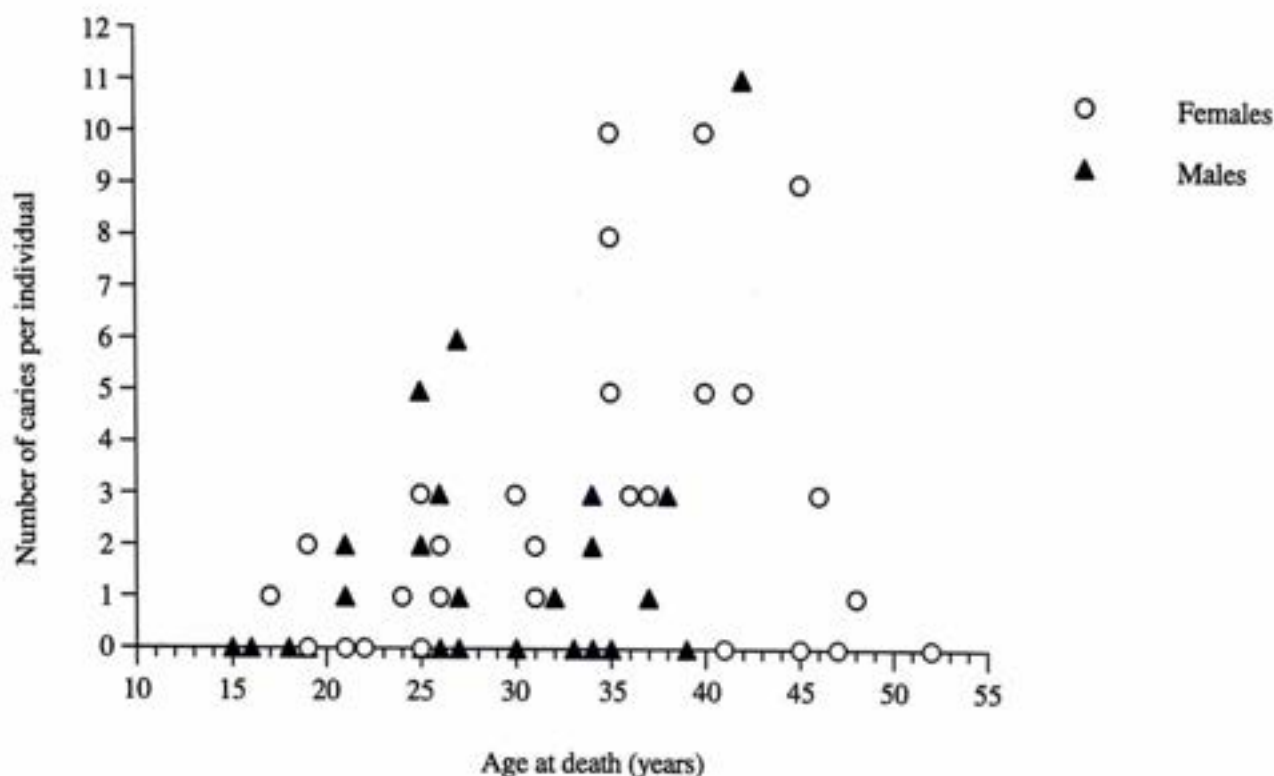


FIG. 77. The number of caries per individual for both sexes in relation to age at death

Table 97 indicates the prevalence of caries in adult dentitions by tooth type in those aged less than 30 years and those 30 years or older at death. In individuals aged less than 30 years at death, all but one of the caries were in molar teeth and more than half were in the third molars. The concentration in the molars and the predominance in the third molars are both statistically highly significant. In those over 30 years at death, there were again significantly more caries in the molars than in other teeth, although they were no longer concentrated in the third molars. The position of the caries on the teeth and the size of the lesions are summarised in table 98 and the variation in caries patterns with age in table 99.

Most caries formed in the pits and fissures of the molar teeth or on the interproximal surfaces of all the posterior teeth. No root caries were found. In many cases the caries had progressed to

TABLE 97: *Prevalence of caries in adult dentitions by tooth type*

	Age <30 years at death n=30 individuals				Age 30+ years at death n=37 individuals				All individuals n=67 individuals			
	Teeth ¹	Carious ²	All caries ³		Teeth	Carious	All caries		Teeth	Carious	All caries	
	N	n	%	%	N	n	%	%	N	n	%	%
Molars												
Third - 8	80	17	21	52	75	21	28	20	155	38	25	27
Second - 7	108	9	8	27	95	25	26	24	203	34	17	25
First - 6	110	6	5	18	90	13	14	12	200	19	10	14
Premolars	187	1	1	3	182	26	14	24	369	27	7	19
Anterior	179	0	0	0	176	21	12	20	355	21	6	15
All teeth	664	33	5	100	618	106	17	100	1282	139	11	100

¹Number of teeth assessed for caries²Number and proportion of teeth with caries³Caries in each tooth or tooth type as a proportion of the total cariesTests for significance: molars vs anterior in adults under 30 y, $\chi^2=38.09$, $p=0.0001$; molars vs anterior in adults over 30 y, $\chi^2=9.69$, $p=0.0018$; M3 vs M1,2 in adults under 30 y, $\chi^2=12.61$, $p=0.0004$, M3 vs M1,2 in adults over 30 y, not significantTABLE 98: *Prevalence of caries in adult dentitions by caries type, position and size for each tooth category*

	Molars		Premolars		Anterior		All Teeth	
	n ¹	% ²	n	%	n	%	n	%
Pit/fissure	30	33	0	0	0	0	30	22
Interproximal	24	26	14	54	1	5	39	28
Smooth surface	1	1	0	0	0	0	1	1
Cervical	10	11	0	0	0	0	10	7
Occlusal	0	0	0	0	6	28	6	4
Massive	23	25	11	42	14	67	48	34
Multiple	4	4	1	4	0	0	5	4
Total	92	100	26	100	21	100	139	100

¹Number of teeth with caries of each type²Proportion of all caries in each tooth type represented by each caries type

destroy the entire crowns (massive caries) on all types of teeth. The ubiquity of the massive caries, particularly in the older individuals but also in those under 20 years of age at death, suggests that, once caries formed, the lesions progressed rapidly. Table 100 summarises the positions of caries on

TABLE 99: *Prevalence of caries of each type by age at death*

	Age < 30 years at death			Age 30+ years at death		
	individuals n ¹	carious teeth n ²	% ³	individuals n	carious teeth n	%
Pit/fissure	11	17	52	10	13	12
Interproximal	4	5	15	13	34	32
Smooth surface	1	1	3	0	0	0
Cervical	0	0	0	6	10	9
Occlusal	0	0	0	4	6	6
Massive	6	9	27	17	39	37
Multiple	1	1	3	4	4	4
Total	14	33	100	25	106	100

¹Number of individuals with at least one tooth with each type of caries²Number of teeth with each type of caries³Proportion of all caries represented by each type of caries

the teeth by sex. The positions of caries varied between the sexes, with pit and fissure caries forming a greater proportion of the caries in the teeth of males and higher numbers and greater proportions of teeth with massive and interproximal caries in females.

TABLE 100: *Sex differences in positions of caries, with proportions of caries in each position*

	Females		Males	
	n ¹	% ²	n	%
Pit and fissure	14	15	16	37
Interproximal	28	29	11	26
Smooth surface	0	0	1	2
Cervical	8	8	2	5
Massive	36	38	12	28
Occlusal (anterior)	5	5	1	2
Multiple	5	5	0	0
	96	100	43	100

¹Number of caries in each position²Proportion of caries in each position

TABLE 101: *Prevalence of caries in the deciduous and permanent teeth of children*

	Deciduous		Permanent	
	Individuals	Teeth	Individuals	Teeth
n	18	178	10	158
n with caries/carious	6	11	4	10
%	33	6	40	6

The prevalence of caries in the dentition of children is summarised in table 101. In the deciduous teeth, all but one of the lesions occurred on the buccal surfaces of maxillary teeth, in areas of discoloured enamel. Eight of the eleven teeth affected were incisors. The remaining single lesion was in the fissures on the crown of a molar and was the only caries in the dentition of an older child. In the permanent teeth, eight of the ten caries were on first and second molars and occurred predominantly in the fissures of the occlusal surfaces or interproximally. Seven of the lesions were on maxillary teeth. One individual, B12.1, had caries on one canine and on the buccal surfaces of the maxillary first and second molars in a band of enamel which is discoloured.

There are difficulties in comparing caries rates among populations because of the lack of standardisation of the method of recording and reporting. Some reports have the individual as the unit of measurement, providing data on the number and proportion of individuals with one or more caries. Others use individual teeth as the unit of measurement, and report on the caries rate for the entire sample without regard for the characteristics of the individuals. The complexity of caries aetiology and epidemiology is largely ignored in favour of simplified statements on 'prevalence' which are then used as the basis for interpretation of interpopulation variations in caries rates. Most reports, and particularly those using teeth as the basic unit, ignore the age at death and sex of the sample, and the variation in patterns of caries within and between individuals. As previously mentioned, caries patterns need to be discussed in the context of the entire pattern of dental pathology, including periapical inflammation, ante-mortem loss and periodontal disease, and the pattern of non-pathological wear. In skeletal populations, the types of teeth and proportions of dentitions preserved are also relevant in determining the true caries rates, as anterior and posterior teeth are susceptible in different degrees to caries, and to ante-mortem and post-mortem loss. However, caries is often the only dental pathology which is systematically reported, and such comparative data as are available from both prehistoric skeletal groups and modern populations are summarised in table 102.

The proportion of individuals with caries at prehistoric South East Asian sites ranges from 37 per cent at Ban Na Di to 67 per cent at Gua Cha, with the proportion at Khok Phanom Di towards the upper end of the range. Where data are available for both sexes, more females than males have caries at all sites. For those sites where the unit of measurement is individual teeth, the proportion carious at Khok Phanom Di is higher than all other groups except the small sample from Non Pa Kluay, which has an extreme level of carious teeth. This suggests that the Khok Phanom Di people suffered a higher rate of caries than most other prehistoric South East Asians, although the relatively

good preservation at Khok Phanom Di, with a greater proportion of individuals having the entire dentition accounted for than at most other sites, increases the possibility of individuals having at least one carious tooth. There are no data available on the ages of those with carious teeth at the other sites, and the variation at Khok Phanom Di from 2 per cent of teeth carious in under 20-year-olds to c. 40 per cent carious in those surviving to over 40 years illustrates the relevance of the ages at death of the affected individuals.

TABLE 102: *Caries in prehistoric South East Asian and Pacific populations, with individuals and/or teeth as the unit of measurement*

	All adults		Individuals		Males		Teeth	
	n/N	%	n/N	%	n/N	%	All adults n/N	%
Prehistoric skeletal								
1. Khok Phanom Di	39/67	58	24/36	67	15/31	48	139/1282	11.0
2. Non Nok Tha								
Posterior	11/25	44	9/17	53	2/8	25		
Anterior	1/22	5	1/14	7	0/8	0		
3. Ban Chiang	23	-					22/326	6.7
4. Non Pa Kluay	15	-					59/150	39.0
5. Ban Na Di	11/30	37	5/12	42	5/13	38		
6. Gua Cha	12/18	67						
Neolithic							7/129	5.4
Hoabinhian							16/262	9.3
Pacific - prehistoric								
7. Nebira, PNG	7/34	21					44/725	6.1
8. Namu, Taumako	6/163	4					12/2084	0.6
9. Tonga	20/75	27					86/985	8.7
South Asia - prehistoric								
10. Harappa	17/39	44	10/16	63	6/17	35	51/751	6.8

2. Pietruszewsky 1974a; 3. Turner 1979; 4. Pietruszewsky 1988b; 5. From material held in the Anatomy Department, University of Otago; 6. Trevor and Brothwell 1962; 7-10. Evans 1987; 11. Lukacs 1992

On a broader scale of comparison, the proportion of individuals with caries at all South East Asian sites in general is high compared with those from the Pacific recorded by Evans (1987). Turner (1979) collected a comprehensive array of data on the prevalence of carious teeth in both skeletal and living, mainly pre-industrial, populations world-wide. He classified these according to mode of subsistence and in a grand summary concluded that the lowest prevalence of carious teeth was found in hunter-gatherer groups (mean teeth carious 1.3 per cent, range 0-5 per cent, both sexes combined, 19 populations), increasing in groups with mixed economies of agriculture

with some hunting, gathering or fishing, to a mean of 4.84 per cent (range 0.4–10.3 per cent, 13 populations), with a maximum in agricultural populations of 10.43 per cent (range 2.3–26.9 per cent, 32 populations). This study ignores the complexity of caries aetiology and the vast array of diets and cultural and genetic variation in these populations, but nevertheless does suggest a pattern of increased dental caries with the transition from the varied hunter-gatherer diet to one with a cultivated starchy dietary staple. The prevalence of 11 per cent at Khok Phanom Di is at the upper limit of the range of populations with mixed economies, and within the range of the agricultural populations.

The concentration of caries in the molar teeth at Khok Phanom Di (66 per cent of all caries) is similar to that found by Evans (1987) on the combined data from Pacific populations (58 per cent) and Powell (1985) on two Amerindian populations (76–84 per cent), although the relative frequency of caries among the molars varies from that reported in the only available closely comparative data from South East Asia on the Gua Cha population (Trevor and Brothwell 1962). At Khok Phanom Di the proportion of individual molars which are carious increases distally from 10 per cent on the first molar to 17 per cent on the second and 25 per cent on the third. Trevor and Brothwell found 12 per cent of first molars carious, 16 per cent of second and only 7.5 per cent of third molars. The high prevalence of third molar caries at Khok Phanom Di, particularly among individuals under thirty years of age at death, may be a reflection of the high proportion of these teeth with hypoplastic enamel. The high proportion of both massive caries and caries in pits and fissures in this age group could be a result of the same factor. In all the populations listed in table 102, a greater proportion of females than males have caries. In some cases the relative proportions are higher than at Khok Phanom Di.

Apart from the single individual with caries in the defective enamel of several teeth (B12.1), the above comments are also applicable to the prevalence of caries in the permanent teeth of children. The co-occurrence of enamel defects with the majority of the caries in the deciduous dentition represent an association which has been seen both in other prehistoric populations and in modern populations, as was previously noted. The association was seen in 14–31 per cent of children in modern populations with low living standards (Jelliffe and Jelliffe 1971), and in 16–24 per cent of deciduous dentitions in prehistoric Amerindians from Illinois with a high proportion of enamel defects (Cook and Buikstra 1979). Duray (1990) also found a significant association of caries with enamel hypocalcifications in particular in the deciduous teeth of 57 children in the Amerindian skeletal population from Libben, and suggests possible mechanisms for this. One involves the substitution of magnesium for calcium in hypoplastic areas of enamel, and the other is a variation in the alignment of the hydroxyapatite crystals in the enamel, which facilitate the cariogenic action. The five children at Khok Phanom Di (28 per cent of individuals) with caries associated with defective enamel is in the same range as observed on these other populations, and the caries in the band of enamel hypocalcification of the permanent dentition in B12.1 are very likely to have the same aetiology.

EXCESS WEAR

The destruction of the enamel surfaces of the tooth crown through chewing food becomes pathological only when there is insufficient hard tissue to prevent the passage of pathogens through the root canal to the supporting tissues. Wear is primarily evident on the occlusal surface (attrition, caused by the regular chewing actions), and although there is slight abrasion of all crown surfaces and interproximal wear between teeth, these affect only the surface enamel. Removal of areas of the occlusal enamel, a common occurrence in prehistoric peoples, is to some extent compensated for by the formation of secondary dentine, which protects the pulp chamber. Ultimately, if attrition continues at a faster rate than formation of secondary dentine, or when at least part of the crown is substantially destroyed, the pulp chamber becomes exposed and the root canal open to infection.

Wear was recorded for all permanent teeth, using the eight point scale devised by Molnar (1971). The degree of wear has been used in the estimation of age at death of the Khok Phanom Di adults. Teeth showing heavy wear (Molnar Grades 6-8) have been examined in determining those which may have pulpal exposure and possible infection. The confirmation of infection is the presence of periapical alveolar cavities in conjunction with extreme wear and the absence of caries.

Table 103 summarises the prevalence of wear which may have exposed the pulp chamber of the teeth (Molnar grades 6-8), providing a potential route for infection of the tooth and the supporting tissues. Under 33 per cent of adults had at least one tooth with excessive wear, and 9 per cent of all teeth were excessively worn. Although the proportion of individuals of each sex with worn teeth did not differ significantly, the total number of excessively worn teeth and the mean number per individual were significantly higher in males than in females. Males with excess wear also had a significantly higher mean number of teeth present. Figure 78 shows the numbers of teeth per individual with excess wear by age at death for both sexes. This illustrates the higher numbers worn in older males than in older females.

Table 104 summarises the prevalence of excessive wear by tooth type. Wear is evenly spread around the dental arcade. The numbers and degree of tooth wear in the older individuals are subject to variations in occlusion and chewing habits resulting from both the presence of caries and the loss of teeth. The interpretation of the data on excess wear will be considered in conjunction with observations on the other features of the teeth.

Interobserver differences in the classification of dental wear make comparisons difficult. The available data from prehistoric Thai are listed in table 105. At both sites, similar proportions of individuals had extreme wear on their teeth, although the age structure of the individuals involved at Non Nok Tha is not known. At Ban Na Di, the pulp cavity was exposed in individuals in their forties (Houghton and Wiriyaromp (1984), and males had a higher degree of wear than females of the same age (Wiriyaromp 1984a). At Khok Phanom Di the pattern was similar, with males also having a higher degree of wear than females of a similar age. The extreme wear was evident in the males from age 35 and the females from age 40.

TABLE 103: *Prevalence of excess wear (Molnar grade 6-8), showing the number and proportion of individuals with excessively worn teeth and the number and proportion of excessively worn teeth*

	Individuals ¹ with excess wear			Teeth ² present excess. worn			Means (s.e.) per individual ³ n teeth			
	N	n	%	N	n	%	present		excess. worn	
All adults	67	20	30	1272	112	9	14.2	1.7	5.6	1.1
Females	36	12	33	644	35	5	11.2	2.3	2.9	0.6
Males	31	8	26	628	77	12	18.6	1.1	9.6	1.9
Sex differences			ns			p=.0001	p=.024		p=.001	
M>F						$\chi^2=18.45$	t=2.5, df=18		t=4.0, df=18	

¹The number of individuals included in the sample and the number and proportion of individuals with one or more excessively worn teeth

²The number of teeth which were assessed for wear and the number and proportion of teeth with excess wear

³The mean numbers of teeth present and excessively worn in those individuals with excessively worn teeth

TABLE 104: *Prevalence of excess wear by tooth number, showing the proportion of teeth excessively worn in those individuals with excess wear*

	Age <40 years n=11 individuals				Age 40+years n=9 individuals				All individuals n=20 individuals			
	Teeth ¹		Excess ²		Teeth		Excess		Teeth		Excess	
	N	n	%	All ³	N	n	%	%	N	n	%	%
Molars	84	26	31	41	41	13	32	27	125	39	31	35
Premolars	60	22	37	34	43	17	40	35	103	39	38	35
Canines	26	11	42	17	19	11	58	23	45	22	49	20
Incisors	19	5	26	8	22	7	32	15	41	12	29	10
All teeth	189	64	34	100	125	48	38	100	314	112	36	100

¹ Number of teeth assessed for wear

² Number and proportion of teeth with excess wear

³ Excess wear in each tooth or tooth type as a proportion of the total excess wear

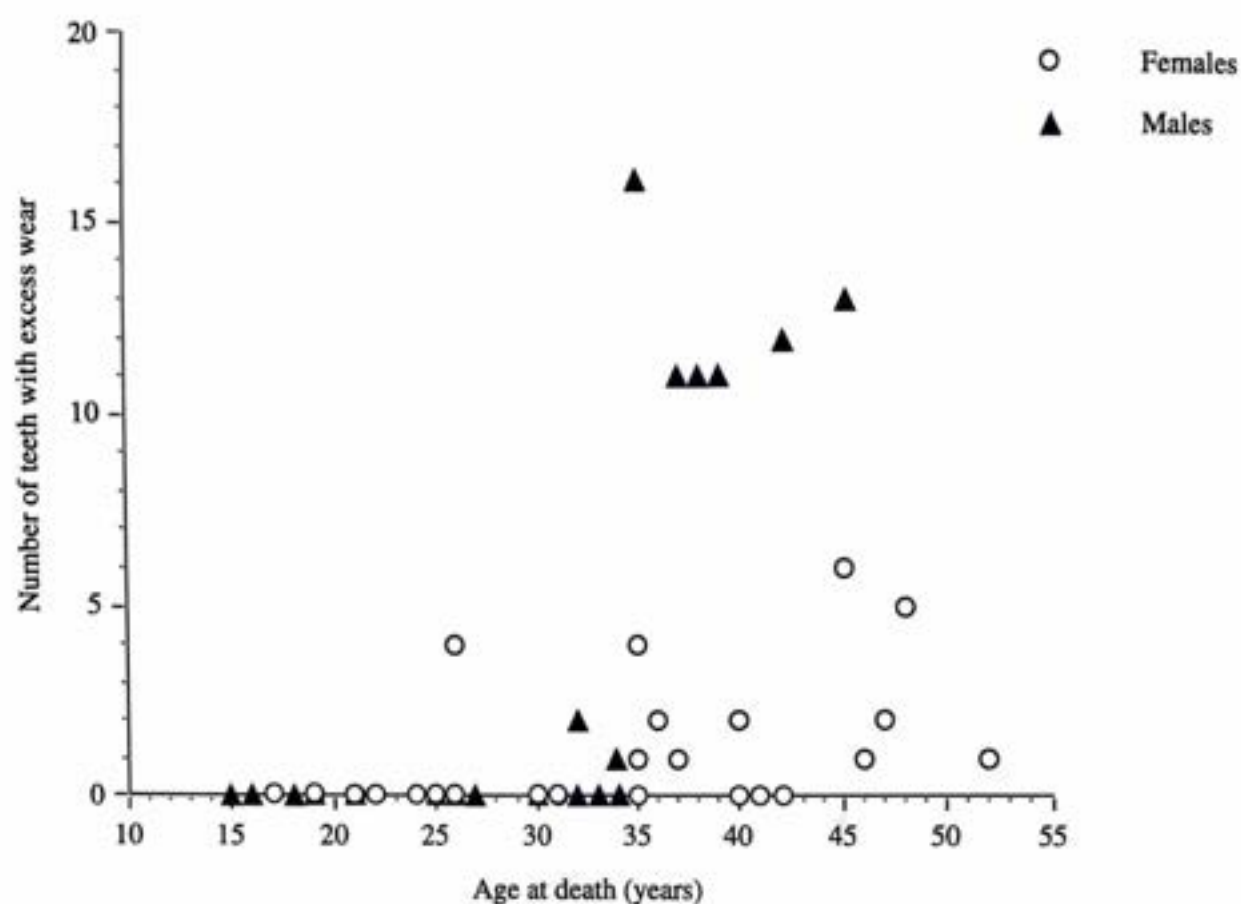


FIG. 78. The number of teeth with excess wear per individual for both sexes in relation to age at death

TABLE 105: *Excess wear (exposure of pulp chamber or wear to root level on posterior teeth) from various South East Asian prehistoric populations. Sources of data as for Table 102*

	Females		Males		All adults	
	n	%	n	%	n	%
Individuals						
Khok Phanom Di	31	16	36	16	67	16
Non Nok Tha	16	12	8	12	24	12

PERIODONTAL DISEASE

Clinically, the other major oral pathology associated with the dentition is periodontitis, which affects the supporting tissues (periodontium) rather than the teeth. The periodontium consists of four different tissues, alveolar bone, periodontal ligament, root cementum and gingiva, which "...constitute and behave as a developmental, biological and functional unit." (Schroeder 1986:12). The initial pathology preceding periodontitis is gingivitis, an inflammation of the soft tissues resulting from an immune response to the micro-organisms present in plaque. As the disease progresses, the deeper supporting tissues become involved, pockets form in the gingiva, the periodontal ligament is destroyed, and the crest of the alveolar bone is resorbed as the overlying gingiva degenerates. Pockets also form in the alveolar bone around the root of the tooth. Ultimately, the tooth is loosened in its socket (Pawlak and Hoag 1984), although the disease may progress intermittently and slowly (Hillson 1986).

As with dental caries, the initiating factor in periodontitis is the presence of bacterial plaque. A number of other factors, both local and systemic, are involved in determining the susceptibility of an individual to the disease through involvement in the bacterial composition and concentration of plaque, and the nature of tissue response to its presence. These include genetic factors, lowered resistance through systemic disease, hormonal changes and nutritional deficiencies, and local factors such as diet, oral hygiene, tooth loss or crowding, caries and calculus (Pawlak and Hoag 1984).

The identification of periodontitis in skeletal remains is complicated by the continuing adaptation of the dentition and remodelling of the alveolar bone to changes such as attrition, and loss of teeth during life. The remodelling rate of alveolar bone is apparently higher than that in most of the other bones, probably in response to adaptation of the dentition in maintaining functional occlusion during rapid reduction of tooth size through occlusal and proximal attrition and abrasion (Schroeder 1986). The distance between the cemento-enamel junction (CEJ) and the crest of the alveolar bone is 1-2 mm in healthy modern dentitions (Pawlak and Hoag 1984). This distance is often measured in skeletal analyses and taken as evidence of alveolar bone resorption as a consequence of periodontitis where it exceeds 2 mm (Brothwell 1981). However, continued eruption of teeth in maintaining occlusal function to recompense for attrition of the occlusal surface will increase the distance even without alveolar bone loss. In establishing if an increase in the distance between the CEJ and the crest of the alveolar bone is due to alveolar resorption rather than continued eruption of the tooth, other aspects of the condition of the interproximal bone such as abnormal porosity and the formation of pockets by resorption adjacent to teeth will indicate periodontitis (Pawlak and Hoag 1984).

The alveolar bone in all adults was examined for evidence of periodontal disease. The post-mortem encrustation of the skeletons with calcareous deposits created problems of diagnosis in many individuals, as the deposits were often particularly heavy around the teeth, and their removal caused some damage to the alveolar bone. As with caries, the periodontal disease recorded represents a minimum prevalence.

As was noted previously, the distance between the cervical-enamel junction and the alveolar crest which is often used in skeletal studies as evidence of alveolar resorption as a consequence of periodontal disease, is not a good indicator. The assessment of the presence of periodontal disease

in the Khok Phanom Di dentitions has, therefore, been based on the condition of the bone of the alveolar crest and the conjunction of two or more of the following indicators:

1. Marked resorption of the alveolar crest, to the extent that the root furcation is exposed, or more than half of the root of a single-rooted tooth.
2. The presence of pockets, particularly around the posterior teeth.
3. Reactive bone (moderate - severe porosity) on the alveolar crest.
4. An irregular alveolar crest, the result of irregular alveolar resorption.

The evidence of periodontal disease is less easily reported systematically and in detail than that of other oral pathologies, as the alveolar bone forms a continuous entity, unlike the discrete units represented by the teeth. The recording was, therefore, made for anterior and posterior divisions of the alveolar bone, rather than for individual teeth.

Table 106 summarises the numbers of adults with evidence for periodontal disease, by age at death. Recording the condition of the alveolar bone was particularly difficult around the anterior teeth, where the ablation of teeth resulted in alveolar resorption. Resorption and remodelling of the alveolar crest in response to periodontal pathology is not always distinguishable from resorption and remodelling following ante-mortem tooth loss, particularly in individuals aged 40 years and over.

In those aged less than 20 years at death, one individual (B140) had evidence of porosity around the first and second maxillary molars and pockets around the same teeth in the mandible, with evidence otherwise minimal in that group. In those aged 20–29 years at death, porosity and resorption was almost exclusively limited to the bone surrounding the molars, both maxillary and mandibular, and the pockets were formed distally to the second and third molars. In those aged 30–39 years at death, most of the recorded porosity and resorption was again around the molars and the degree of resorption was greater. The formation of pockets in the bone was again almost exclusively limited to the alveolar crest distal to the second and third molars. As has already been mentioned, in the oldest individuals the ante-mortem loss of multiple teeth and consequent remodelling of the alveolar crest made observation of the bony response to specifically periodontal pathology haphazard.

The data for each sex show that the proportions of individuals of both sexes subject to periodontal problems were similar at all ages, although, apart from the single female aged under 20 years at death, the numbers of teeth involved in those males with pockets were generally greater in males of all ages than in female dentitions.

At Khok Phanom Di evidence occurred in the form of porosity, resorption of the alveolar crest or pockets in the bone adjacent to the crest, in one third of individuals aged less than 20 years. By the age of 40 years all individuals had some problems. At Non Nok Tha, Pietrusewsky (1974a) recorded medium-marked resorption of the alveolar crest in just over half of adult dentitions, compared with evidence found in 77 per cent of the Khok Phanom Di dentitions, but variation in the method of identifying the pathology and the question of age at death of the individuals concerned rule out drawing too many conclusions from this information.

TABLE 106: *Prevalence of periodontal disease by sex and age at death in adults*

	Porosity/resorption ¹			Pockets ²		
	N	n	%	N	%	Mean
All adults						
15–19 years	6	2	33	2	33	3.00
20–29 years	20	14	70	6	30	2.33
30–39 years	22	19	86	9	41	3.11
40+ years	9	9	100	2	22	1.50
Females						
15–19 years	3	1	33	1	33	5.00
20–29 years	11	7	63	4	36	2.25
30–39 years	9	8	89	4	44	2.75
40+ years	7	7	100	1	14	1.00
Males						
15–19 years	3	1	33	1	33	1.00
20–29 years	9	7	78	2	22	2.50
30–39 years	13	11	85	5	38	3.40
40+ years	2	2	100	1	50	2.00

¹Number and proportion of individuals with evidence of periodontal disease in the form of porosity and/or resorption of the alveolar crest; ²Number and proportion of individuals with evidence of periodontal disease in the form of pockets and the mean number of teeth affected per individual

CALCULUS

The actions of bacterial plaque in forming caries require conditions of low pH in the mouth, but in the absence of low pH, plaque becomes mineralised as calculus (Hillson 1986). Calculus may form supragingivally, or in the pockets formed by periodontal disease in the gingiva adjacent to the teeth. The exact relationship between subgingival calculus and periodontal disease is not clear. Subgingival calculus formation may be the effect of periodontitis rather than the cause, and the presence of plaque on the surface of the calculus deposit is more likely to be pathogenic than the calculus itself (Legler and Menaker 1980). The teeth most commonly affected are the lingual surfaces of the mandibular incisors and the buccal surfaces of the posterior maxillary teeth. The composition of the diet, oral hygiene, and genetic factors such as the composition of the saliva are all involved in the individual susceptibility to calculus formation. Calculus is easily dislodged from dentitions

during excavation, transport and cleaning and its absence is not necessarily indicative of absence during life.

Recording the presence of calculus on the Khok Phanom Di dentitions was difficult because of the post-mortem calcareous encrustations. These may have bonded with calculus on the tooth surfaces, and their removal resulted in the removal of the calculus. All adult dentitions were inspected for evidence of calculus, and its presence recorded where possible. Calculus was recorded as present on the dentitions of 14 individuals, 10 males and 4 females. The prevalence is summarised in table 107.

TABLE 107: *Prevalence of calculus*

Grade of calculus	Anterior			Posterior			Entire arcade		
	1	2	3	1	2	3	1	2	3
Maxilla	1	1	-	1	2	1	1	1	1
Mandible	3	-	1	1	1	-	1	2	-

The minimal data warrant little further analysis, as they cannot be assumed to reflect the rates of dental calculus formation or the extent of calculus within individual dentitions. They do indicate that calculus was forming on at least some of the dentitions and that in some cases deposits were heavy. Most of the cases of slight deposits were individuals under the age of 25 years and the moderate and heavy deposits individuals over the age of 25 years. There is a predominance of males recorded with calculus. No published information is available for calculus prevalence at other prehistoric sites in South East Asia.

PERIAPICAL INFLAMMATION

Infection of the dental pulp may occur as a result of exposure of the pulp to oral bacteria through caries, attrition or trauma. Once an infection is established, it spreads down the root canals and through the apical foramen into the periapical region. This produces an inflammatory response in the periapical tissues comprising the periodontal ligament and surrounding alveolar bone. If the host immune response is unable to overcome the infection, the inflammatory reaction will become chronic, most commonly resulting in the development of a periapical granuloma. The evidence of this in dry bone is a smooth-walled cavity around the root apex. Over time, the granuloma may develop into an periapical cyst. Almost all the well-demarcated periapical bony cavities seen in maxillae and mandibles are created by these benign lesions. Most periapical granulomata and cysts are asymptomatic. Even when symptoms such as slight pain when biting on solid food are present, they are quite mild. As long as the bony cavity is occupied by a granuloma or cyst, there will be no systemic effects.

If the infection is severe and involves pyogenic (pus-producing) organisms, a periapical abscess will form. This may result from acute infection of the pulp, or more commonly establish secondarily

in a pre-existing granuloma or cyst. If the infection causing an acute abscess is partly but not fully overcome, the abscess may persist and become chronic. Chronic abscesses do not have any systemic effects, so the individual will feel quite healthy. As with a periapical granuloma or cyst, the pain is mild but is accompanied by sinuses discharging a foul-smelling exudate. Nevertheless any lesion represents the potential for involvement of dental infection in an individual's death, as impairment of the immune system or virulence of the causative organism could result in the development of a serious acute abscess or osteomyelitis within the lesion.

Periapical cavities are usually easily identified in skeletal remains, although they are often incorrectly identified as abscesses or abscess cavities. It is also sometimes possible to identify variation in the bone which indicates remodelling of a periapical cavity after removal of the infection. In this analysis, the identification of a cavity as an abscess is based on the formation of a draining sinus. The prevalence of periapical cavities, including remodelled cavities, has been recorded in all Khok Phanom Di dentitions with permanent teeth. The initial source of the infection has also been identified, where possible, by considering the condition of the relevant teeth and alveolar bone.

TABLE 108: *Prevalence of periapical cavities, showing the number and proportion of individuals with periapical cavities and the number and proportion of tooth positions with periapical cavities*

	Individuals ¹ with cavities			Tooth positions ² with cavities			Means (s.e.) per individual ³ n tooth positions			
	N	n	%	N	n	%	present		with cavities	
All adults	67	37	55	2047	122	6	30.4	0.5	3.3	0.5
Females	36	21	58	1103	78	7	30.7	0.5	3.7	0.6
Males	31	16	52	944	44	5	30.1	1.1	2.8	0.6
Sex differences		ns		$F > M \chi^2 p = .002$						

¹The number of individuals included in the sample and the number and proportion of individuals with one or more periapical cavities; ²The number of tooth positions which were assessed for periapical cavities and the number and proportion of tooth positions with periapical cavities; ³The mean numbers of tooth positions present and with periapical cavities in those individuals with cavities

Periapical cavities, either active or remodelled, were recorded in 37 adult dentitions (Table 108). More than half of adults had at least one periapical cavity and 5 per cent of all tooth positions (including those where the tooth had already been lost) had a periapical cavity. The proportions of adults affected and the mean number of periapical cavities per individual was similar in both sexes. Figure 79 shows the increase in periapical cavities with age, which was similar in both sexes. Despite the similarity, the data for individual tooth positions shows that there were significantly more tooth positions with periapical cavities in females than in males. The three adults discussed in chapter V with evidence of abscesses draining into the maxillary sinuses are all female.

Table 109 shows the distribution of periapical cavities around the dental arcade according to age. Periapical cavities were more common in the area of the molars than the anterior teeth in

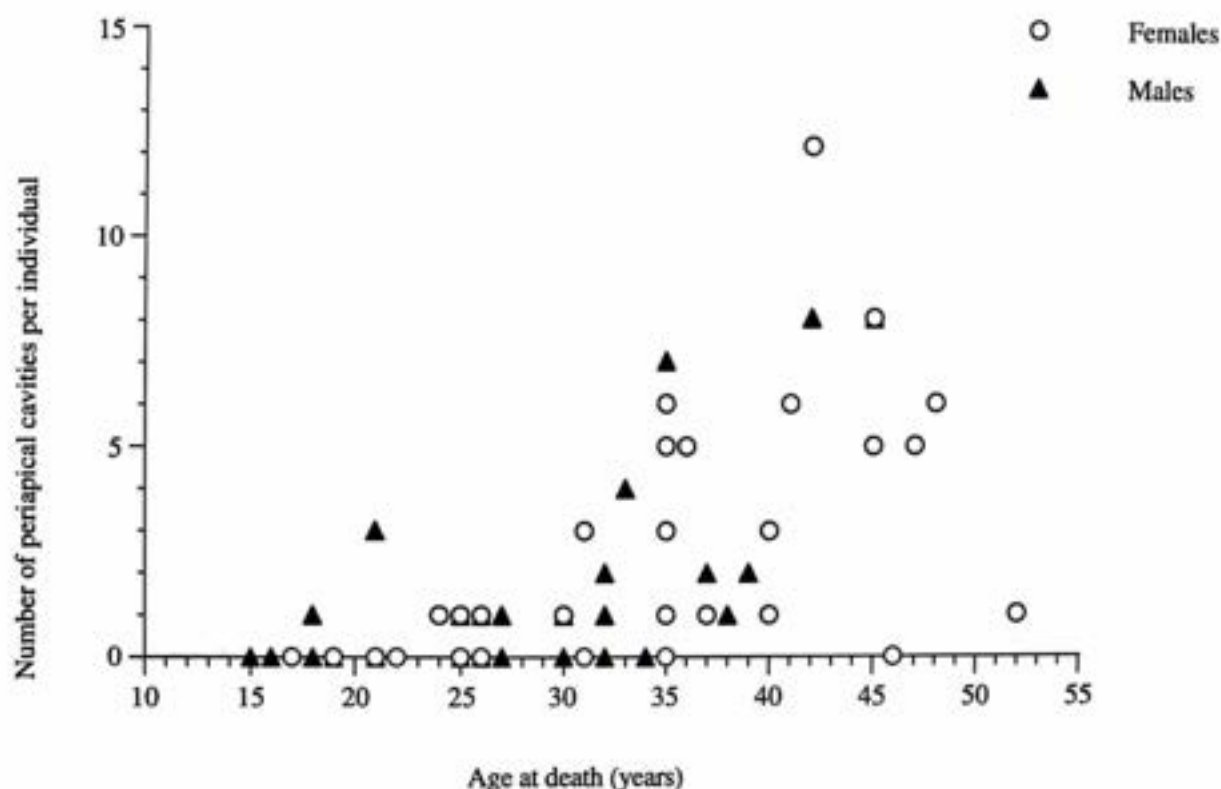


FIG. 79. The number of periapical cavities per individual for both sexes in relation to age at death

younger individuals, although the difference is not significant. In those over 30 years old, the infections were spread throughout the dental arcade. Seventeen per cent of the periapical cavities had been remodelled in these older individuals, as a result of the loss of the tooth which had been the source of the infection.

Table 110 shows that the primary cause of periapical inflammation in individuals aged less than 30 years at death was caries. In older females caries and exposure of the pulp chamber through excess wear contributed equally, whereas in males excess wear was the cause of a greater number of periapical cavities.

Periapical cavities in the dentition are less likely to be subject to interobserver variation in recording than other characteristics, although they may be misidentified as 'abscesses'. The observations made on other prehistoric South East Asian populations are listed in table 111. At Ban Na Di, four of the individuals aged over 45 years at death had periapical cavities. A greater proportion of the population at Khok Phanom Di had dental periapical cavities than at Ban Na Di or Non Nok Tha, except in the males of the latter site. In comparison with the data from Gua Cha, where the unit of measurement is teeth rather than individuals, the proportion of periapical cavities at Khok

TABLE 109: *Pattern of periapical cavities by tooth number and age, showing the proportion of tooth positions with periapical cavities*

	Age <30 years n=30 individuals				Age 30+years n=37 individuals				All individuals n=67 individuals			
	Teeth ¹		P Cavity ²		Teeth		P Cavity		Teeth		P Cavity	
	N	n	%	All ³	N	n	%	All	N	n	%	All
Molars												
M3	118	3	3	27	129	7	5	6	247	10	4	8
M2	118	2	2	18	140	17	12	15	258	19	7	16
M1	118	2	2	18	141	24	17	22	259	26	10	21
Premolars	234	0	0	0	286	27	9	24	520	27	5	22
Canines	117	0	0	0	143	17	12	15	260	17	7	14
Incisors	225	4	2	37	278	19	7	18	503	23	5	19
All teeth	930	11	1	100	1117	111	10	100	2047	122	6	100
Molars vs Anterior	ns (Molars > anterior $\chi^2 3.09, p=.079$)				ns							

¹Number of tooth positions assessed for periapical cavities; ²Number and proportion of tooth positions with periapical cavities; ³Periapical cavities in each tooth or tooth type as a proportion of the total periapical cavities

TABLE 110: *Summary of primary sources of dental infection*

	Female			Male			All adults		
	<30 years	30+ years	All	<30 years	30+ years	All	<30 years	30+ years	All
Caries	1	19	20	4	7	11	5	26	31
Wear		19	19		20	20		39	39
Unknown		37	37	4	5	9	4	42	46
Total	1	75	76	8	32	40	9	107	116

Phanom Di is not high. At Non Nok Tha there was a higher prevalence of periapical cavities in the males than in the females, but at Khok Phanom Di slightly more females had periapical cavities.

TABLE 109: *Pattern of periapical cavities by tooth number and age, showing the proportion of tooth positions with periapical cavities*

	Age <30 years n=30 individuals				Age 30+years n=37 individuals				All individuals n=67 individuals			
	Teeth ¹	P Cavity ²	All ³		Teeth	P Cavity	All		Teeth	P Cavity	All	
	N	n	%	%	N	n	%	%	N	n	%	%
Molars												
M3	118	3	3	27	129	7	5	6	247	10	4	8
M2	118	2	2	18	140	17	12	15	258	19	7	16
M1	118	2	2	18	141	24	17	22	259	26	10	21
Premolars	234	0	0	0	286	27	9	24	520	27	5	22
Canines	117	0	0	0	143	17	12	15	260	17	7	14
Incisors	225	4	2	37	278	19	7	18	503	23	5	19
All teeth	930	11	1	100	1117	111	10	100	2047	122	6	100
Molars vs Anterior	ns (Molars > anterior $\chi^2 3.09, p=.079$)				ns							

¹Number of tooth positions assessed for periapical cavities; ²Number and proportion of tooth positions with periapical cavities; ³Periapical cavities in each tooth or tooth type as a proportion of the total periapical cavities

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	<30 years	30+ years	All	<30 years	30+ years	All	<30 years	30+ years	All
Caries	1	19	20	4	7	11	5	26	31
Wear		19	19		20	20		39	39
Unknown		37	37	4	5	9	4	42	46
Total	1	75	76	8	32	40	9	107	116

Phanom Di is not high. At Non Nok Tha there was a higher prevalence of periapical cavities in the males than in the females, but at Khok Phanom Di slightly more females had periapical cavities.

TABLE 111: *Prevalence of periapical cavities in South East Asian prehistoric populations (Sources of data as for table 102)*

Individuals	Females		Males		All adults	
	n	%	n	%	n	%
Khok Phanom Di	36	58	31	52	67	55
Non Nok Tha						
- anterior	17	6	8	25	25	12
- posterior	18	6	8	50	26	19
Teeth						
Khok Phanom Di	1103	7	944	5	2047	6
Gua Cha						
- Hoabinhian					235	4
- Neolithic					107	4

In a number of studies of prehistoric populations reviewed by Alexandersen (1967), between 2–9 per cent of teeth were affected. The sources of infection were cited as caries, attrition and periodontitis.

ANTE-MORTEM TOOTH LOSS

The ultimate effect of severe dental pathology can be loss of the tooth. Any pathology or combination of pathologies which results in sufficient resorption of the alveolar bone to loosen the tooth in its socket can be responsible for tooth loss. Teeth which have continued to erupt to compensate for extreme occlusal wear will be less well supported by bone and will be relatively easily lost after further minor alveolar resorption. Remodelling and resorption of the alveolar crest after tooth loss makes identification of the cause of the loss difficult unless it occurred shortly before death. Other possible explanations of missing teeth include loss following trauma, and deliberate ablation as a cultural practice. The anterior teeth are more likely to be involved in these cases, whereas the posterior teeth are more greatly affected by pathology, so the pattern of loss may provide some clues as to general cause. The greatest difficulties arise in identifying the cause of loss in older individuals who have very few teeth or are edentate.

The indication that a tooth was lost ante-mortem is the remodelling and infilling of the tooth socket in the alveolar bone. Table 112 summarises ante-mortem loss through pathology. As the alveolar bone was virtually complete in most individuals, the data on teeth lost during life are a reasonably accurate assessment of the true prevalence of the problem. The diagnosis of loss was

straightforward in most cases, except where there had been severe periodontal disease. In those cases it was difficult to distinguish a socket partially remodelled following loss of a tooth from one where the tooth may have been present but the supporting alveolar bone was partially resorbed. It was also difficult to confirm whether a missing third molar was lost ante-mortem or had never formed, particularly where the adjacent molars were also lost. The ablation of anterior teeth also created difficulties in the identification of exact numbers of teeth lost during life from pathological causes. Particularly in older individuals, where teeth were lost around the entire dental arcade, it was not always possible to establish whether there had been any deliberate evulsion of teeth, or which teeth had been removed. In cases of doubt, the missing teeth were classified as being lost through pathological causes.

TABLE 112: *Prevalence of ante-mortem tooth loss from pathological causes, showing the number and proportion of individuals with teeth lost and the number and proportion of teeth lost*

	Individuals ¹ with tooth loss			Tooth positions ² lost			Means (s.d.) per individual ³			
	N	n	%	N	n	%	n tooth positions present		teeth lost	
All adults	67	34	51	2047	183	9	30.4	0.5	5.4	5.0
Females	36	24	67	1103	142	13	30.7	0.5	5.9	5.3
Males	31	10	32	944	41	4	29.4	1.7	4.1	4.2
Sex differences							ns		ns	

¹The number of individuals included in the sample and the number and proportion of individuals with one or more teeth lost; ²The number of tooth positions which were assessed and the number and proportion of teeth lost; ³The mean and standard deviation of tooth positions present and teeth lost in those individuals with teeth lost

Half of all adults had lost at least one tooth from pathological causes during life, and 9 per cent of all teeth had been lost. Two-thirds of females had lost at least one tooth, compared with one-third of males. Females had lost a mean of six teeth each compared with four lost by the males, although this difference was not statistically significant. Again, the use of dental pathology in estimating age at death in older adults negates the value of detailed comparison of the rate of tooth loss with age, although figure 80 illustrates that more females than males had lost teeth before the age of thirty.

Table 113 summarises the teeth lost by tooth position. In those aged less than 30 years at death, one male (B28) has an anomalous number of teeth lost compared with the remainder of this age group. Apart from in this individual, all losses were of molar teeth and the majority were third molars in young individuals. In those aged 30 years and over at death, there were losses of all types of teeth, although the highest rate was again recorded in the molar teeth. Third molars appear to be the most common teeth lost, although the difference between third and other molars is no longer significant.

At Khok Phanom Di, 9 per cent of all adult teeth were lost during life. At Non Nok Tha, the total was 7.6 per cent of teeth, 6 per cent in females and 10.2 per cent in males. The Neolithic population

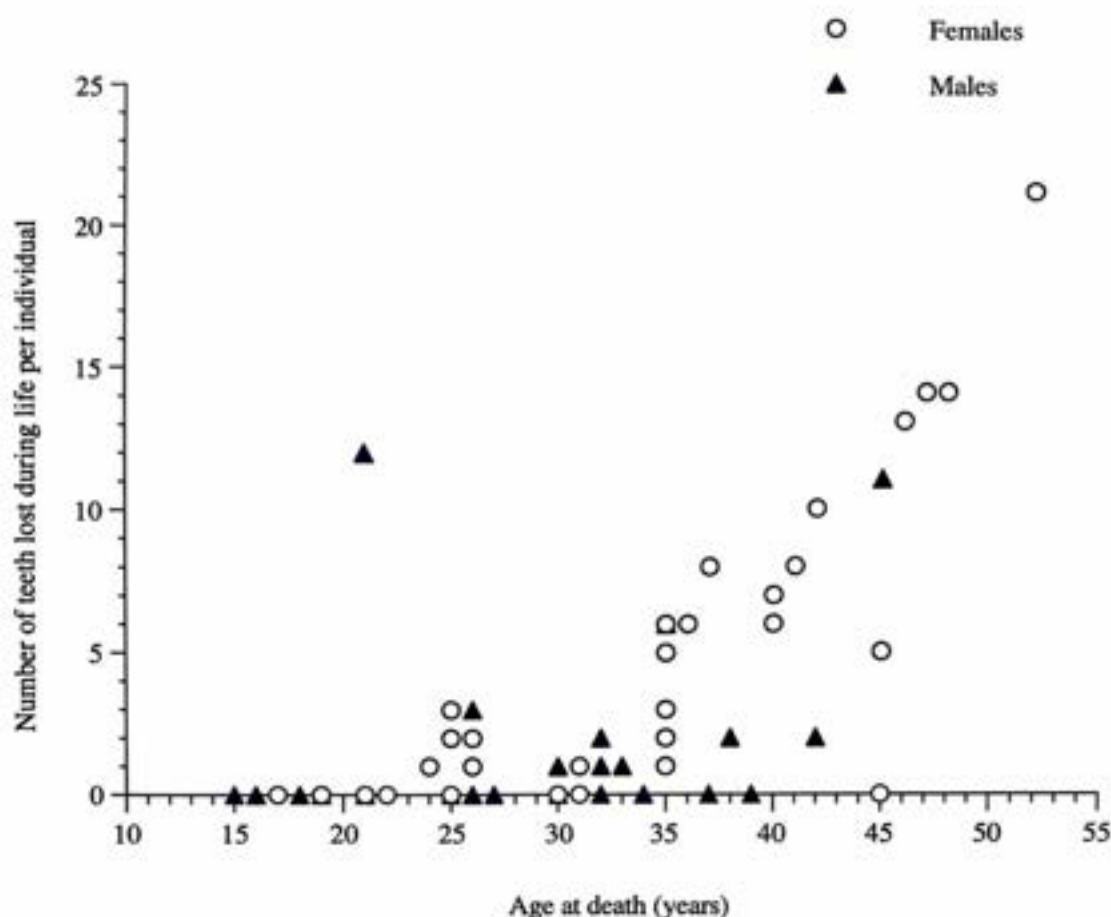


FIG. 80. The number of teeth lost per individual during life for both sexes in relation to age at death

at Gua Cha lost a similar proportion of teeth during life as the Khok Phanom Di people, and the Hoabinhian sample lost 6 per cent. There is a sex difference in losses at Khok Phanom Di with females having lost 14 per cent and males 3 per cent. The pattern of higher tooth loss in the females partly reflects their higher age at death, but the pattern was already established before the age of 40, as mentioned in the previous section.

COMPARISON WITH MODERN POPULATIONS

In modern populations, dental epidemiology is generally based on rates of decayed, missing and filled (DMF) teeth. Data on DMF rates in modern Thai adults are available from surveys done in the 1960s (Leatherwood *et al.* 1965). The sample of 890 females and 750 males was drawn from all

TABLE 113: *Prevalence of ante-mortem tooth loss by tooth type, showing the proportion of teeth lost*

	Age <30 years N=30 individuals				Age 30+years N=37 individuals				All individuals N=67 individuals			
	Teeth ¹		Lost ²		Teeth ¹		Lost ²		Teeth ¹		Lost ²	
	n	n	%	%	n	n	%	%	n	n	%	%
Molars												
M3	118	12	10	46	129	32	25	21	247	44	18	24
M2	236	6	3	23	281	49	17	31	517	55	11	30
Premolars	234	4	2	15	286	33	12	21	520	37	7	20
Canines	117	2	2	8	143	10	7	6	260	12	5	7
Incisors	225	2	1	8	278	33	12	21	503	35	7	19
All teeth	930	26	3	100	1117	157	14	100	2047	183	9	100
With B28 excluded												
	N=29 Individuals								N=66 individuals			
	Teeth		Lost						Teeth		Lost	
	n	n	%	%					n	n	%	%
Molars												
Third	116	10	9	71					245	42	17	25
First, second	232	4	2	29					513	53	10	31
Premolars	230	0	0	0					516	33	6	19
Canines	115	0	0	0					258	10	4	6
Incisors	221	0	0	0					499	33	7	19
All teeth	914	14	2	100					2031	171	8	100
Molars >	$\chi^2=23.12$				$\chi^2=17.43$							
Anterior	p=.0001				p=.0001							
M3 > M1,2	$\chi^2=9.53$				ns ($\chi^2=3.03$							
	p=.002				p=.0818)							

¹ Number of tooth positions assessed for tooth loss; ² Number and proportion of teeth lost; ³ Teeth lost of each tooth type as a proportion of the total tooth losses

areas of Thailand. Almost all individuals were from rural areas, and the brief information given on the average diet shows that it was adequate in calories, 65 per cent of which were supplied by rice, but low or partially deficient in calcium, iodine, vitamin C, thiamine and riboflavin. It is assumed that the diet included some processed foods containing sugar. Table 114 shows the comparison of DMF rates in the modern Thai sample with decayed and missing teeth recorded at Khok Phanom Di.

Table 115 compares the rates in a smaller subsample of modern Thai from Central and Southern Thailand (Leatherwood *et al.* 1965). The size and sex composition of the modern sample is not given, but it appears to be greater than 2,000 individuals, of whom about two-thirds were males. The information is also shown graphically in figures 81 and 82.

TABLE 114: *Comparison of prevalence of teeth decayed and missing (DM) in the Khok Phanom Di adults with teeth decayed, missing and filled (DMF) in a sample of modern Thai (Leatherwood et al. 1965:1795)*

Age	Khok Phanom Di				Modern Thai			
	Females		Males		Females		Males	
	n	Mean DM	n	Mean DM	n	Mean DMF	n	Mean DMF
0-4	-	-	-	-	36	0.0	33	0.0
0-9	-	-	-	-	137	0.2	134	0.1
10-14	-	-	-	-	160	0.8	139	0.4
15-19	3	1.0	5	0.0	82	1.1	43	0.8
20-24	5	0.4	1	0.5	76	1.2	40	0.8
25-34	11	2.7	18	1.8	149	0.9	112	0.6
35-44	11	11.3	5	5.0	90	1.5	112	1.5
45+	6	13.3	1	11.0	160	8.1	137	9.2

These data show that the DMF rates were slightly higher in modern Thai adolescents, but from the age of 20 the Khok Phanom Di people experienced a more rapidly increasing rate of tooth decay and loss. Decay and tooth loss in females at Khok Phanom Di accelerated rapidly in the thirties and in the males in the forties rather than the fifties as in the modern group. Leatherwood *et al.* (1965:1797) note that the rapid increase in the DMF rate after the age of 45 in the modern Thai is due to loss of teeth through rampant terminal and severe periodontal disease. They do not give separate data for males and females for the sample from southern and central Thailand, but in a larger nation-wide sample there is no sex difference in the increase of the DMF rate from age 45. On the basis of a series of large-scale surveys in the decades following the second World War (including the survey reported by Leatherwood *et al.* 1965), DMF rates in three countries in South East Asia (Thailand, Vietnam, Myanmar (Burma)) were shown to follow a similar pattern. The rates were judged relatively low on an international scale (Russell 1966). The pattern of increase with age was similar in all three countries. None of the reports of these surveys gives details of the teeth which are carious or of the prevalence of dental care, although the latter is likely to have been minimal. The prevalence of periodontal disease was also similar in the three countries, with relatively high rates of severe periodontitis in individuals aged over 40 years compared with international standards

TABLE 115: *Comparison of the mean numbers of teeth decayed and missing in Khok Phanom Di adults with mean numbers of teeth decayed, missing and filled in a sample of modern Thai from Central and Southern Thailand (Leatherwood et al. 1965:1795)*

Age	Khok Phanom Di n	DM(F)	Modern DMF
0-9	5	0.60	0.36
10-19	13	0.77	1.16
20-29	21	2.55	1.37
30-39	25	4.32	1.44
40-49	11	12.18	4.31
50+	1	21.00	13.59

TABLE 116: *Association of dental pathologies in females and males aged 30-39 years at Khok Phanom Di*

	Females		Males	
	Individuals %	Teeth %	Individuals %	Teeth %
Caries	100	23	50	4
Excess wear	36	4	43	18
Periodontal disease	86	-	89	-
Periapical cavities	82	8	57	5
Ante-mortem loss	82	10	43	3

(Russell 1966). Because of different methods of defining and scoring periodontal disease in the modern populations compared with the methods used for the Khok Phanom Di dentitions, the data from the two groups are not directly comparable. Nevertheless, periodontal disease recorded in the Khok Phanom Di people appears to have started earlier and progressed at a more rapid rate than in modern populations. Overall, it appears that the dental health of the Khok Phanom Di people followed a similar pattern to that in modern South East Asians, except that deterioration progressed at an accelerated rate. This is assuming that the age estimations for the Khok Phanom Di are correct.

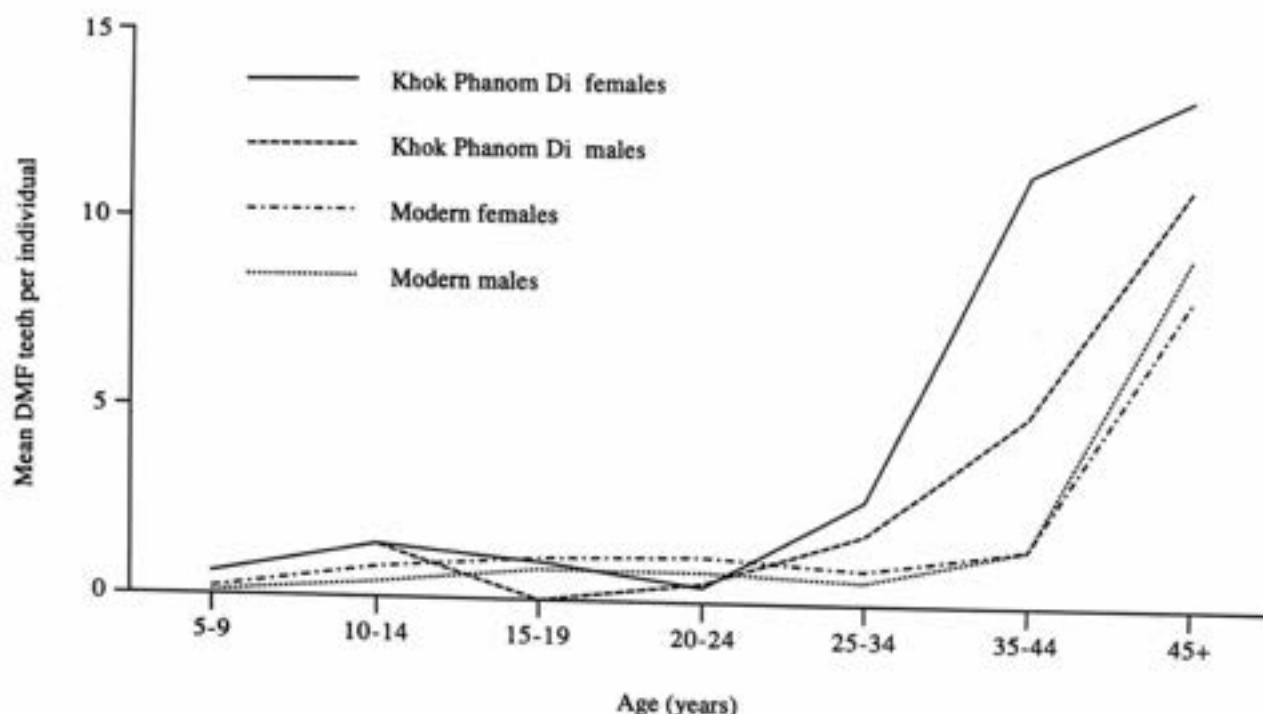


FIG. 81. Comparison of the mean numbers of teeth decayed and missing in the Khok Phanom Di adults with mean numbers of teeth decayed, missing and filled in a sample of modern Thai (composition of samples as in table 115)

SEX DIFFERENCES IN DENTAL HEALTH

The association among dental pathologies in the Khok Phanom Di people is shown in table 116. This provides data for the 30–39 year age group where the samples of males and females are of similar size and the pattern of dental degeneration is established.

This shows that there is a sex-specific pattern of dental deterioration in this age group. The females have high rates of caries, periodontal disease, and loss of teeth during life, whereas in the males the major pathology is periodontal disease, together with extreme wear. They have lower rates of periapical cavities and ante-mortem loss than females. Similar proportions of individuals of both sexes have some teeth with excessive wear, but the males have a higher proportion of worn teeth per individual. That similar proportions of individuals have excessively worn teeth in both sexes suggests that the cause of wear is similar in both sexes, but that the females are perhaps selective in the areas of the dental arcade used in mastication because of caries. The proportion of

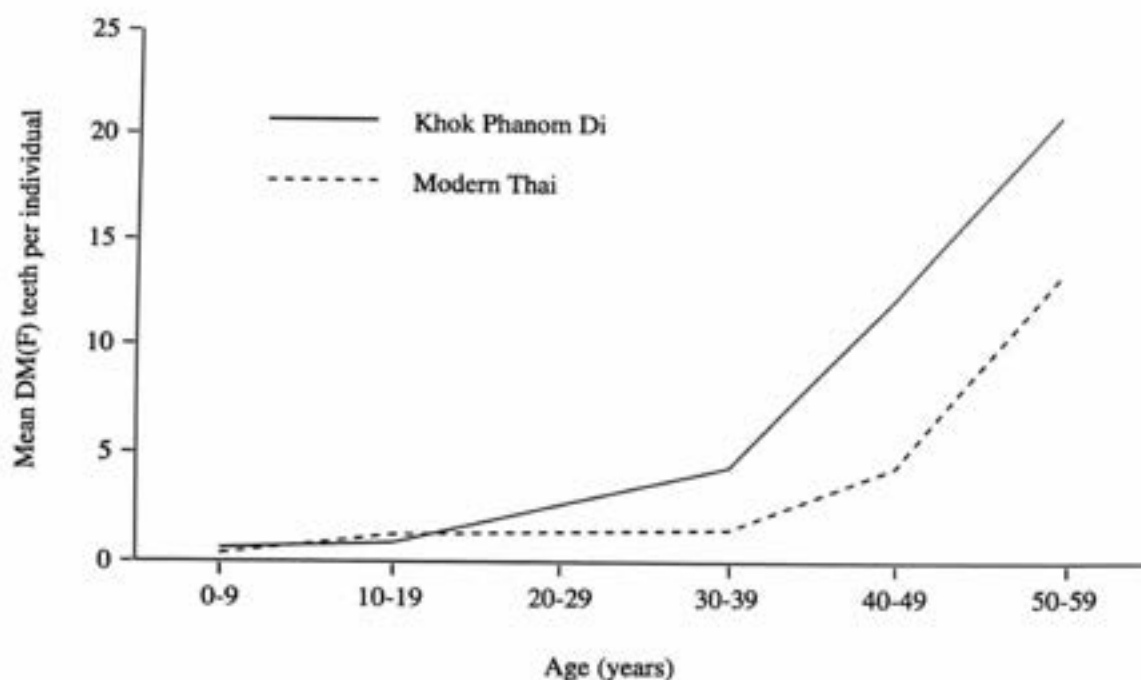


FIG. 82. Comparison of the mean numbers of teeth decayed and missing in the Khok Phanom Di population with mean numbers of teeth decayed, missing and filled in a sample of modern Thai from Central and Southern Thailand (Leatherwood *et al.* 1965:1795)

decayed teeth may have caused them to adapt chewing patterns to minimise pain. The high proportion of teeth lost during life would have reduced the numbers which were occluding and therefore reduced the wear on the remaining teeth. An alternative explanation for the sex differences is that more rapid wear in the male dentitions reduced the chances of caries developing by removing the caries-prone occlusal fissures earlier in life.

None of the other prehistoric Thai sites where data are available had such a distinct sex difference in overall dental pathology other than Non Nok Tha, where twice as many females as males had caries and half of the males had evidence of infection in the form of periapical cavities compared with less than ten per cent of females.

SUMMARY

Compared with almost all prehistoric samples from north eastern Thailand, the dental health of the adults at Khok Phanom Di is poor, with slightly higher rates of caries. These, combined with periodontal problems, contributed to a markedly higher frequency of periapical inflammation and resulted in relatively high rates of tooth loss during life. The sample from Gua Cha, northern

Malaysia, is the only prehistoric group with dental health comparable to that at Khok Phanom Di. However, when compared with international standards in modern populations, the dental health of the people at Khok Phanom Di is good, with relatively low rates of decay and tooth loss, particularly in early adulthood. This pattern is similar to that of the modern South East Asian peoples. In later adulthood, the condition of the Khok Phanom Di dentitions deteriorated at a more rapid rate than in their modern counterparts. There are marked sex differences in dental health at Khok Phanom Di, with that of the females very much poorer.

VII. AETIOLOGY AND EPIDEMIOLOGY: INTERPRETATION AND EXPLANATION OF HEALTH AND DISEASE

AS discussed in chapter I, health in this context is defined as the result of the combined influences of genetics and nutrition, disease, and patterns of physical activity on the phenotype. The skeletal and dental evidence of health and pathology from the people at Khok Phanom Di has been reported and summarised in comparison with other prehistoric peoples in previous chapters. As the next stage in the analysis, this chapter first outlines non-skeletal evidence for diet and disease. This is then considered in conjunction with the results of the skeletal analysis, in a discussion of the diagnosis and epidemiology of the diseases affecting the Khok Phanom Di people and the interrelationship of other factors determining their health status. It concludes with a summary of the suggested explanations for patterns of health, morbidity and mortality.

NUTRITION

The discussion of nutrition will first establish what the people at Khok Phanom Di were most likely to have been eating, through a consideration of non-skeletal archaeological evidence and of ethnographic evidence for diet in modern rural Thai. This information then forms the basis for a discussion of the nutritional quality of the prehistoric diet. The association between diet and dental pathology was mentioned in chapter VI. The extent to which the reconstructed diet correlates with and contributes to the patterns of dental pathology at Khok Phanom Di is considered in the section on pathology.

Archaeological evidence for diet

The archaeological evidence for food resources available to the people at Khok Phanom Di has been summarised in chapter I and is considered in more detail here. As with all archaeological sites, the material recovered from excavation which can be assumed to represent the debris from food is biased in that it is dominated by those items which have an inorganic component. In some circumstances organic materials will be preserved and recovered but it is not likely that the proportions of organic and inorganic materials will give an accurate indication of the proportions of these materials in the diet. In addition to differential preservation, there is also the question of the extent to which the excavated sample is representative of the normal diet of the people. This is particularly relevant at Khok Phanom Di, where a substantial depth of the excavation area represented a burial ground rather than occupation area. The food material deposited in these levels is possibly representative of particular foods selected for feasts associated with the mortuary ritual. Although these items may also have been included in the normal diet, there is the possibility that some items were consumed only on special occasions, or that the relative quantities consumed varied significantly

from the normal consumption rate. The evidence from food residues are, therefore, considered here principally as an indication of the foods available. The relative quantities of different foods identified can only be assumed to represent the quantities in the normal diet in a very general way.

The most obvious food residues recovered were the large numbers of edible shellfish. These are almost all species which are commonly found in mangrove swamps, mud flats and a sandy shore. Although there are at least fourteen edible species represented, a 'cockle' (*Anadara granosa*) was by far the most numerous, with over 900,000 shells recovered. This coastal species prefers silty, muddy conditions such as offshore from a river mouth or estuary. The next most common species are a marine clam (*Meretrix lusoria*) with over 26,000 shells and *Polymesoda erosa*, a mangrove bivalve with over 11,000 shells. All other species were represented by less than 3,000 shells (Mason 1991). Marine crabs of the species *Scylla serrata* were present in substantial quantities (Kijngam 1991). Many fish bones were recovered and included marine, freshwater and estuarine species. Bagrid catfish were the most common. The range of habitats occupied by the fish includes freshwater and estuarine areas. *Lates calcarifer*, an anadromous fish, Tachysurid catfish which favour estuarine conditions, and the freshwater fish *Clarias* sp. were also present. Direct evidence of the consumption of a freshwater species of fish, *Anabas testudineus*, and of the stingray (*Dasyatis* sp.) comes from the identification of remains of these fish in food residue from the pelvic area of B56 (Greenwood 1991).

There were few mammal bones, and most represent animals from swampy, estuarine areas with vegetation such as mangrove. The most common mammals were pigs *Sus scrofa*, (minimum number of individuals (MNI) 82), deer (*Cervus* sp., MNI 145), the crab-eating macaque (*Macaca* sp., MNI 76) and the leaf monkey (*Presbytis* sp. MNI 12). Other mammals with smaller minimum numbers (<10) of individuals include the water buffalo (*Bubalus bubalus*), cattle (Bovidae), small deer (*Muntiacus muntjak* or *Axis porcinus*) cats, civets and otters (Grant and Higham 1991). Relatively large numbers of rats (*Rattus rattus* and *Rattus* sp. MNI 305) were recovered (West 1991). A variety of aquatic bird species were also represented but in small numbers (total MNI 12) (Grant and Higham 1991, West 1991). On the basis of ethnographic analogy, West (1991) suggests that all these species may have been exploited for food. A single bone from a species of jungle fowl or chicken (*Gallus gallus*) raises the possibility that the bird was domesticated, as it is anomalous in a mangrove environment (West 1991), although presumably more bones would have been recovered if it was domesticated for use as food.

There was temporal variation in the composition of these potential animal foods. In MP1-3, there were many crabs and *Lates calcarifer* but few of the larger mammals. Eight of the nine most numerous edible shellfish species were present during this period. In the later mortuary phases, there were relatively large numbers of the freshwater and estuarine species of fish and shellfish (Kijngam 1991, Mason 1991, West 1991). The number of crabs declined steadily during MP4-7 and by the end of the zone the variety of edible shellfish species was reduced with only four of the nine most common species present. Although large numbers of *A. granosa* were consumed, the density per 10 cm spit in MP4-7 was less than half that in MP1-3 (Mason 1991). This may be sampling effect or reflect cultural factors such as a change in the composition of mortuary feasts, although it may also be a reflection of the environmental change discussed in chapter I. There was a wider variety of mammal species in MP4-7.

The other significant evidence for diet from the excavation is the indication of the consumption of rice. Rice husks were identified in the coprolite from B67, the pelvic contents from B56, within the deposits at all levels in the occupation layers, and as temper in pot manufacture (Thompson 1996). The presence of artefacts such as shell knives with wear compatible with rice harvesting (Higham T. 1993), sandstone grinding stones and granite hoes (Pisnupong 1988) all support the presence of rice in the diet. Thompson (1996) has identified the rice husks from the coprolite from B67 as that of a domesticated species, indicating the probable cultivation of rice rather than collection from wild plants. The remains of a beetle found in the B67 coprolite have been identified as *Oryzophilus surinamensis*, which is a pest of stored products, including rice (Harris 1991). This offers the possibility that rice was produced in such quantities that there was some available for storage against seasonal food shortages. There were no other plant remains from the site which were unquestionably identified as food debris, although some were of wild plants recorded ethnographically as food. These include the genera *Eleocharis* (water chestnut) and *Amaranthus* (spinach-like leaves) (Thompson 1996). Evidence from the pollen cores indicates that other potentially cultivated food plants growing in the vicinity of the site in prehistory include the coconut palm (*Cocos nucifera*, Maloney 1991). Less likely, but possible, cultivars include *Citrus* comp. (possibly pommelo, lemon, lime, orange), *Eugenia* comp. (rose apple), *Zizyphus* (juzube) and *Garcinia* (*G. mangostana* = mangosteen). Their presence in the area at least indicates that their fruits were available for gathering and consumption. Other possible non-cultivated plant food species identified in the pollen cores are *Pandanus* sp. (used as rice flavouring - Jacquat 1990).

Ethnographic evidence for modern Thai diet and nutrition

The earliest ethnographic information located on diet in rural Thailand is from data collected in the 1950s. This includes anecdotal summaries of personal observations (Suvarnakich 1950), a detailed study undertaken in the 1950s in a rural village north east of Bangkok (Hauck and Sudsaneh 1959, Hauck *et al.* 1959) and a series of other less detailed observations of diets and eating habits (Bisolyaputra and Bocobo 1959, Chalawyo and Tansathit 1959, Chandrapanond 1959).

The most important constituent of the diet of the modern Thai is rice, which is eaten at all meals. It is considered to be so essential to eating that the word is used as a synonym for food and it is traditionally believed to provide all the sustenance necessary for human consumption (Suvarnakich 1950, Wilson 1975). All other foods are prepared as accompaniments to rice. Based on the Bang Chan survey (Hauck *et al.* 1959, Hauck and Sudsaneh 1959), the second most important food is fish, which is eaten fresh, dried, or fermented and concentrated in the form of fish sauce. Other animal foods were relatively unimportant and only small quantities of beef and pork were eaten. Chicken was eaten only on special occasions but eggs were regularly used. A variety of vegetables and fruit including bananas, tamarind, pineapples, and leafy green vegetables such as swamp cabbage (*Ipomea aquatica*), are eaten in varying quantities. The swamp cabbage, which was the most common vegetable eaten at Bang Chan is not cultivated but grows wild along the canals. Coconut is eaten daily as coconut meat, milk or cream. Spices and condiments are an essential component of Thai meals. Wild creatures such as eels, frogs, turtles, cobra and other snakes were used for curry, sometimes on special occasions for men, and field rats (presumably the large paddy rat *Bandicota indicus*) were roasted and eaten all year round.

A variety of food plants which form part of the traditional Thai diet grow wild in the habitats identified as existing in the vicinity of Khok Phanom Di in prehistory. These include seaweed, young succulent leaves from a variety of plant types, and leaves and stems from plants which grow in freshwater swamps (Thompson 1996).

Much of the data available on the nutritional status of the Thai is from studies based in urban Bangkok, where the diet includes non-traditional items, or in the rural areas of Northern and North eastern Thailand where the diet is inadequate because of poverty and the relative infertility of the land. No detailed data on the diet of modern coastal Thai have been found but Bisolyaputra and Bocobo (1959) report figures for consumption of foods by gross weight for families from the city of Chonburi (on the south-eastern coast) compared with villagers in North and North East Thailand. These data show that the consumption of fish by the coastal Thai was four to five times that of the inland peoples and that their diet included an overall consumption by weight of twice the animal foods. Wilson, on the basis of a study of diet in an East Coast Malay fishing village, suggests that:

.....most Southeast Asian subsistence rice eaters who have access to fresh fish have, despite a high starch diet, a relatively good amino acid intake pattern and, therefore, protein intake, and their nutritional status with respect to this nutrient is usually satisfactory. (Wilson 1985:68)

When variation in diet on the basis of socio-economic status is disregarded, the nutritional value, both in quantity and quality, of the foods available to the Thai in the more fertile areas of Central Thailand is more than adequate in virtually all respects.

Rice provides the major source of calories and a substantial proportion of the vegetable protein. The relatively high biological value of the protein in rice is reflected in the lower frequency and lesser severity of protein malnutrition in rice-eating communities compared with communities relying on other cereal staples such as maize (FAO 1954). The nutritional value of the protein in rice is enhanced by the consumption of fish at the same meal. Of the eight amino acids essential in protein synthesis only lysine is missing from rice and as fish is a good source of this (Geiger and Borgstrom 1962, Wilson 1985), the amino acid quality of the rice is increased. The protein in fish has a high nutritive value, possibly above that of beef, and is adequate as a principal source of dietary protein (Geiger and Borgstrom 1962, Guha 1962). The protein content of crustaceans in general is also high, equivalent to that in fish, and although that of molluscs is somewhat lower, it is nevertheless potentially a significant source (Borgstrom 1962b). Access to fish and other aquatic foods in addition to good supplies of rice provides a diet adequate in protein and calories. The good protein status of coastal dwellers with ready access to fish compared with their inland counterparts has been documented in various parts of the world outside South East Asia, including Brazil and Senegal (Geiger and Borgstrom 1962).

Rice is low in some vitamins and minerals, particularly vitamin A, the B group of vitamins, calcium and iron (FAO 1954). Methods of preparing and cooking foods are important in determining the extent to which the nutrients are retained and available. The only 'frank' modern Thai deficiency disease is beriberi (Suvarnakich 1950, Chalawyo and Tansathit 1959, Hauck and Sudsaneh 1959, Jelliffe 1968). This reflects inadequate supplies of thiamine and has been a problem only since the

introduction of modern rice-milling. Both the preference for highly milled white rice and methods of cooking involving excess water which is then discarded greatly reduce the nutrients in the rice (FAO 1954). Chalawyo and Tansathit (1959) illustrate this clearly in comparing the estimated daily nutrient intakes of Thai naval personnel and military convicts. The latter have much higher intakes of vitamin A, thiamine and riboflavin, as they are fed under-milled rice which is cooked without discarding water, whereas the naval personnel eat highly milled rice steamed in individual bowls with consequent nutrient loss. The nutritional status of those eating rice prepared by traditional husking and home pounding methods is high. The extent to which the nutrients deficient in rice can be obtained from fish varies greatly between species. Methods of cooking fish are also important in determining the calcium and iron available in the diet. The consumption of whole small fish and the cooking of larger fish on the bones greatly increases the accessible calcium in the diet (Borgstrom 1962a) and the supply of vitamin A is greatly increased if the liver is eaten. Shellfish are a rich source of calcium and thiamine (Borgstrom 1962b).

The evidence of surveys of modern Thai diets suggest that the supply of iron was adequate even in the poorer areas (Bisolyaputra and Bocobo 1959, Hauck and Sudsaneh 1959), although the intake of iron does not necessarily indicate its biological availability. This is influenced by the source of iron, the mix of foods consumed and the presence of substances such as phytates, oxalates, tannins and crude fibre (Monson *et al.* 1978, Pellett 1987). The absorption of iron is inhibited by the phytates in rice but fish protein improves the biological availability of iron in vegetable foods by two or three times (Gordon 1987). Some species of fish are also rich in iron. The inclusion of fish in a meal with rice and vegetables has been shown to be particularly effective in increasing the absorption of iron (Hallberg *et al.* 1978).

Jelliffe (1968) reported that infant feeding in modern Thailand in the 1950s was considered good compared with other tropical countries. Prolonged breast feeding supplemented by boiled rice and banana provides an adequate infant diet, and weaning in later infancy onto an adult diet of rice with added animal protein does not present any problems. Infants are described as "...healthy and relatively plump, although perhaps somewhat flabby and anaemic." (Jelliffe 1968:50). In the neighbouring South East Asian countries of Myanmar and Malaysia, the nutritional status of infants is not as good as in Thailand but Jelliffe notes that this is not because of the lack of availability of food but stems from cultural prejudices and taboos in the feeding both of pregnant women and infants (1968:48).

Diet and nutrition at Khok Phanom Di

The geographical situation of Khok Phanom Di would have been suitable for the exploitation of non-terrestrial foods. Shellfish can be exploited with minimal technology by all members of a group, including less physically active children and older adults. These factors and the spatial and temporal reliability of the beds, make exploitation of the shellfish a desirable option despite high transport and processing costs (Yesner 1987). Crustaceans would also be relatively easy to exploit and freshwater fish could be netted or caught with lines without any requirement for hazardous excursions out to sea.

The pattern of exploitation of terrestrial plant foods may have been similar to that still seen in Central Thailand, with cultivation of rice and collecting of coconuts, fruits and vegetables. Many of

the latter are now cultivated but the pollen analysis suggests that many were at least growing wild in prehistory and some may have been cultivated. The plant foods were occasionally supplemented through the hunting of small mammals such as macaques and pigs.

The diet of the people at Khok Phanom Di therefore probably included rice, fish and coconuts, the major food items which dominate the modern diet of the majority of countries of South East Asia (Wilson 1975). As has been discussed above, this diet is nutritionally balanced when the foods are available in sufficient quantities and are accompanied by fresh fruit and vegetables. Whether the same diet was also nutritionally adequate in prehistory depends on the reliability of all food sources and the quality of the prehistoric cultivars. The reliability of seafoods as a source of protein has already been mentioned but the majority of fish, shellfish and crustaceans are comparatively low in fat (Yesner 1987) and are not a rich calorie source. This is particularly so in tropical areas, although there are seasonal variations with a peak in fat and protein content during the spawning season (Yesner 1987). Protein can be used by the body as a source of energy but it is a relatively inefficient source and may be able to provide safely only up to 50 per cent of the daily calorie requirements (Noli and Avery 1988).

The value of the combination of carbohydrate-rich rice with seafoods in improving protein uptake has already been discussed and the evidence of both the cultivation and storage of rice at Khok Phanom Di increases the possibility that the supply was both reliable and adequate in quantity to supply the food energy requirements of the population through at least part of the year. Supplementary sources of calories in the event of seasonal shortages could have included coconut and plants which are now used seasonally by aboriginal groups foraging in similar coastal environments in Northern Australia. These include taro (*Colocasia* spp.), yams (*Dioscorea* spp.) and water chestnuts (*Eleocharis* sp.) (Meehan 1982, Petterson 1977, cited in Thompson 1996). From the mangrove, the 'fruit' (germinating embryo or hypocotyl) of *Bruguiera* sp. is rich in both protein and carbohydrate and has been widely documented as a food source. Their abundance and reliability makes them a potentially valuable source of nutrition but as they require elaborate processing they could have been less favoured than other food items (Thompson 1996). Bananas are another possible source of carbohydrates. There is taxonomic evidence that bananas originated from a primary centre on the Malayan Peninsula or closely neighbouring territories (Simmonds 1976). Yams originated in Myanmar (*Dioscorea alata*) and South East Asia (*Dioscorea esculenta*) (Onwueme 1978). Taro (*Colocasia esculenta*) also originated in South East Asia (Purseglove 1972). It is possible that they were present in Central Thailand at the time of the occupation of Khok Phanom Di.

The excavation evidence suggests that terrestrial animal foods were not consumed on a scale that could have provided a significant source of fat-derived energy but the peak in fat and protein quality of the marine foods would have provided an alternative energy source during the seasonal fluctuations in the availability of rice. Sources of vitamins and minerals could have been adequate, as they would be similar to those available in the modern Central Thai diet, with the improvement of unpolished rice.

The reduction in the variety and probably also the quantity of aquatic foods following the environmental change during MP4 could have resulted in deterioration in the quality of the diet. The most important loss is protein, although iron and some vitamins are also likely to have been reduced. There is the possibility that the fish protein was replaced by protein from terrestrial animals,

although the adequacy of this replacement is not clear. Thompson (1996) suggests that the rice was unlikely to have been growing in the immediate vicinity of the site in the early period of occupation, as the plants are not salt-tolerant and are unlikely to have survived in the mangrove habitat. This would have necessitated travel beyond the influence of the tidal estuary to find suitable freshwater swamps. With the environmental change, as Thompson suggests (1996:301), the reduction in the salinity of the site environs would have improved the accessibility of rice-growing land. This implies improved availability of this dietary staple, together with other freshwater food plants. Increasing reliance on these foods for energy and perhaps also protein is probable. Although the diet may have continued to provide adequate calories, there is the possibility that seasonal shortages were compounded and that the replacement of the lost animal protein with inferior quality vegetable protein resulted in an overall deterioration in nutritional quality.

In summary, the aquatic foods available to the people at Khok Phanom Di should have provided adequate protein, particularly in the early period of occupation of the site. Proficiency in the cultivation of rice and access to coconuts and a proportion of the other fruits and vegetables which grow in habitats similar to those around the site could have provided adequate calories, vitamins and minerals. This assessment of necessity includes many assumptions as to quality, quantity and reliability of food supplies. The combination of rice and fish is particularly nutritious and although there appears to have been a variety of potential calorie resources when rice supplies were inadequate, replacement sources of protein in the later period of occupation of the site may have been difficult to find. The questions of seasonal fluctuations in diet and the maintenance of the high quality nutritional level throughout the period of occupation of the site are left open.

Regardless of the availability of a nutritionally adequate diet, the synergistic interaction between infection and nutrition can create malnutrition through interference in the absorption or use of nutrients. Infections which cause loss of appetite, loss of blood, or chronic diarrhoea are of major importance. The types of infection which are of particular relevance in this interaction at Khok Phanom Di are discussed in more detail below.

Apart from the quantity and nutritional quality of the food available at Khok Phanom Di, the diet of individuals could have been affected by human factors such as cultural or social taboos and feeding preferences. Examples of the food taboos in modern groups involve the feeding of expectant and nursing mothers and infants. The persistence of such practices in modern Myanmar and Malaysia have already been mentioned and they have also been recorded in Thailand (Stahlie 1960, Suttapreyasri *et al.* 1975) and Cambodia (Sargent *et al.* 1983). These include the beliefs that pregnant women should not eat green vegetables, or that postpartum or lactating women should not eat crustaceans, molluscs, fish without scales, eggs, meat or raw vegetables. At its most extreme, the diet is restricted to rice and salt water. Among some indigenous groups in Malaysia, there is a belief that babies should not be fed eggs or fish (Jelliffe 1968). These practices have a significant effect on the health of the neonate and compromise the survival of the infant, particularly during the vulnerable period when the immune system is developing, and during weaning. The preferential consumption of wild foods such as turtles, eels and frogs by the men in Bang Chan (Hauck *et al.* 1959) is a modern Thai example of the restriction of high-protein foods to one sex.

PATHOLOGY

In identifying the diseases suffered by the people at Khok Phanom Di, the skeletal evidence of specific skeletal pathology reported in chapter V and dental pathology in chapter VI is the first and most direct source of information. There is also indirect archaeological evidence of relevance in identifying specific pathogens. Ethnographic evidence for pathogens in modern Thailand is also considered, where appropriate.

The major questions arising from the study of specific skeletal and dental evidence are the aetiology of the anaemia and the explanations for patterns of skeletal and dental degeneration. The remainder of specific bony reactions have affected a small proportion of the population and often only a single individual. Some contribute to the identification of the aetiology of the anaemia. Others are not as epidemiologically important with almost all a reflection of the risks of a physically active life in a tropical environment.

Aetiology of anaemia

As there is evidence that a proportion of the population at Khok Phanom Di suffered from anaemia at some time in their lives, the aetiology of the condition is of prime importance in determining the effect on their general health. The epidemiology of anaemia in modern Thailand is outlined first in order to define the parameters for discussion of non-skeletal archaeological evidence relating to the presence of anaemia, as the latter is largely circumstantial. The skeletal evidence specific to anaemia has been summarised in chapter V, together with an estimate of the minimum frequency of the condition in the population. The similarity of skeletal response to both acquired and inherited anaemia was mentioned in chapter V. The involvement of the facial bones seen in cases of severe genetic anaemia has not been reported in cases of iron deficiency anaemia but virtually all other skeletal changes are common to sufferers of severe anaemia regardless of the aetiology of the condition. Because of these similarities, a differential diagnosis from the skeleton alone is difficult where the skeletal response is minimal. This section combines the evidence from all sources and attempts to identify the aetiology.

Anaemia is widespread in the tropics (Manson-Bahr and Apted 1982) and is recognised as a major health problem in modern Thailand, where there is an average prevalence of 35-40 per cent (Areekul 1980b). This includes both acquired and inherited anaemia. The major effect of all types of anaemia is to reduce productive capacity through chronic fatigue. The other effects and complications of each type of anaemia are included in the following discussion.

The present-day prevalence of anaemia in Thailand and South East Asia in general offers a variety of suggestions as to the aetiology of the anaemia in the prehistoric Thai. The correlation of modern epidemiology with that in prehistory is not intended in this discussion, as the distribution and prevalence of pathology depends on demographic, social and environmental conditions which are not comparable between the two time-periods. Disregarding the unknown possibility of a deleterious gene which has subsequently disappeared from the population, the modern pattern of causes of anaemia provides a useful limit to the possibilities that need to be considered.

Anaemia in Thailand is most commonly identified as the result of iron deficiency. This can be caused by inadequate intake, inadequate absorption, increased requirement or excessive loss of iron

(Manson-Bahr and Apted 1982) and is often a reflection of a combination of these factors. In Thailand, its prevalence varies among geographic areas and correlates with socio-economic status. It is highest in areas where the diet is most likely to be low in iron and lacking in fish protein. This is often compounded by high rates of intestinal parasitism, resulting from unhygienic living conditions. Hookworm in particular causes excessive blood loss, although the extent to which the parasitism causes anaemia depends on a variety of factors including the worm load, iron intake and reserves and the duration of the infection (Areekul 1980a). When iron intake is barely adequate, the presence of even a light load of hookworms can cause anaemia, once iron reserves are exhausted (Roche and Layrisse 1966, Areekul 1980a). There is also a suggestion that iron deficiency can develop secondarily to iron-withholding as a defence mechanism against infection in the body (Weinberg 1984). This is believed to be stimulated by the competition from the invading organism for serum iron, and is cited as a positive, adaptive response to infection (Stuart-Macadam 1992), although it would be counter-productive for such an adaptation to compromise survival by causing chronic, severe anaemia.

The precise identification of the cause of the anaemia is therefore often difficult, with a combination of nutritional anaemia and parasitism believed to be the most common cause of high levels of anaemia in Thailand in recent times (Areekul 1980b). The effect on the skeleton of iron deficiency anaemia has already been discussed in chapter V. The condition is not fatal but can be very debilitating. It is most common in women of reproductive age and is a major problem during pregnancy when there is increased demand for iron. Iron deficiency during late pregnancy reduces the reserves of the infant in the first year and reduces its ability to cope during periods of infection (Jelliffe 1968).

Malaria is another cause of acquired anaemia and has been a major health problem in Thailand (Harinasuta K. 1986). Control of the insect vector, the Anopheline mosquito, has significantly reduced the scale and distribution of the parasitism since 1949 but prior to this it was the major cause of morbidity and mortality in the country (Ayurakit-Kosol and Griffith 1962). The disease was most prevalent in the hilly areas of northern Thailand but the infection is present throughout the country and localized epidemics were reported in all regions during the period 1935-1960 (Ayurakit-Kosol and Griffith 1962, Harinasuta 1987). There is a lack of clear radiological evidence that anaemia due to malaria produces a skeletal response as has already been mentioned in chapter V. As with hookworm infection, the common complication of multiple pathologies in populations in malarial areas makes precise identification of the primary cause of anaemia difficult (Weatherall *et al.* 1983). McGregor *et al.* (1956, 1966) showed that recurring episodes of *Plasmodium falciparum* malaria were a major cause of chronic anaemia in infants in Gambia, although other pathologies and covert nutritional problems were also involved. They reported bone changes which they ascribed to clinical nutritional problems but these included frontal bossing of the cranium which may have reflected a response to the anaemia. *P. falciparum* produces a virulent form of infection with associated haemolytic anaemia which can be very severe (Weatherall *et al.* 1983). Bone marrow reactions to the anaemia of *Plasmodium vivax* are similar to those of *P. falciparum* (Wickramasinghe *et al.* 1989). At Khok Phanom Di, individuals with evidence of anaemia were spread through all levels of the site. If it is accepted that the anaemia is genetic, as discussed below, this endurance of the genes implies that the population was exposed to malaria throughout the same period.

Inherited haemoglobin disorders

Although not as common in modern Thailand as acquired anaemia, the prevalence of anaemia caused by inherited haemoglobin disorders is nevertheless high. The genetic anaemias are the result of mutations which produce either qualitative or quantitative abnormalities in the structure of the haemoglobin molecule. Qualitative abnormalities involve an alteration in the amino acid sequence of a globin chain. Although there are many of these haemoglobin variants, most are not pathological (Wickramasinghe 1986). The best known of the haemoglobinopathies is haemoglobin (Hb) S which results in sickle-cell anaemia. Quantitative deficiencies, where the rate of synthesis of one of the globin chains is depressed, result in an imbalance in the α - and β -globin chains of the molecule, or their juvenile analogues. The amino acid sequences of the globin chains which are synthesized are usually normal. The diseases resulting from the globin chain imbalance are collectively known as the thalassaemia syndromes (Weatherall and Clegg 1981, Wickramasinghe 1986).

Modern South East Asian populations carry genes for several of the thalassaemia syndromes and haemoglobinopathies. The most prevalent are α -thalassaemia, β -thalassaemia, HbE, and Hb-Constant Spring. HbS (sickle-cell anaemia) is unknown east of India in modern populations.

α -thalassaemia in South East Asia usually results from deletion within the α -globin gene complex, resulting in a variety of interactions. Normal individuals have four α -globin genes. Individuals with the deletion of one gene (α -thalassaemia 2: $\alpha\alpha/\alpha-$) are haematologically indistinguishable from normal and the deletion of two genes (α -thalassaemia 1: $\alpha/\alpha-$ or $\alpha\alpha/-$) produces mild anaemia similar to iron deficiency anaemia (Wasi 1981, Fucharoen and Winichagoon 1987). The deletion of three genes ($\alpha/-$) results in the clinically identifiable HbH disease, so named because of the presence of the abnormal haemoglobin HbH in sufferers (Higgs *et al.* 1989). The clinical expression of the disease is highly variable, although it always involves some degree of anaemia (Weatherall and Clegg 1981). Babies are mildly anaemic at birth and the full clinical picture develops within the first year of life (Wasi *et al.* 1969). The most common effect is lassitude, recorded in 90 per cent of 138 cases by Na-Nakorn *et al.* (1965). They also recorded frequent attacks of fever in over half of 113 patients. There is increased susceptibility to infection and pyrexia associated with acute infection often results in a severe haemolytic crisis, similar to that in G6PD deficiency (Winichagoon *et al.* 1988). Defective development was recorded in 1 per cent of 260 cases reported by Wasi *et al.* (1969) and the facial and post-cranial skeleton was affected in 35 per cent of cases reported by Na-Nakorn *et al.* (1965) and 36 per cent of cases reported by Wasi *et al.* (1969), although according to Winichagoon *et al.* (1988) thalassaemic facies are 'uncommon'. Leg ulcers have also been associated with the disease (Weatherall and Clegg 1981).

The complete absence of α -globin genes ($-/-$) results in a condition known as Hb Bart's hydrops foetalis. This results in death of the infant *in utero* in late pregnancy, or within hours of birth. In the majority of cases the babies are born prematurely, at an average of 34 weeks. Toxaemia of pregnancy is common in the mothers, recorded in up to 50 per cent of Thai women (Wasi *et al.* 1969). Thumasathit *et al.* (1968) reported complications in 21 of 27 Hb Bart's hydrops foetalis pregnancies in Chiang Mai. One-third of the women were toxemic and other problems included polyhydramnios, placenta previa and mispresentations. The prevalence of the more severe

α -thalassaemia syndromes in Thailand is 1 per cent of all conceptions, with half of these dying of Hb Bart's hydrops foetalis and half suffering HbH disease (World Health Organisation 1983).

Recently, advances in molecular biology have resulted in the clarification of the complex genetics of the α -thalassaemia syndromes and permitted identification of population frequencies (Higgs *et al.* 1989). These DNA studies have shown a wide variability in the molecular biology of the gene deletions. The prevalence of the α -thalassaemia syndromes in Thailand recorded from haematological examination show that there is a marked geographical variation. In southern Thailand the frequency of the trait is 16 per cent, in Bangkok it is about 20 per cent and in Northern Thailand it reaches 30 per cent. An exception to the high frequency is in the North East, where it is just over 5 per cent (Fucharoen and Winichagoon 1987). The focus of the highest frequencies is the junction of Northern Thailand with Laos (Wasi 1981). Reports based on DNA mapping suggest that these frequencies may be understated (Fucharoen and Winichagoon 1987). Detailed molecular analyses of the distribution of α -thalassaemia in Thailand have not yet been reported. Frequency estimates in Laos range from 20 per cent of the population (DNA analyses - Anderson and Ranney 1990) to 43 per cent (haematological survey - Wasi 1981). DNA analyses also estimate 20 per cent in Cambodia and 7 per cent in Vietnam (Anderson and Ranney 1990).

β -thalassaemia is also a highly variable group of disorders clinically, although the genetic basis is less complex as there is only one β -globin gene per haploid genome. Nevertheless, DNA analysis has shown that there is a wide variety of mutations responsible for β -thalassaemia (Thein *et al.* 1990). Homozygous β -thalassaemia (originally identified as 'Cooley's anaemia' or thalassaemia major) is the most extreme of the β -thalassaemia syndromes. The disease is usually identified during the first year of life when the normal pattern of replacement of the γ -globin chain by β -chain synthesis does not occur. The production of the β -chain may be reduced or totally absent. The growth of the child is retarded and there is a wide variety of other symptoms including diarrhoea and recurrent fever. The skeletal changes develop and progress with age and there are recurrent infections with a wide variety of complications including leg ulcers. There is cardiac and endocrine dysfunction and growth continues to be retarded (Weatherall and Clegg 1981). The condition is treated today with blood transfusions but without this treatment mean life expectancy is five years and rarely lasts beyond the second decade (Weatherall and Clegg 1981, World Health Organisation 1983). From this extreme form of the disease, the range of clinical expression is highly variable, with the other extreme in heterozygous β -thalassaemia. This is usually asymptomatic or associated with a mild degree of anaemia. The only major complication is worsening of the anaemia during pregnancy, sometimes to a degree requiring blood transfusion (Weatherall and Clegg 1981).

The prevalence of β -thalassaemia in Thailand, based on haematological studies, ranges from c. 2 per cent in Bangkok and some areas of North East Thailand to almost 8 per cent in some villages in Northern Thailand. The national prevalence is 4.8 per cent. There are relatively high frequencies in some minority ethnic groups, ranging up to almost 10 per cent in some hill tribes who immigrated from southern China in the twentieth century (Flatz *et al.* 1965, Fucharoen and Winichagoon 1987). In other countries of South East Asia, the frequencies range from 1 per cent to up to 25 per cent in some minority ethnic groups in Vietnam. It affects 9 per cent of the population in Laos (Fucharoen and Winichagoon 1987).

A study of the molecular biology of the gene in Thailand shows that the four most common mutations in Thai β -thalassaemics are also the most common mutations in South China (Thein *et al.* 1990). Given the distribution within Thailand of β -thalassaemia homozygosity, it appears most likely that the mutation was brought into Thailand with the immigrations of the Thai people from Southern China in the first millennium AD. The Hb E variant arises from a mutation on the β -globin chain. HbE heterozygotes are asymptomatic and homozygotes commonly so (Wasi 1981, Weatherall and Clegg 1981), although recent DNA analyses have found that some homozygotes have severe anaemia (Fucharoen *et al.* 1990). The prevalence of the more serious form is not known, as some may have been misdiagnosed as compound heterozygotes of HbE/ β -thalassaemia in the past (Fucharoen *et al.* 1990). In Thailand there is a high prevalence of double heterozygotes for HbE/ β -thalassaemia. The interaction of the two conditions produces a highly variable clinical picture but can be as severe as homozygous β -thalassaemia, with similar growth retardation, skeletal responses and increased susceptibility to infection. Childhood mortality is high, with only a small proportion of individuals surviving into adulthood (Weatherall and Clegg 1981). Mean life expectancy is 20 years (World Health Organisation 1983). α -thalassaemia/HbE produces a similar clinical picture to that of HbH disease (Wasi *et al.* 1969).

The distribution of HbE is almost entirely limited to South East Asia. It is particularly prevalent in the area around the junction of Thailand, Laos and Cambodia and is largely confined to "...groups such as the Khmer, Laos, Mon-Khmer speaking people and the aborigines of Malaysia." (Fucharoen and Winichagoon 1987:81). In North East Thailand it is present in up to 50 per cent of the general population. In minority ethnic groups it reaches 43 per cent (Poothai), 54 per cent (Khmer) and 74 per cent (So). In provinces of Laos it ranges from 25–50 per cent, in Cambodia from 30–60 per cent and in Vietnam from 1–42 per cent, except for the minority Mon-Khmer Stieng, where it reaches 73 per cent. In Malaysian aboriginal groups it ranges from 20–50 per cent. Frequencies are lower in North and South Thailand than in the North East. It is not present in significant frequencies in the Chinese (Flatz *et al.* 1965). The distribution in Thailand is reciprocal to that of β -thalassaemia (Flatz *et al.* 1965, Fucharoen and Winichagoon 1987). DNA analysis has shown that the mutation for HbE has arisen at least twice in South East Asia, with one mutation restricted to Cambodians (Antonarakis *et al.* 1982).

The frequency and heterogeneity of the thalassaemia syndromes present in South East Asia results in a complexity of gene interactions with individuals with up to four abnormal genes. More than 60 genotypes had been identified in South East Asia by the early 1980s (Wasi 1981). Table 117 lists an estimate of the percentage of cases of each of the major diseases in 1969 (adapted from Wasi *et al.* 1969:79). In addition to these major haemoglobinopathies, there are a number of other less common haemoglobin variants present in South East Asia. These can interact with the milder α - and β -thalassaemias to intensify the clinical condition. The most common in Thailand is Hb Constant Spring (Fucharoen and Winichagoon 1987).

At least 40 per cent of the population carries one of the thalassaemia traits, in 64 per cent of marriages one or both partners carries a thalassaemia gene, and altogether 5.6 per cent of couples risk producing a homozygote for a major haemoglobin disorder (World Health Organization 1983). These proportions cover the entire population of Thailand and must vary significantly among the

minority ethnic groups. Similar information is not available for the neighbouring countries of Laos and Cambodia.

There are several inherited defects of the enzymes involved in the metabolism of glucose via the hexose monophosphate shunt and in the metabolism of glutathione (Dacie 1985). The most common is deficiency of glucose-6-phosphate dehydrogenase (G6PD). It is a cause of anaemia in several forms, including in response to certain oxidant drugs or infections (Dacie 1985:364). The gene is X-linked and the defect is fully expressed only in homozygous females and hemizygous males. A survey of a large sample of adult and neonate males ($n=4368$) in Thailand in the early 1960s found an average of 12 per cent were G6PD deficient (Flatz and Sringam 1963). It affects less than 5 per cent of recent Chinese immigrants to Thailand and most Tibeto-Burmese ethnic groups in Northern Thailand but ranges from 10–30 per cent in other groups in Northern Thailand and in Mon and Khmer (Flatz and Sringam 1964). The disorder is biochemically and probably genetically heterogenous. Six different variants were identified among 50 individuals in Thailand alone (Panich and Sungnate 1973, cited in Dacie 1985:371). G6PD deficiency can cause neonatal jaundice (Dacie 1985). Severe neonatal jaundice was found in 5 per cent of G6PD deficient neonates in a study in Bangkok (Flatz *et al.* 1963). Appearance of the condition in the absence of drug administration appears to have a familial association (Dacie 1985). Chronic non-spherocytic congenital haemolytic anaemia can also be caused by G6PD deficiency. It is not common but there is one case reported in a Thai boy (Talalak and Beutler 1969).

TABLE 117: *Estimated proportions of patients in Thailand affected by each of the more common thalassaemic diseases*

Condition	Proportion of patients (%)
HbH disease (α -thalassaemia)	47
Hb Bart's hydrops foetalis (α -thalassaemia)	25
HbE/ β -thalassaemia	15
Homozygous β -thalassaemia	6
HbH/HbE	7
	100

Iron deficiency anaemia commonly occurs with anaemia secondary to malaria in Thailand, as already mentioned. There is yet another potential complication with the combination of iron deficiency anaemia with inherited haemoglobinopathies. The co-occurrence of iron deficiency anaemia with carriers of thalassaemia or one of the haemoglobinopathies was recorded in a small sample of patients with hypochromic microcytic anaemia in Bangkok by Patmasirawat *et al.* (1985). In 73 patients admitted with this condition, 60 had iron deficiency anaemia and more than one third of these were also heterozygous for thalassaemia or one of the other haemoglobinopathies. The combination of β -thalassaemia trait with iron deficiency significantly increased the degree of anaemia compared with individuals with β -thalassaemia trait only. The diagnoses were made haematologically.

A recent study of the aetiology of anaemia among island populations of Vanuatu in the South West Pacific (Bowden *et al.* 1985) has produced results which are of interest. High levels of anaemia had been attributed to iron deficiency in the past, mainly because of the absence of any overt indication of the presence of inherited haemoglobin disorders. DNA analysis showed unexpectedly that in the majority of cases, anaemia was the result of single-gene-deletion α -thalassaemia. The implication of this is that large-scale DNA analysis of South East Asian populations have the potential to show a similar picture. Where anaemia is attributed to iron deficiency by default rather than on the basis of studies of iron status, the diagnosis may be incorrect.

Non-skeletal evidence for the aetiology of the anaemia

The evidence for the possibility of iron deficiency anaemia from nutritional inadequacy depends on the analysis of the diet at the beginning of this chapter. There is no strong reason to suggest that the diet would have been inadequate in iron. Despite the iron absorption-inhibiting phytates in rice, the quantities of fish and shellfish consumed should have been sufficient to ensure that iron intake was adequate to overcome the problem.

The second recognised cause of iron deficiency anaemia in modern Thailand, hookworm infestation, leaves no archaeological evidence other than eggs in coprolites. There were coprolites recovered from the excavation at Khok Phanom Di but apart from that recovered with B67, these were probably canine (Wharton, pers. comm.). As the infection is specific to humans, there are no other hosts or vectors to be taken into consideration. The parasites are transmitted through contamination of soil with faeces containing eggs. In the soil, the eggs hatch into larvae which can penetrate the skin, so infection is easy where hygiene is poor and people are barefooted (Harinasuta 1983). The larvae survive only in damp conditions and aerated soil, so vegetation cover and abundant, frequent rainfall is ideal (Harinasuta 1962). The maintenance of infection in the community, therefore, requires an adequate source of infection in the human population and the combination of suitable environmental conditions with inadequate sanitation (Manson-Bahr and Apted 1982). The mound is likely to have had at least a surface layer of uncompacted soil, although it is not sandy (Aitken, pers. comm.). The vegetation cover on the mound itself at the time of prehistoric occupation is unknown but even the present vegetation, which is planted or weedy regrowth (Maloney 1991), would have been sufficient, at least at the edges of the mound, to have provided favoured sites for defecation and to have maintained moist soil conditions. However, the seasonal drying of the soils on the mound may have been sufficient to have prevented survival of the larvae. Desiccation, flooding and salt water are fatal to the larvae (Manson-Bahr and Apted 1982), so the brackish water in the river estuary and the seasonal flooding and drying of low-lying areas cleared of vegetation around the mound would not have been a suitable environment for their survival. The usual pathogenic life span of the worms is probably measured in months in the absence of re-infection (Stephenson 1987). Periodic infection or re-infection of individuals may have occurred during visits to infected areas but the maintenance of the infection in the population at Khok Phanom Di is doubtful.

The possibility that iron deficiency anaemia resulting from a response to chronic infection by micro-organisms affected the people at Khok Phanom Di depends on the availability of pathogens. Any infection which has a short lifespan and confers lasting immunity will not survive in a small community, as the reservoir of susceptible individuals will soon be eliminated either by death or

through the development of immunity (Black 1980). These include most of the viral and many of the bacterial infections common today. Community size is not such an important factor in those infections which confer only partial immunity or have alternative hosts other than humans. Long-term survival is enabled by the supply of children as susceptible individuals and the infection remains endemic within the population. Nutritional standards, personal and community hygiene, access to clean water, type of housing, attitudes to illness, association with animals and environmental conditions all influence the exposure and response of individuals to infection. Nevertheless, there is some possibility that an unidentified chronic infection could have been an indirect cause of anaemia at Khok Phanom Di.

The determination of the possibility of malaria being a cause of anaemia at Khok Phanom Di depends on indirect evidence, both for the presence of the parasite in South East Asia at the time of the occupation of the site and for the local environmental and host conditions being suitable for the presence and survival of the infection in the community. There are four species of the *Plasmodium* parasite which infect humans and two of these, *P. falciparum* and *P. vivax*, are present in South East Asia today. The initial infection of humans with the parasites is believed to have resulted from infection with a simian malaria, as there are similarities between species affecting non-human primates and those affecting humans (Bruce-Chwatt 1965). The only species of non-human primate malaria with the same clinical characteristics as *P. falciparum* is *P. reichenowi*, which is found only in Equatorial Africa. As this is the only possible source of *P. falciparum*, the original infection of humans was in Africa (Fiennes 1978). The source of *P. vivax* is less clear as there is a variety of simian Plasmodiae which are similar and some of these have accidentally or experimentally infected humans. Included amongst these are *P. cynomolgi* and *P. knowlesi* which infect *Macaca* sp. and *Presbytis* sp. in Malaysia, raising the possibility that the initial infection *P. vivax* of humans was in South East Asia (Fiennes 1978, Steele 1982). The relative virulence of the infection from *P. falciparum* compared with that of *P. vivax* suggests that the latter is an older parasite of humans, as the host-parasite relationship has begun to stabilise. There is reference in historical documents to fevers associated with enlarged spleens in India from about 1500 BC and in China from 2700 BC, which is believed to indicate that the malarial parasites were present in those countries (Bruce-Chwatt 1965). There is a case for the presence of *P. vivax* at least in South East Asia at the time of the occupation of Khok Phanom Di.

The infection of the population at Khok Phanom Di would have depended on the presence of a suitable vector. Although the highest rates of infection in modern Thailand are transmitted by vectors which favour the conditions in hilly areas, there are secondary vectors which are responsible for local areas of infection in lowland and coastal areas of wider South East Asia. The species most likely to have been present at Khok Phanom Di are *Anopheles aconitus* and *A. sundaicus*, as the equable climate of the coastal areas of South East Asia allows these otherwise 'feeble' carriers to maintain transmission of the parasite throughout the year (Russell *et al.* 1963:450). On the basis of circumstantial evidence, *A. aconitus* is believed to have been responsible for low levels of malaria in rural areas to the north of Bangkok in the 1960s (Gould *et al.* 1967, Pinichpongse and Ratanaritkul 1968). This species breeds on exposed sheets of water such as rice fields, ponds or swamps, or in stream or river-beds (Russell *et al.* 1963, Bruce-Chwatt 1980). *A. sundaicus* is a coastal vector which breeds in brackish water, particularly lagoons or swamps behind coastal embankments

at a water level between high neap tide and spring tide (Russell *et al.* 1963, Bruce-Chwatt 1980). A survey in Central Thailand in 1931 found no malaria in people living at the delta of the Chao Phraya River, but in a coastal area of Southern Thailand there was evidence of a high rate of infection where *A. sundaicus* was breeding in the lower sections of a stream fringed with Nigah (*sic* = nypa?) palm (Anigstein 1932). In this very limited survey the rate of infection appears to have varied dramatically over short distances, according to local conditions. *A. sundaicus* tends to have a patchy distribution but breeds in large numbers and has a wide flight range so despite having a low level of infectivity it can be an important, if sporadic, vector (Reid 1968). The environment around Khok Phanom Di would have been ideal for the breeding of *A. sundaicus*, in the coastal marshes and the swamps among the nypa palms on the lower reaches of the Bang Pakong and it is possible that *A. aconitus* was breeding inland in the river, in tributary streams or freshwater swamps.

The third question in the maintenance of the infection is whether host conditions were favourable to the parasite. In areas of endemic malaria with year round transmission, the host population builds up a high degree of immunity but at the same time enables the parasite to survive in small numbers (Bruce-Chwatt 1980). This is termed stable malaria. Infants in these populations acquire a passive immunity from their mothers. This protects them for the first few months of life but they then become susceptible to infection from the reservoir of parasites in the community. This is the most critical period in the host-parasite relationship and the child may suffer successive attacks which can prove fatal, either through the malarial infection or reduced ability to survive secondary infections. Children who survive this period will have built up a residual immunity by the age of about five years (Bruce-Chwatt 1980). Where malaria is unstable, with cyclical epidemics dependent on climate or vector characteristics, the degree of immunity will be dependent on the frequency and intensity of the epidemic (Russell *et al.* 1963). Morbidity and mortality will affect individuals of all ages during epidemics. As well as acquired immunity, the human host can also have genetic resistance to malaria. This is discussed below.

The coastal areas of the Gulf of Thailand are included in the Malaysian Zone of malarial epidemiology (Russell *et al.* 1963). Before control measures were implemented, the zone was characterised by unstable malaria, with many fluctuations of the disease, frequent evanescent malaria, the presence of uninfected Anophelenes and common epidemics (Russell *et al.* 1963). Both *A. sundaicus* and *A. aconitus* are characteristic vectors of unstable malaria and *P. vivax* is considered the characteristic parasite, because its life cycle results in the faster development of epidemics. Despite this, the coastal area of Rayong Province in South East Thailand was described as an area of high malarial endemicity in 1960, although the species of parasite was not identified (Kruatrachue *et al.* 1961). Both *P. falciparum* and *P. vivax* malaria are now present in Thailand, with *P. falciparum* twice as prevalent as *P. vivax* in most areas (Ayurakit-Kosol and Griffith 1962, Tansathit *et al.* 1962). One survey (Kruatrachue *et al.* 1962) recorded the parasites in equal frequencies in areas to the north and south of Khok Phanom Di and Gould *et al.* (1967) reported 90–100 per cent of malarial infections in communities on the Chao Phraya River north of Bangkok in Central Thailand to be *P. vivax*.

The human, parasite and vector factors necessary to the presence and maintenance of malaria appear to have been favourable at Khok Phanom Di, although the species of parasite and the epidemiology of the infection cannot be established.

Apart from the present distribution of the haemoglobinopathies and G6PD deficiency in South East Asia, the non-skeletal evidence that these conditions may have existed in the population of Khok Phanom Di is dependent on the presence of malaria in the population.

Genetic disorders of the red cell, including the thalassaemias and haemoglobin variants, are among the most common genetic disorders in the world population (Weatherall 1987). Given the deleterious effects of most of the homozygous states of these disorders, the survival of the genes in high frequencies in some areas presents an apparent anomaly. The explanation for this was suggested by the worldwide similarity in the distribution of the genes with the distribution of *P. falciparum* malaria. This coincidence was first recognized in the late 1940s and stimulated the hypothesis that the carriers of the genes have increased immunity to the infection (Haldane 1949, cited in Livingstone 1967). The suggestion is that the prevalence of the haemoglobin disorders reflects a state of balanced polymorphism, where the frequency of the gene has increased to a point where the numbers of disadvantaged homozygotes are balanced by the numbers of protected heterozygotes (Weatherall 1987).

Many attempts have been made to test the malaria hypothesis for all the haemoglobinopathies. The relative fitness of HbS (sickle-cell) carriers in malarial areas in Africa has been well documented in epidemiological studies. Cellular mechanisms which could explain the protection that the sickle-cell gene gives carriers have been demonstrated (Weatherall 1987). The evidence for the relative advantage of carriers of the thalassaemia and HbE genes in malarial areas is not as conclusive as that for HbS. The relative protection of β -thalassaemia heterozygotes has been documented in Africa (Willcox *et al.* 1983) but evidence from other parts of the world is not as conclusive (Weatherall 1987). *In vitro* experiments have not yet succeeded in providing a universally accepted explanation of the cellular mechanism by which growth of the parasite is impaired in β -thalassaemia carriers, although several possible explanations have been mooted (Brockelman *et al.* 1987, Thanomsu *et al.* 1989). Population studies of α -thalassaemia have been difficult in the past because of the complexity of the genetics and the difficulty in identifying carriers haematologically. The advent of DNA analysis has allowed identification of carriers of the α -thalassaemia trait.

The frequency of carriers identified through DNA in Melanesia and Nepal has been shown to be highly correlated with malaria endemicity, suggesting selection for the gene (Flint *et al.* 1986). As with β -thalassaemia, no explanation for the cellular mechanism of the protection of carriers has been universally accepted despite numerous experiments (Friedman 1979, Brockelman *et al.* 1987, Yuthavong *et al.* 1988, Thanomsu *et al.* 1989, Luzzi *et al.* 1990). *In vitro* experiments in Africa have shown inhibition of growth of *P. falciparum* in the red cells of HbH sufferers but not in trait carriers (Ifediba *et al.* 1985). The situation is similar with HbE, with a geographical correlation of trait carriers with high levels of malaria in Thailand (Flatz *et al.* 1964) but no obvious explanation for the mechanism involved (Santiyanont and Wilairat 1981, Thanomsu *et al.* 1989, Nagel 1990). There may be no single explanation for the protection of carriers of the thalassaemia or HbE genes but a variety of mechanisms operating. Most of the experiments have concentrated solely on *P. falciparum*, although Modiano *et al.* (1991) found that heterozygous α -thalassaemics were also relatively protected against *P. vivax* and Ray *et al.* (1964) found that HbE thalassaemics were found to have significant resistance to *P. vivax* infection.

As with the haemoglobinopathies, the geographical distribution of G6PD deficiency correlates with the distribution of malaria prior to control world-wide (Dacie 1985). Flatz and Sringam (1963) demonstrated this correlation in Thailand. This suggests that the deficiency provides a selective advantage against malaria. The mechanisms involved have been demonstrated in some cases but the type and degree of resistance to malaria appears to vary among different genotypes (Dacie 1985).

The diet of the people at Khok Phanom Di and the poor chance of survival of hookworm on the site or environs suggest that iron deficiency is unlikely to have been the major cause of the anaemia in the population. Females are more frequently affected by iron deficiency anaemia than males and as similar proportions of both sexes have evidence of anaemia at Khok Phanom Di, this also suggests that iron deficiency is an unlikely aetiology. There is a stronger case for the presence of malarial parasites at Khok Phanom Di, however, and it is most likely that malaria contributed to the anaemia. Following from this is the possibility that one or more of the genetic blood disorders was also present at Khok Phanom Di and was responsible for the more severe cases of anaemia discussed at the end of the chapter V.

As this has implications for the long-term adaptation of the people and genetic relationships of the population, the possibility is considered in some detail. Skeletal evidence for the possibility includes the infants with hypertrophy of the facial bones. The hypertrophy of the maxilla and sequelae "...almost certainly establish the diagnosis of thalassemia major." (Moseley 1971:698). Although the hypertrophy of the facial bones has not been definitively identified in any older individuals, this could reflect the severity of the disease which produces this response. It is likely that individuals with such a degree of illness would not have survived beyond infancy in prehistory. The evidence of eight individuals with specific skeletal responses (detailed in chapter V) is in favour of genetic haemoglobinopathy. These skeletal responses have been identified in modern case studies only in individuals with severe anaemia due to one or more of the haemoglobinopathies.

Against the possibility of the anaemia having a genetic origin is the apparent virtual absence of unequivocal evidence of the 'classic' lesion of porotic hyperostosis of the cranial vault associated with severe anaemia. However, as with the hypertrophy of the facial bones, this may reflect the inability in prehistory of many individuals with such severe anaemia to survive beyond infancy. A further possibility suggested by Lanzkowsky (1977, cited in Hart 1980), is that a cranial response is likely to develop only in association with premature birth, protein malnutrition, or nutritional rickets which increase skeletal plasticity. Infants born prematurely in prehistory would have had a low chance of survival and dietary evidence from Khok Phanom Di implies that there were unlikely to be deficiencies in either protein or vitamin D. The extreme variability of the skeletal response to the disease emphasised by clinicians (Weatherall and Clegg 1981) is also to be considered. The degree of skeletal response does not necessarily correlate with the severity of the underlying genetic disorder.

It can be concluded that one or more of the haemoglobinopathies was present at Khok Phanom Di and was the cause of the most severe cases of anaemia. Along with malaria, it may also have been the cause of some of the mild cases of anaemia. The wide range of haematological and molecular studies in Thailand and South East Asia have shown that there are differing gene frequencies among geographic areas and ethnic groups. The distribution of the genes has been discussed above but is summarised briefly to illustrate the point.

Homozygous β -thalassaemia is concentrated in the north of Thailand and in recent Chinese immigrants to Thailand. The frequency is low in Khmer speakers, Mon speakers and in North eastern Thailand generally. HbE is high in the north east of Thailand, in Cambodia and in Laos. It has a reciprocal distribution with β -thalassaemia and is not common in the Chinese, or in northern or southern Thailand. α -thalassaemia is particularly frequent in an area focussing on northern Thailand and Laos and is also relatively frequent in Central and southern Thailand but not in the north east. The distribution of this syndrome is less well-known because of the complexity of the genetics and the difficulty in diagnosing carriers without DNA analysis. Deficiency of G6PD is high in the north and north east of Thailand and in the Khmer and Mon peoples.

Based on the distribution of the haemoglobinopathy and G6PD genes, it is possible that the genes present at Khok Phanom Di could have included any of those identified in modern Thailand with the probable exception of the β -thalassaemia gene. This leaves one or more of α -thalassaemia, HbE and G6PD deficiency as the possible genetic explanation or explanations for the anaemia at Khok Phanom Di. On the basis of modern clinical evidence α -thalassaemia in the form of HbH disease appears the most likely cause of the severe anaemia in infants and children, although the severe form of homozygous HbE identified by Fucharoen *et al.* (1990) suggests that HbE is also a possible cause of the condition in this age group. The higher frequency of this gene in modern Thailand and its relatively mild effect in almost all cases both argue for a longer history. The gene may have evolved through natural selection over the last four thousand years and it is possible that the relationship between the genetic condition and malaria was not as well-balanced at the time of occupation of Khok Phanom Di as it is believed to be today. G6PD deficiency is unlikely to have produced anaemia of the same severity. At first glance, it is tempting to suggest that homozygous α -thalassaemia (Hb Bart's hydrops foetalis) is an obvious explanation for the high level of neonate deaths. This is not as acceptable as it appears, for reasons which are discussed later in this chapter. Beyond this, it is not possible to identify precisely whether α -thalassaemia or HbE genes were present at Khok Phanom Di.

Other pathology

Apart from malaria, many of the pathogens affecting the people in modern Thailand are not relevant to the consideration of the epidemiology of disease in the prehistoric population. The small, comparatively isolated prehistoric group at Khok Phanom Di would not have been exposed to many of the infectious diseases affecting the modern Thai, as they would not have existed in such an environment. An extrapolation from the epidemiology of disease in modern Thailand to that of the prehistoric population at Khok Phanom Di is not intended, but selective consideration of the diseases affecting the modern population is useful in determining the possibility of such infections as helminthic parasites. Parasitism by protozoa and helminths is the major public health problem today (Sucharit *et al.* 1986). Malaria is the most important disease and anaemia and diarrhoea are among the most obvious effects of parasitism, although these often have multiple aetiologies.

The most important of the modern parasites in South East Asia is the protozoa *Plasmodium* sp. which causes malaria. Others include *Entamoeba histolytica*, which causes amoebiasis and *Giardia lamblia*, which causes giardiasis. Both are transmitted from person to person, without an intermediate host or vector. Amoebiasis is associated with poor sanitation, poor nutrition and crowded

living conditions. The infection is asymptomatic in many cases but can cause dysentery and liver abscesses. It does not stimulate immunity in infected individuals (Manson-Bahr and Apted 1982), and can survive in endemic form in isolated groups (Black 1980:48). There is a prevalence of 10–20 per cent in low socio-economic rural Thailand (Sucharit 1986). Giardiasis is endemic in many tropical areas. Infection rates are high in children, peaking at the age of 11. About 20 per cent of infected individuals suffer acute diarrhoea and in 10 per cent this can become chronic. In most cases, the infection is spontaneously eradicated (Manson-Bahr and Apted 1982).

A variety of nematodes are endemic in Thailand, some in restricted areas. For example, the mosquito-transmitted nematodes which cause filariasis are present only in certain areas of the south and the west (Harinasuta C. 1986). Others are more widespread but with local areas of higher prevalence. Infection with hookworm, mainly *Nestor americanus* but also *Ancylostoma duodenale*, is common in South East Asia generally (Harinasuta 1983). In a nationwide survey in the early 1950s, 21 per cent of Thai were found to be infected with hookworm (Setasuban 1986). It was present throughout the country, although the highest prevalence was in the south (Harinasuta 1962). In Central Thailand, the prevalence was 11 per cent (Harinasuta and Vajrasthira 1964). As the most debilitating effect of infection is anaemia, hookworm has been discussed in more detail in the section on anaemia earlier in this chapter. Other nematodes which infect humans include *Gnathostoma spinigerum*. Humans are probably accidental hosts for this parasite which infects many species of vertebrates. It has two intermediate hosts, the snail *Cyclops* and certain species of fish, frogs or snakes. Humans can become infected by eating raw or inadequately cooked fish (Manson-Bahr and Apted 1982). The principal effects are initially internal pain and later chronic migrating subcutaneous oedema. It can cause more serious illness. Angiostrongyliasis (eosinophilic meningitis), infection with the rat lungworm *Angiostrongylus cantonensis*, is usually transmitted to humans in Thailand through the consumption of improperly cooked *Pila* sp. snails. A higher prevalence in males than females reflects eating habits. The most common symptoms are severe headache and neck stiffness. It is rarely fatal. Ascariasis and trichuriasis are infections from soil-transmitted helminths. Mild infestations are often asymptomatic but heavy infestations, particularly in individuals in poor health, can cause diarrhoea and a variety of other symptoms. They are both present in Thailand, although the distribution and frequency are not known.

Of a variety of trematodes present in Thailand, some are endemic in areas in the north and north east. *Opisthorchis viverrina* (lung fluke) is transmitted to humans through the habit of eating raw fish. *Schistosoma mekongi*, which causes a mild form of schistosomiasis, is endemic only in the valley of the Mekong River. Trematodes endemic in other areas in Thailand include the giant intestinal fluke, *Fasciolopsis buski*, which is endemic to a restricted area on the central plain north west of Bangkok. Despite the large size of the fluke, infections are usually asymptomatic unless large numbers are present. Heavy infections can cause toxic diarrhoea and oedema. Longer term symptoms are the result of malabsorption, causing slight anaemia, growth impairment and emaciation (Daengsvang and Mangalasmaya 1941). The infection can be fatal.

Direct non-skeletal archaeological information which is useful in establishing the pathogens affecting the people at Khok Phanom Di is effectively limited to evidence for intestinal parasites in the form of an egg in a coprolite. The presence of the remains of known vectors of intestinal parasites in the site is indirect evidence for the potential for parasites in the population.

The coprolite found with B67 contained a single parasite egg. This has been identified as a trematode, possibly *Fasciolopsis buski* (Morseth 1991) and this suggestion is strengthened by the presence of the shells of one snail host, *Segmentina cf trochoideus*, at Khok Phanom Di (Mason 1991). *F. buski* is a parasite of pigs, which were present throughout the occupation of Khok Phanom Di. The infection of humans is the result of the ingestion of plants which carry the metacercariae and the distribution of the parasite is limited to the areas where these plants grow. They are the water caltrops (*Trapa bicornis*), lotus (*Nymphaea lotus*), water cress (*Neptunia oleracea*) and water morning glory (*Ipomea aquatica*). There were no parasite eggs found in the other coprolites from Khok Phanom Di (Wharton, pers. comm), although most, if not all, are thought to come from dogs.

Other molluscs found at Khok Phanom Di which are known intermediate hosts or vectors of one or more helminths are single shells or fragments of *Pila pesmei*, a potential intermediate host of the nematode *Angiostrongylus cantonensis*, and *Filopaludina sumatrensis*, which can carry any of four *Echinostoma* sp., a trematode of minor importance as an intestinal parasite. *A. cantonensis* is a parasite of rats of the genus *Rattus*, which were common at Khok Phanom Di and *Echinostoma* are parasites of rats, ducks or dogs. The parasites can be ingested by humans through the consumption of raw or partly cooked snails (Mason 1991).

Fish, which are second intermediate hosts of the nematode *Gnathostoma spinigerum*, are abundantly represented at Khok Phanom Di. These are the freshwater species *Clarias batrachus* and *Ophicephalus striatus* (Kijngam 1991).

The possibility of the maintenance of malaria and the case against hookworm infection at Khok Phanom Di has already been discussed in connection with anaemia. Of the other diseases caused by protozoan and helminthic parasites, there are multiple sources of evidence for the potential presence of fasciolopsiasis in the population. Other helminthic diseases including gnathostomiasis and angiostrongyliasis were potentially present. Whether diseases caused by parasites without hosts or vectors other than humans were present at Khok Phanom Di can only be a matter of conjecture, but the possibility cannot be excluded.

Skeletal remodelling and degeneration

No documented ethnographic data on the epidemiology of clinical degenerative joint disease in modern South East Asia has been located. The climate may be an explanation for this, as it has been shown that there is an association between the climate and the pain and disability of degenerative joint disease. Despite similarities in the proportion of individuals with radiographically diagnosed moderate-severe joint degeneration, the proportion with symptoms is lower in tropical than in temperate climates (Lawrence 1977). In view of this, the lack of data from tropical countries where medicine is occupied with the more pressing problems of high levels of morbidity and mortality from a multitude of infectious and parasitic diseases is easily understood.

The evidence that joint degeneration at Khok Phanom Di is osteoarthritis rather than any other form of the disease is based on the pattern of the joints involved. It is difficult to distinguish osteoarthritis from rheumatoid arthritis on the basis of the bony remodelling alone (Steinbock 1976). Rheumatoid arthritis affects more females than males, is polyarticular, affects joints symmetrically and does not involve eburnation in extreme cases as the affected joint is immobilised and ultimately ankylosed (Steinbock 1976, Cockburn *et al.* 1979). It is less common in the tropics than in temper-

ate climates. The higher frequency of joint degeneration in males than in females at Khok Phanom Di, the presence of eburnation in some of the more severely affected joints and the absence of ankylosis suggests that rheumatoid arthritis is unlikely to be prevalent at Khok Phanom Di. None of the joints has the extreme reactive bone formation and remodelling that is associated with the advanced stages of septic arthritis. In one individual, B18, there is marked asymmetry in the degree of degeneration in the knee joints but there are no grounds for proposing differing aetiologies.

Five individuals with degeneration in the lumbar spine have spondylolysis. This is a separation of the neural arch, usually bilaterally, from the vertebra. It involves both genetic and mechanical factors, with biomechanical stress acting on an inherited predisposition (Bridges 1989). The separation of the arch can occur at an early age, when a child first starts walking, or in later life with repetitive biomechanical stress from vigorous physical activity involving flexion, extension or rotation of the spine. It is most common in the fifth lumbar vertebra, with decreasing prevalence in the higher lumbar spine. In many cases it is asymptomatic, with the arch held in place by the restraining action of muscles and ligaments, but it can result in the separation of the arch and the body. This condition (spondylolisthesis) can alter the articulation of the apophyseal joints.

Three adults (Bs 28, 87, 152) from Khok Phanom Di with spondylolysis had moderate-severe degeneration of discs in the lower lumbar vertebrae but otherwise minimal joint degeneration. The degeneration may have been exacerbated by alterations in the geometry of the joints as a consequence of the separation of the neural arch, although, as noted in chapter V, spinal degeneration was generally apparent earliest and most frequently in the lumbar spine. Of the two other adults with spondylolysis of the fifth lumbar vertebra, one (B93), had a pattern of lumbar degeneration which suggests spondylolisthesis. This included phytosis and porosity of the anterior bodies of the lumbar vertebrae and the sacrum, posterior compression of the fifth vertebral body and articulation between the spinous processes of the fourth and fifth vertebrae. These cases of spondylolysis are the only instances of joint degeneration in which an aetiology other than primary osteoarthritis or incipient osteoarthritis is proposed.

Joint degeneration at Khok Phanom Di is unlikely to be attributable solely to aging changes, because of the relatively early mean age at death compared with modern populations. Biomechanical stress is often accepted as the predominant aetiology of degenerative joint disease in prehistoric populations where life expectancies were short. Despite the potentially complex aetiology of joint degeneration, this is the most likely explanation for the patterns found at Khok Phanom Di. This would mean that the Khok Phanom Di people were living a physically active life from an early age. Both sexes were engaging in activities which placed considerable stress on the lower spine, the joints of the shoulders and hands in the upper limb and all lower limb joints except the hips.

Apart from in the lower vertebrae, there were sex differences in spinal degeneration. These suggest that males were involved in activities which placed greater stress on their spines than experienced by the females. Males had more vertebral degeneration from an early age than females and they had a higher prevalence of joint degeneration in the upper spine at all ages. The activity appears to have exerted stress on the spine which affected both the intervertebral discs and the vertebrae themselves. It also involved the ribs, producing degeneration on the costo-vertebral joints, and the entire shoulder girdle with consequent degeneration of the shoulder joints and sternal articulations. Female activities appear to have exerted similar levels and patterns of stress on the major

limb joints as in males but the pattern of spinal degeneration suggests that their activities placed particular stress on their lower backs.

The overall greater frequency of degeneration in younger males than in younger females is common in many modern populations before the age of 45 (Masi and Medsger 1989). Although no explanation for this has been conclusively demonstrated, greater physical occupational stress and minor trauma (Lawrence 1977, Moskowitz 1989) and greater physical size (breadth and girth) with associated greater mass (Masi and Medsger 1989) are suggested as contributing factors. The behaviour which results in bony remodelling and ultimately degeneration is not necessarily normal daily activity but actions which place either abnormal stresses or normal stresses for abnormal lengths of time on joints and muscles of the limbs or spine (Merbs 1983). The sex differences in the patterns of degeneration in older individuals at Khok Phanom Di suggest the sexual division of labour.

The correlation between specific activities and joint degeneration is not possible. It is not appropriate to embark on a detailed consideration of the evidence from the Khok Phanom Di people in this study but some speculation as to possible explanations for broader patterns is offered. Skeletal remodelling of sites of origin and insertion of muscles and ligaments have not been systematically recorded as they do not fit within the prime objective of this study. Nevertheless, it was observed that patterns in many of the males of the development of the upper arm and shoulder girdle suggest movements involving vigorous lifting of the arms (abduction and flexion), turning and bending of the back (rotation, flexion) and stretching of the arms across the body (medial rotation). Both arms are involved. Together with degeneration of the spine and upper body, this pattern is consistent with paddling a canoe or small boat, although this is only one of a many possible explanations. In the females, the degeneration of the discs in the lumbar spine suggests bending, perhaps with lifting. Tending rice crops and gathering shellfish and plant foods are two possible explanations. Again, this is not offered as evidence for these activities.

As with males, subjective observations were made of remodelling of the muscle and ligament attachments in the upper arms and hands. These followed a different pattern from that in males and suggests that females were using their arms to exert vigorous pressure with the hands, perhaps in the manufacture of pots. Particular patterns of remodelling of the muscle and ligament attachments were not observed in the lower limb, but the degeneration of the joints in both sexes at Khok Phanom Di indicates that the lower limbs were exercised as vigorously as the upper. An unusual resorption was observed at the attachment of the medial head of the gastrocnemius muscle at the posterior distal femur above the medial condyle. This was seen in twelve individuals, of both sexes. Gejvall (1983:86) describes a similar feature in prehistoric skeletons from Sweden and suggests one possible explanation is the forceful paddling of a canoe in a half-standing posture with one knee bent. In Peninsular Malaysia, flat-bottomed sledges are used to skim over intertidal mudflats to reach shell beds. These are used with one knee resting on the sledge and the other providing the propulsion (Sandbukt 1983, cited by Thompson 1996:315). It is possible that the individuals from Khok Phanom Di produced the pitting through engaging in water-related activities. Again, it is emphasised that these comments are speculative.

Dental pathology

The discussion of dental pathology in chapter VI has shown that dental health in the Khok Phanom Di people, in common with modern South East Asians, was good by modern international standards. It is comparable with that of many prehistoric populations but poorer than that of their prehistoric counterparts in North East Thailand. Despite overall comparability with modern South East Asian dental health, deterioration in the dentitions of older adults proceeded at a more rapid rate at Khok Phanom Di.

The aetiology of dental caries is complex, as was discussed in chapter VI. The explanations for both the relatively high levels of caries and sequelae at Khok Phanom Di compared with North eastern Thai and the more rapid decay and tooth loss compared with modern Thai may be equally complex. One contributing factor which has already been suggested (chapter VI) is the frequency of enamel hypoplasia in the third molars. There are high levels of caries and antemortem loss in these teeth. The increased caries susceptibility of hypoplastic enamel has already been discussed in connection with caries in deciduous dentitions. The frequent formation of pockets in the periodontal tissues adjacent to the posterior teeth in young adults is an indication that plaque accumulates around the molars. The combination of susceptible teeth and plaque bacteria results in the formation of caries in the presence of a suitable substrate. This is dependent on factors such as the type and concentration of carbohydrate in the diet, the physical characteristics of food, the frequency of eating and standards of oral hygiene (Nikiforuk 1985). Carbohydrates in the diet of the Khok Phanom Di people may have included highly cariogenic sugars (sucrose) from bananas (Stephan 1966) or from 'palm sugar', a brown sugar made from the inflorescence of the coconut palm (Wilson 1985). The consumption of palm sugar was recorded in Central Thailand in the 1950s (Hauck *et al.* 1959). The most common source of sugar in modern populations, sugar cane, is unlikely to have been available at Khok Phanom Di. One species of sugar cane, (*Saccharum officinarum*), originated in Papua New Guinea and another (*S. spontaneum*) in India (Purseglove 1972). A hybrid of these two, *S. sinense*, was sent as canes from Cambodia to China in AD 285 and had been chewed in South East Asia "since ancient times" (Purseglove 1972:214). However, there is no evidence to support the contact which would be necessary to explain the presence of either of the original cultivars in South East Asia in 2000 BC and it is unlikely to have been consumed at Khok Phanom Di. The starch in rice is not highly cariogenic nor is that in the other potential carbohydrate sources.

The physical characteristics of food contribute to caries susceptibility through the cleansing action of fibrous foods and adherence to tooth surfaces. At Khok Phanom Di, the rice would have contributed some fibre. Despite the stickiness of polished rice, the low caries rate in modern South East Asians suggests that it does not contribute to caries formation even in the North East, where glutinous rice is favoured. It is unlikely that the rice eaten at Khok Phanom Di would have been cariogenic. The proportion of pit and fissure caries in individuals under 30 years at death suggests that the diet was not sufficiently fibrous to wear the teeth at a rate which removed the occlusal fissures before caries could form. Sand and small bones ingested with seafood might have had some cleansing action on the teeth but seafoods are generally soft. Such behavioural factors as oral hygiene and the frequency of eating are unknown but the presence of a suitable substrate for caries formation on the Khok Phanom Di dentitions is possible. The combination of host susceptibility

(enamel hypoplasia), the presence of an agent (bacteria) in a cariogenic environment (substrate) results in caries. This model has not taken into account such factors as the flow rate and pH composition of saliva, which contribute to caries prevalence.

The DMF index does not differentiate between teeth missing as a result of caries and those lost through periodontal disease or excessive wear. It is possible that either or both of these progressed faster in the Khok Phanom Di dentitions than in modern South East Asians but the data are not directly comparable. An alternative approach to the explanation of earlier dental disease at Khok Phanom Di is to consider the reasons for low caries in modern South East Asians. The precise reasons have not been identified. Leatherwood *et al.* (1965) suggest the low caries rates in modern Thailand may be due to fluoride intake as dental fluorosis was recorded throughout the country. Despite high levels of fluoride excretion in urine, levels of fluoride in drinking water in Thailand were not high. A possible source is the universal condiment, fish sauce (*nam pla*). This is made from salt and fermented whole fish and although it is used in small quantities, it has a very high fluoride content (Russell *et al.* 1965). Whole fish have a high fluoride content when eaten with bones and skin (Møller 1982) and although the gut contents from B56 contained fish bones and scales it is unlikely that the daily consumption of these at Khok Phanom Di could contribute an equivalent quantity of fluoride to that derived from concentrated fish sauce. Another explanation for the low caries rate in modern Thai is offered by Kamalanathan *et al.* (1960a). From a survey of a small sample of adults in the Central Thai village of Bang Chan in 1953, they found adults who chewed betel nut had a lower caries rate than those who did not. The cariostatic effect of betel chewing has been recorded elsewhere (Schamschula *et al.* 1978). The habit is detrimental to the gingival tissues and could be partly responsible for an acceleration of periodontal disease and tooth loss in older individuals in the modern Thai samples. There is no evidence that the people at Khok Phanom Di were chewing betel nut, as the teeth did not have the characteristic staining or extreme calculus buildup associated with the habit.

Although the caries susceptibility of the prehistoric dentitions through enamel hypoplasia of the third molars and more rapid periodontal deterioration and tooth wear may be factors in accounting for their relatively rapid dental deterioration, there is no comparative information available on enamel hypoplasia in modern South East Asians. The cariostatic effect of both betel chewing and the ingestion of high levels of fluoride in the diet by the modern populations offer a more acceptable explanation.

This discussion has not accounted for the higher rates of dental pathology at Khok Phanom Di compared with prehistoric north eastern Thai. In the absence of directly comparable data the complexity of aetiology makes this difficult. Without getting into the realms of conjecture about such matters as fluoride intake, potential for enamel hypoplasia and relationship of deterioration with age, the only factor which can reasonably be considered is diet. At all north eastern Thai sites, there was evidence for the cultivation and consumption of rice for at least part of the period of occupation. The constituent of the diet which was different from that at Khok Phanom Di was in animal foods. Domesticated pig, cattle and deer were consumed along with fish in contrast to the emphasis on seafood at Khok Phanom Di. It is possible that the different consistency of meat from that of seafoods was sufficient to alter the availability of substrate around the dentition. There may also have differences in the availability and consistency of carbohydrates and the amount of fibrous veg-

etable food in the diet. Although dietary differences are unlikely to be the complete explanation for lower caries in the north eastern Thai sites, they may have made a contribution. It is more difficult to explain the greater proportion of dental infection in the Khok Phanom Di dentitions resulting in the formation of periapical cavities. As in the comparison with modern Thai, this may reflect an earlier onset of caries and periodontal problems in the Khok Phanom Di people, as periapical cavities form only in cases of chronic infection.

The sex difference in dental pathology merits further consideration. The higher caries rates in females appear to be the key to the other sex differences in pathology. Higher caries rates in females are not uncommon in prehistory, as illustrated in table 102. The levels of enamel hypoplasia are similar in both sexes at Khok Phanom Di, excluding this as an explanation. It has been recognised in modern populations that the earlier eruption of the permanent dentition in females means that the teeth are exposed to caries formation at an earlier age (Nikiforuk 1985) but this is unlikely to explain the extreme difference seen at Khok Phanom Di. Other causes of caries susceptibility are genetic or environmental factors which would affect both sexes equally, the only explanation which can be further considered is social. Indicators of growth and longevity have shown that females were not nutritionally disadvantaged but it is possible that diet and eating habits varied between the sexes. Lukacs (1992:144) suggests that the high caries rates in females in the Harappan people of the Indus Valley, similar to those at Khok Phanom Di, reflects sex differences in diet as a consequence of the sexual division of labour. He suggests this on the basis of the correlation of sex differences in dental pathology with differences in the sexual division of labour in modern African tribes with different subsistence modes (Walker and Hewlett 1990). At Khok Phanom Di, the sexual division of labour has already been suggested on the basis of sex differences in joint degeneration. It is possible that the caries differences, with the associated forms of other dental pathology, can be ascribed secondarily to the same social factor, through dietary differences. Cultural restrictions on the diet of either sex are also a potential explanation.

EXPLANATION FOR HEALTH STATUS - CONTRIBUTION OF NUTRITION, DISEASE AND 'LIFESTYLE'

This section attempts to answer the major questions arising from the results of the survey of health, using the evidence from the foregoing discussion of nutrition, disease and physical activity.

Neonatal mortality

As there is no evidence from the neonate skeletons which offers any clue as to the cause of their death, this discussion relies on indirect evidence. There are multiple potential causes of morbidity and mortality during the highly vulnerable perinatal period in a tropical environment. As commensal bacteria such as *Staphylococcus* and *Streptococcus* must have always been present in human populations and are found even in isolated groups (Hare 1967), they would have been present in the Khok Phanom Di population. Septicaemia is one of the main causes of neonate mortality in unhygienic conditions, with the umbilicus being a common route for entry of the bacteria (Jelliffe

and Stanfield 1978). The chances of infection can be increased through cultural attitudes towards the treatment of newborn infants, including methods of treating the umbilicus.

One or more of the thalassaemias or haemoglobinopathies may have contributed to the mortality rate. The most extreme form of α -thalassaemia (the condition of Hb Bart's hydrops foetalis) precludes survival of affected infants more than a few hours beyond birth, although the high rate of complications of pregnancy would have led to a high mortality rate in the mothers of such infants in prehistory. In the absence of evidence of a high rate of mortality in young females at Khok Phanom Di, it is difficult to justify the conclusion that homozygous α -thalassaemia was responsible for the deaths. A second possibility is neonatal jaundice associated with G6PD deficiency. This has a familial predilection for fatality in severe cases, though with at least one of the major haemoglobinopathies present at Khok Phanom Di, to suggest a second genetic disorder was present in the population creates a complex scenario. Haemoglobinopathic conditions other than homozygous α -thalassaemia do not affect neonates severely, with the typical clinical picture of HbH disease not developing until later in the first year of life (Weatherall and Clegg 1981).

Parasitism by intestinal helminths or protozoa is uncommon in neonates, as exposure to infection is minimal. Malaria is a more likely aetiology, as pregnant women, particularly primigravids, have increased susceptibility to infection. The placenta is a favoured site for parasitic growth and maternal immunity does not completely protect against exacerbation of parasitaemia and the clinical effects of the infection (Madrigal 1989). In areas of epidemic malaria where the level of acquired immunity in a population is low, severe malaria in pregnancy results in high rates of abortion, stillbirths, prematurity and congenital malaria (McGregor 1984).

In areas of endemic malaria, the levels of reproductive wastage "...vary inversely with the degree of tolerance of the disease possessed by the community." (Bruce-Chwatt 1978:833). The risk of low birthweights is increased, with attendant high rates of perinatal and neonatal mortality (Brabin 1991). In areas of endemic malaria where the mother does not become infected during the pregnancy, infants are protected for the first six-nine months by the transplacental transfer of antibodies (Jelliffe 1968). There is no convincing evidence of differential fertility between carriers of the sickle-cell gene and normal controls, suggesting that the suppression of immunity during pregnancy affects even those heterozygous for the blood polymorphisms which provide some protection against morbidity and mortality in non-pregnant individuals (Madrigal 1989). High levels of neonatal mortality from malaria may, therefore, have been possible at Khok Phanom Di despite the postulated presence of one or more of these genetic polymorphisms in the population.

Another possibility which cannot be absolutely discounted is infanticide. This has been commonly practiced among humans in the past (Dickeman 1975). Nevertheless, the evidence of ritual associated with the burial of many of the neonates argues against this being a major cause of infant death at Khok Phanom Di.

There is not necessarily a single explanation for the increase in neonatal mortality above that which could be expected in prehistory. The comparability of the rate with that from Lerna, a prehistoric population which lived in a malarial environment in Greece and included haemoglobinopathies (Angel 1971), suggests that the malarial parasite contributed to the high rate despite the presence of the blood polymorphism in the population.

Morbidity and mortality in infancy and childhood

The aetiology of the ubiquitous episodes of growth disruption in infancy and later childhood is potentially complex, and individually variable according to genotype. Depending on the degree of endemicity of malaria epidemiology, individuals with normal haemoglobin would have been exposed to the parasite from early in infancy once any maternally-acquired immunity dissipated. In endemic areas, children who survive any acute attacks during infancy gradually build up an immunity over about five years. Individuals who were possibly carriers of one of the thalassaemia genes would have been less susceptible to malaria, although probably not totally immune. In non-endemic areas, deaths can occur at all ages in non-immune individuals during an epidemic. The virtual absence of mortality of older children in the early mortuary phases at Khok Phanom Di suggests that malaria was endemic rather than epidemic. Repeated malarial attacks may be a principal aetiology in the growth disruption in infancy at Khok Phanom Di, along with seasonal malnutrition, the complications of intestinal parasites, low-grade anaemia in haemoglobinopathy carriers, bacterial infections and dietary problems at weaning. These problems are more than adequate in accounting for the high levels of ill health in infants.

The episodes of growth disruption in later childhood are less likely to reflect malarial attacks as the individual immunity is built up by this time. Seasonal dietary inadequacies, intestinal parasites, infections, and the anaemia of haemoglobinopathy carriers continue to be possible explanations, potentially together with social or cultural factors such as the quality of the diet available to children and the age which they were expected to contribute to the work of the community.

The primary cause of death of six post-neonate infants and children listed in table 77 may be a consequence of the haemoglobinopathy diagnosed in these cases. The ultimate cause of death is most likely to have been secondary infection, as this is the most common complication of the haemoglobinopathies. The remaining two-thirds of infants and half of children could have died from malaria alone or in combination with any of the conditions listed in the previous paragraph. The mortality rates of these infants and children is again almost identical to the rate at Lerna.

Adult morbidity and mortality

As mature bone is less responsive to acute ill health than immature bone, skeletal evidence of systemic morbidity in adulthood is not necessarily distinguishable from the long-term effects of ill health in childhood. The individuals with slight cribra orbitalia were not necessarily, therefore, suffering from anaemia at the time of death.

The good cortical thickness in both sexes may be a reflection of relatively good health in adolescence and early adulthood compared with health early in life. Permanent stunting when catch-up growth is inadequate after episodes of growth disruption is not necessarily matched by permanent loss of bone cortex. There are two possible explanations for this. Cortical apposition continues after the cessation of growth in stature at the time of skeletal maturation. In areas of malarial endemicity where individuals over the age of about 10 years have acquired an immunity to the parasite, adolescents and adults will be relatively healthy. With this reduction of the effect of the pathology, cortical thickness will continue to increase into adulthood. The alternative explanation is a reflection of a suggested relative sensitivity of cortical thickness compared with longitudinal growth to recovery

after a period of protein-calorie malnutrition (Himes 1978). The availability of adequate nutrition after the episodes of childhood morbidity may have enabled restoration of cortical thickness which was not matched by catch-up of growth in stature. This does not necessarily suggest the unavailability of adequate nutrition, but could reflect the interaction between nutrition and infection.

The joint degeneration would have caused pain and immobility only in the small proportion of mainly older adults with multiple joint involvement. The dental degeneration could potentially have had a more significant impact on systemic health. Individuals with multiple caries would have suffered pain and discomfort as well as interference in their ability to eat. Individuals with multiple periapical cavities were not, however, necessarily ill. The presence of so many lesions even suggests that, although infections had persisted, they were healthy enough to withstand its effects. Nevertheless any lesion represents the potential for involvement of dental infection in an individual's systemic health, as impairment of the immune system or virulence of the causative organism could result in the development of a serious acute abscess or osteomyelitis.

The potential contributing factors to the early adult mortality include the long-term effects of parasitic infection, including malaria and intestinal parasites, dental infections, and anaemia and increased susceptibility to infection of carriers of the haemoglobinopathy. Decreasing mobility in those who survived beyond the age of 40 years would have affected their ability to take an active part in the life of the community. The combination of increasing reliance on others for food and the reduction through tooth loss in the ability to eat many foods could have affected the nutritional balance of the diet.

Sex differences in health and mortality

Most of the above discussion of both childhood and adult health applies to both sexes equally. It is, however, unfortunate that the sex of the infants and children is not known, as the comparison of the health of boys and girls may have helped explain the apparent anomaly of the stronger evidence of growth disruption (in the form of Harris lines) in females but shorter adult stature of males. Other than the possibility of differential recovery from illness already discussed in chapter V, the only further explanations that have arisen from the evidence presented in this chapter rely on cultural differences in the behaviour or treatment of the sexes. One possibility, suggested by the early onset of joint degeneration in males, is that adolescent males were expected to contribute from an early age to the physical workload of the community to the extent that growth was disrupted. For example, restrictions on the quality or quantity of food at the crucial stage of adolescent growth could have impeded full achievement of growth potential. An alternative explanation is a sex difference in nurturing of infants and children. This is not uncommon, even in modern societies, and can favour either sex (Hrdy 1990). The people of Khok Phanom Di may have preferentially treated female children with the result that they recovered more quickly from illness. Alternatively, as first postulated by Bannanurag (1989), the society may have been matrilineal, with at least some of the males having spent their vulnerable childhood years in a different environment. Whatever the explanation for the male stature, once adulthood was reached, both sexes were sufficiently healthy for maintenance of cortical thickness.

The relatively short lifespan of males compared with females is also difficult to explain. The cost and potential dangers of pregnancy would be expected to have resulted in relatively high fe-

male mortality in early adulthood. The earlier onset of dental pathology in females does not appear to have had an effect on life expectancy. The possibility of a long-term effect of the same factors that may explain their stature is argued against by the good cortical thickness. Joint degeneration would not have been a factor as both sexes appear to have suffered to a similar degree with moderate-severe degeneration. The possibility of a social explanation again needs to be considered. The sexual division of labour has already been suggested on the basis of skeletal and dental pathology. It is possible that the difference in male and female activities exposed them to different pathogens or physical dangers which affected health. Given the difficulties of estimating the age at death of adults, discussed at length in chapter II, the possibility that the age estimates are inaccurate unfortunately cannot be discounted.

VIII. CHRONOLOGICAL VARIATION IN MORPHOLOGY AND HEALTH

GIVEN the interpretation from the radiocarbon dates that the cemetery represented a period of about half a millennium, and the evidence from biological and inorganic analyses that there was substantial change in the local environment during that time, there was considerable potential for variation in the health of the human population over time. This chapter will consider this question. Tied in with this is the potential complication of a change in the gene pool, through migration or microevolution. Variation in morphology may represent either genetic variation or phenotypic adaptation to the environmental change. The combined factors of the lack of sedimentological evidence for abandonment of the site for any period and the continuity of aspects of the mortuary practices suggest that occupation of the site at different times by two or more unrelated groups is unlikely, despite the hiatus in the burials between MP3 and 4. However, interpretation of any phenotypic variation is complicated by the effects of such factors as the possible immigration of one or two prolific breeders. These people would not be easily identified but could have a long-term influence on the morphology of the population. Another complication is the potential for genetic drift resulting from increasing domination of the gene pool by healthier families. This question will be addressed with these caveats in mind. The interaction of genetic and environmental influences on morphology and health is complex and the effects of each are not easily teased out from the limited skeletal evidence. The selection of data used in addressing the questions is discussed below.

As noted in chapter I, the matrix surrounding the burials was virtually undisturbed, either by subsequent human activity or biological or geomorphological processes. This permitted the identification of graves at the stratigraphic level from which they were cut. In combination with the rapid build-up of deposits over graves and the practice of superpositioning of burials, this permitted the relative dating of graves from the initial identification of the grave cut in relation to a surface datum point. In the following analyses, this 'depth below datum' of the graves is used as the primary temporal variable. This removes any imposition on the data of subdivisions based on non-biological criteria. Nevertheless, where the data are available in sufficient numbers, mortuary phases are used as secondary variables to identify whether there is any relationship between cultural and/or social variation and human biology. In some cases, the sample is subdivided at the MP3-MP4 hiatus, which roughly corresponds to the major environmental change and gives two relatively equally-sized samples representing early (MP1-3) and late (MP4-7) burials.

PHYSICAL APPEARANCE

The metric characteristics of the adult skeletons and teeth are considered here. The selection of measurements to be used in this exercise is problematic because of the undoubted but unknown extent to which environment interacts with genetics in determining size and shape. The relationship

between genotype and phenotype has long been recognised as highly complex and yet is often ignored in studies of population relationships. The debate over the selection of traits, either metric or non-metric, for such a purpose is very wide-ranging and detailed discussion is not appropriate in this context. One school of thought advocates using as many traits as possible in the belief that some must be genetically controlled and therefore the truth will shine through. Another advocates rebutting such attempts altogether, in the belief that not only can environment confound the genetic underpinnings to the phenotype to too great an extent, but also the polygenic nature of most morphological characteristics is too complex for any simple association between genotype and phenotype to be exhibited. This section documents rather than infers possible causes from the data.

Individual metric data are given in the appendix tables to chapter III. Summary statistics for early (MP1-3) and late (MP4-6) periods are given in tables 140-143. The craniometric data were separately analysed for males and females, with immature individuals (≤ 18 years) excluded. For most cranial and facial dimensions, there are not enough measurements in the later burials for confident statements to be made about changes over time. Univariate regression analyses against depth below datum showed no change over time, with the single exception of nasal breadth in females, which became significantly narrower. Regression plots showed that many variables included outliers, which were not consistently the same individuals as none stood out for more than two measurements. Comparing early with late burials, the mean of most dimensions decreased in females and increased in males over time. Non-parametric univariate analyses (Mann-Whitney) showed that in addition to nasal breadth, mean bimaxillary breadth was significantly smaller in females in the later burials. Multivariate analysis was not attempted because of the uneven distribution of data. The small sample sizes and the amount of missing data cast doubt on the validity of the statistical results. Because of missing data, the presence of true outliers for cranial size and shape cannot be ruled out.

There were larger sample sizes for mandibular dimensions than for cranial dimensions. Multivariate regression analysis showed that there were no clear trends over time in either sex. Univariate regression against depth below datum showed a significant increase in the gonial angle and the anterior width (bimental foramen width and bimaxillary foramen width) in females. In males, the ramus breadth decreased significantly. Nonparametric univariate analyses of early against late burials repeated the finding of a significant increase in anterior width in females but only through the bimental foramen width. Results for the males showed a significant increase in the mean gonial angle, although the small sample in the later period was strongly affected by an outlier, B9, who had a very small mandible. There appears to have been a change in shape in the female mandibles, to a more U-shape. Given that it is reflected in an increase in the anterior width, rather than a decrease in the posterior width, this doesn't correlate with the decrease in facial width suggested by the bimaxillary measurement. It is difficult to see a biological correlate of these data, which suggests that the statistical analysis cannot be relied on as evidence of a change over time. Overall, there is no clear trend over time and again, a lack of consistency between the sexes.

In the dental measurements, there were no significant differences in both sexes between left and right tooth sizes so the measurements were averaged. Using the crown index (mesio-distal length x bucco-lingual breadth), most teeth increased in size over time. Univariate regression showed that the increases were significant in the anterior maxillary teeth of females and the maxillary first mo-

lar and mandibular lateral incisor and first premolar in males. The comparison of the early and late burials through univariate nonparametric analysis confirmed the results in females but gave different results for males. The maxillary first and second molars and mandibular lateral incisor and first molar were close to significantly larger but the mandibular premolar was not.

Most postcranial measurements did not change significantly over time. In males, almost all mean dimensions, including lengths, shaft and joint widths were smaller in the later period than earlier, but none of the differences was significant with the single exception of the maximum diameter of the right humerus head. Conversely, in females, the few mean dimensions which changed at all became larger over time. The only increases were the left femur head and the medio-lateral diameter in the right tibia, which indicates that the bones became more rounded over time (less platycnemic).

There is no clear pattern of change in skeletal and dental dimensions over time, either within or between the sexes. The mean dimensions of the cranium and the mandible in the females suggest a change in shape of the head with narrower mid faces and broader lower faces. The anterior maxillary teeth became larger which correlates with the latter. There is a trend to larger bodies and limbs. In the males, the crania did not change significantly, although most means were larger in the late burials. Conversely, the significant changes in the mandibles suggest a decrease in masticatory musculature, although there was an increase in size of some posterior teeth. The postcranial measurements also mainly decreased over time but none significantly so. The inconsistency in the pattern of changes within the males may be an artifact of the small sample sizes. The lack of a clear pattern in males and females suggests that there was not a universal phenotypic or genetic adaptation or change in the gene pool through significant immigration of both sexes. It does not rule out the immigration of either individuals or small numbers of males or females.

TOOTH ABLATION

Although the deliberate removal of teeth is obviously not a biological characteristic, it is briefly reviewed here to complete the consideration of the changes over time. Full details of the ablation patterns are given in Tayles (1996). The patterns are summarised in table 118.

The earliest example was B147, in MP1. Over half the adults, both males and females, had teeth ablated in MP2. All involved the lateral maxillary incisors (Fig. 83A) or mandibular central incisors only, with one exception. There were no significant differences between males and females in the patterns. In MP3 the proportion increased to 74 per cent. Patterns were more varied, but in many cases involved lateral maxillary incisors only or with either maxillary canines or all mandibular incisors. Again there were no significant sex differences.

All the adults and one of five children had teeth ablated in MP4. These involved central maxillary incisors in all cases, together with maxillary lateral and mandibular incisors in a variety of patterns (Fig. 83B). None was exclusive to this phase. In MP5 and 6, all adults and children had teeth ablated, in a variety of patterns similar to that in MP4. Neither of the two children in MP7 had any teeth missing.

TABLE 118: *Patterns of tooth ablation, with numbers and proportions of individuals with each pattern; by mortuary phase and for the total sample*

	MP1-3			MP4- 7			Total		
	n	%	% ¹	n	%	% ¹	n	%	% ¹
	39			30			69		
No missing teeth	14	36		5	17		19	28	
Maxillary incisors									
Lateral x2	12	31	48	0	0	0	12	17	24
Lateral x1	4	10	16	0	0	0	4	6	8
Lateral x2; canines x2	2	5	8	0	0	0	2	3	4
Central x2; lateral x1	1	3	4	4	13	16	5	7	10
Central x2; lateral x2	0	0	0	3	10	12	3	4	6
Central x2	0	0	0	6	20	24	6	9	12
Total with maxillary teeth missing only	19	49	76	13	43	52	32	46	64
Mandibular incisors									
Central x2	2	5	8	0	0	0	2	3	4
Maxilla and Mandible									
Max lateral x2; Mand central x2, lateral x1	3	8	12	0	0	0	3	5	6
Max lateral x1; Mand central x2, lateral x1	1	2	4	0	0	0	1	1	2
Max central x2; Mand central x2, lateral x2	0	0	0	6	20	24	6	9	12
Max lateral x2; lateral x2; Mand central x2, lateral x2	0	0	0	4	14	16	4	6	8
Max central x2; Mand central x2	0	0	0	1	3	4	1	1	2
Max central x2, lateral x1; Mand central x2, lateral x2	0	0	0	1	3	4	1	1	2
Total with maxillary and mandibular teeth missing	4	10	16	12	40	48	16	23	32
Total with teeth missing	25	64	100	25	83	100	50	72	100

¹ percentage of sample with missing teeth

The pattern of dental ablation changed between MP3 and MP4, from a predominance of lateral maxillary incisors ablated in a proportion of adults to a predominance of central maxillary incisors in all adults. The patterns were therefore mutually exclusive between the early and late burials, with a single exception. It is interesting that the change in the patterns of tooth ablation occurred at the same time as the arrival of the unusual turtle carapace ornaments in male skeletons, which may indicate influence from a new group of people. It is interesting to note that the only individual prior to MP4 with an ablation pattern found in the later phases (B57) was also the only individual prior to MP4 with a turtle carapace ornament. These cultural changes are contrary to the lack of a clear metric evidence for a change in the genetic composition of the population.

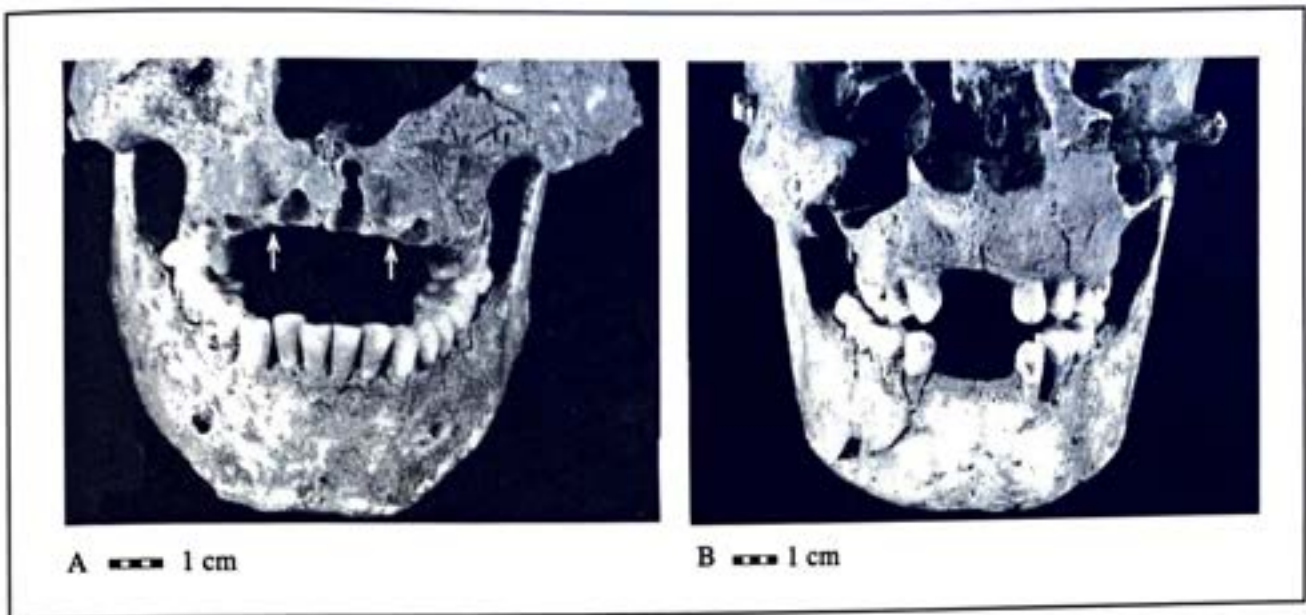


FIG. 83. A. Dentition of burial 92, male aged c. 18 years. The lateral maxillary incisors have been ablated. The other anterior maxillary teeth were lost postmortem. B. Dentition of burial 36, female aged c. 21 years. The central and right lateral maxillary incisors and all mandibular incisors are missing. Despite the asymmetry in the maxilla, these were probably deliberately ablated

HEALTH

This section addresses the second question, whether there is evidence for changes in health and disease over time. The data presented are a selection from those used in the detailed analysis of health and disease in chapters V and VI. Characteristics included are those for which evidence is available for all, or most, of the life of the cemetery. The results are then summarised for each mortuary phase and the overall evidence considered.

Stature and mass

As discussed in the previous section, there is no evidence for significant change in individual postcranial long bone lengths over time. Table 119 summarises the data for early and late phases and figure 84 shows individual stature estimates against depth of burial.

In the females, there is no statistically significant trend in stature over time. In the males there is a trend towards decreasing stature from MP4, which regression analysis shows to be significant at the 10 per cent level ($p=.095$).

Despite the lack of statistically significant results, there are trends, with the three individuals in MP1 all taller than average and the two males and four of the five females in MPs 5 and 6 shorter than average. The very short outlier in MP3 is B56, discussed in chapter V.

TABLE 119: *Variation over time in mean stature*

	MP1-3			MP4-6			Significance
	n	Mean	s.d.	n	Mean	s.d.	
Females	21	154.18	4.52	15	154.47	4.59	ns
Males	19	163.44	5.20	11	160.20	4.75	p=0.10

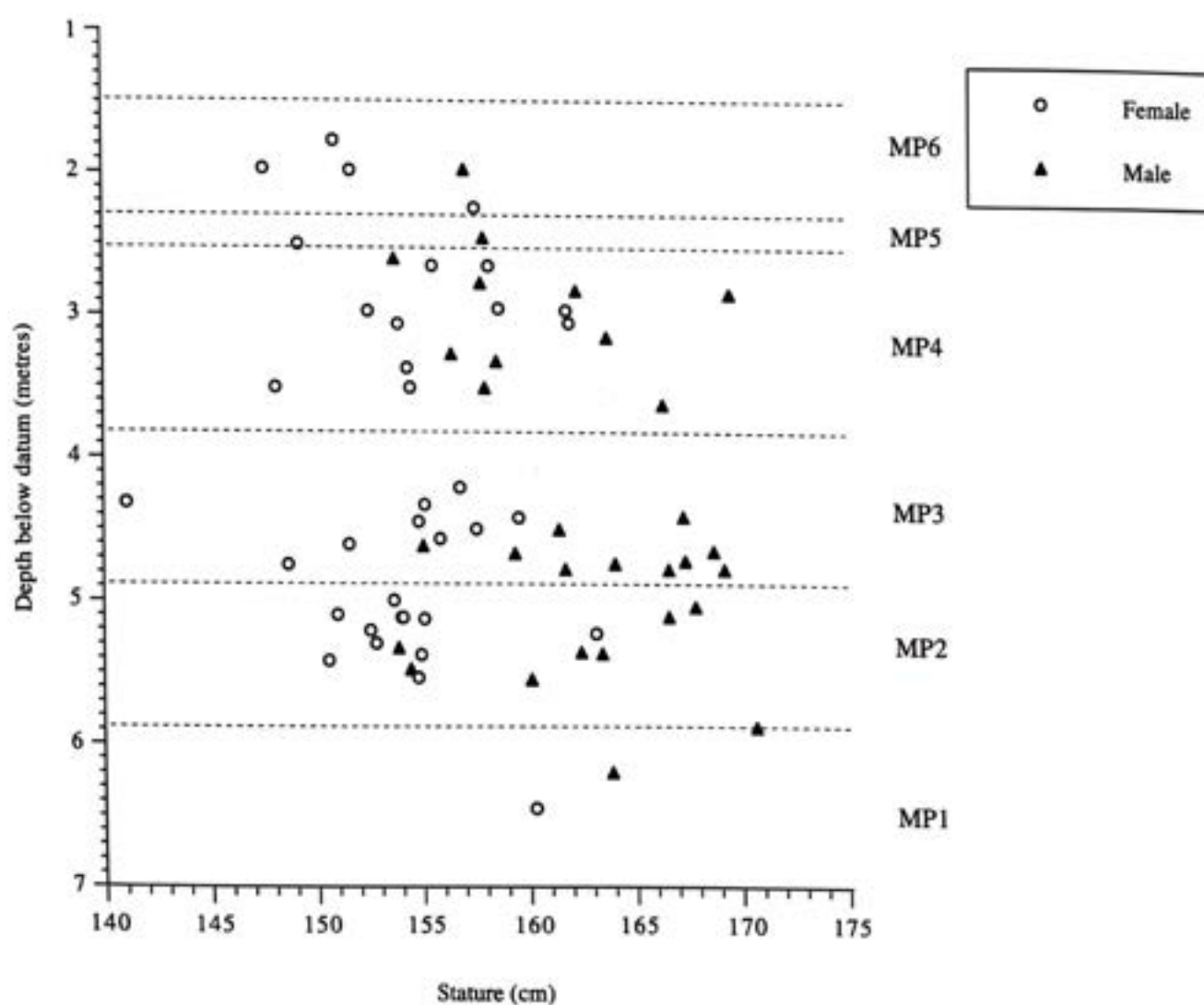


FIG. 84. Stature of adults shown against depth of burial and mortuary phase

The smaller sample of mass estimates, summarised in table 120, show that this measure of body size also does not change significantly over time. Figure 85 shows individual mass estimates against depth of burial. Despite the lack of statistical significance overall, all males except one are relatively large in MP3 and early MP4, compared with MP2 males and later burials. Females are similar in mass throughout, with the exception of the large outlier in MP4 and the two individuals in MP6 being below average. There are no estimates for the three individuals in MP1.

TABLE 120: *Variation over time in mean mass*

	n	MP2-3		n	MP4-6		Significance
		Mean	s.d.		Mean	s.d.	
Females	13	46.89	5.52	4	45.48	3.89	ns
Males	13	55.26	4.68	6	53.42	4.16	ns

A comparison of the mean size data by mortuary phase (Table 121; Fig. 86) shows that the males were not only on average tallest (apart from the two individuals in MP1) and heaviest in MP3 but the low stature:mass ratio shows that they were also heaviest for their height. The two earliest males in MP4 had a similar large body size. Burial 103 is the outlier in MP3 as he had relatively slender bones for his height. Females in MP3 were also heavier on average than in MP2 but were not taller. Apart from the heavy outlier in MP3, B61, they were of similar size in MP2 and MP3. With the exception of the two large males in early MP4, the few individuals in the later burials were small but within the range for MP2.

TABLE 121: *Variation over time in mean stature and mass*

	Stature				Mass				Stature:Mass ratio			
	Females		Males		Females		Males		Females		Males	
	n	mean	n	mean	n	mean	n	mean	n	mean	n	mean
MP1	1	160.3	2	167.3	-	-	-	-	-	-	-	-
MP2	11	154.2	7	161.2	6	44.92	5	51.92	6	3.43	5	3.16
MP3	9	153.5	10	164.1	7	48.58	8	57.35	7	3.23	8	2.84
MP4	10	156.0	8	160.8	2	47.42	5	54.39	2	3.27	5	3.01
MP5-6	5	151.4	2	157.6	2	43.55	1	48.60	2	3.51	1	3.23

Sexual Dimorphism

The degree of dimorphism in stature and body mass in each mortuary phase is listed in table 122. As would be expected, males are on average taller and heavier than females in all phases, with the

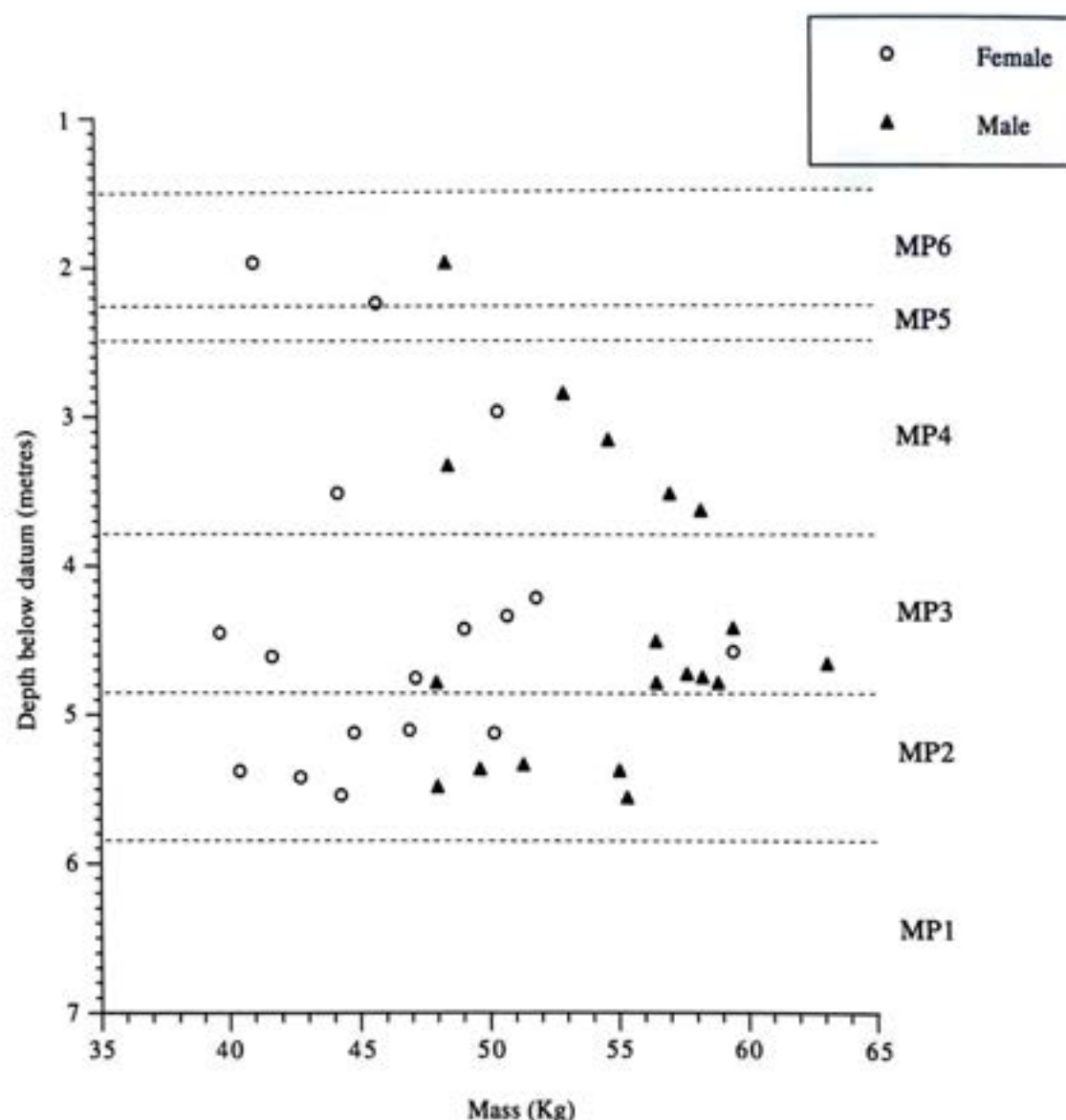


FIG. 85. Mass of adults shown against depth of burial and mortuary phase

difference statistically significant in MP2-4. Dimorphism reaches its highest level in MP3 and reduces in the later phases. The data on long bone dimensions shows that dimorphism in the circumference of the humerus is significant in MP2-4 (sample sizes in MP1 and MP5-6 are too small for assessment). The circumference of the femur is also significantly different in MP2 and 3 but ceases to be so in MP4.

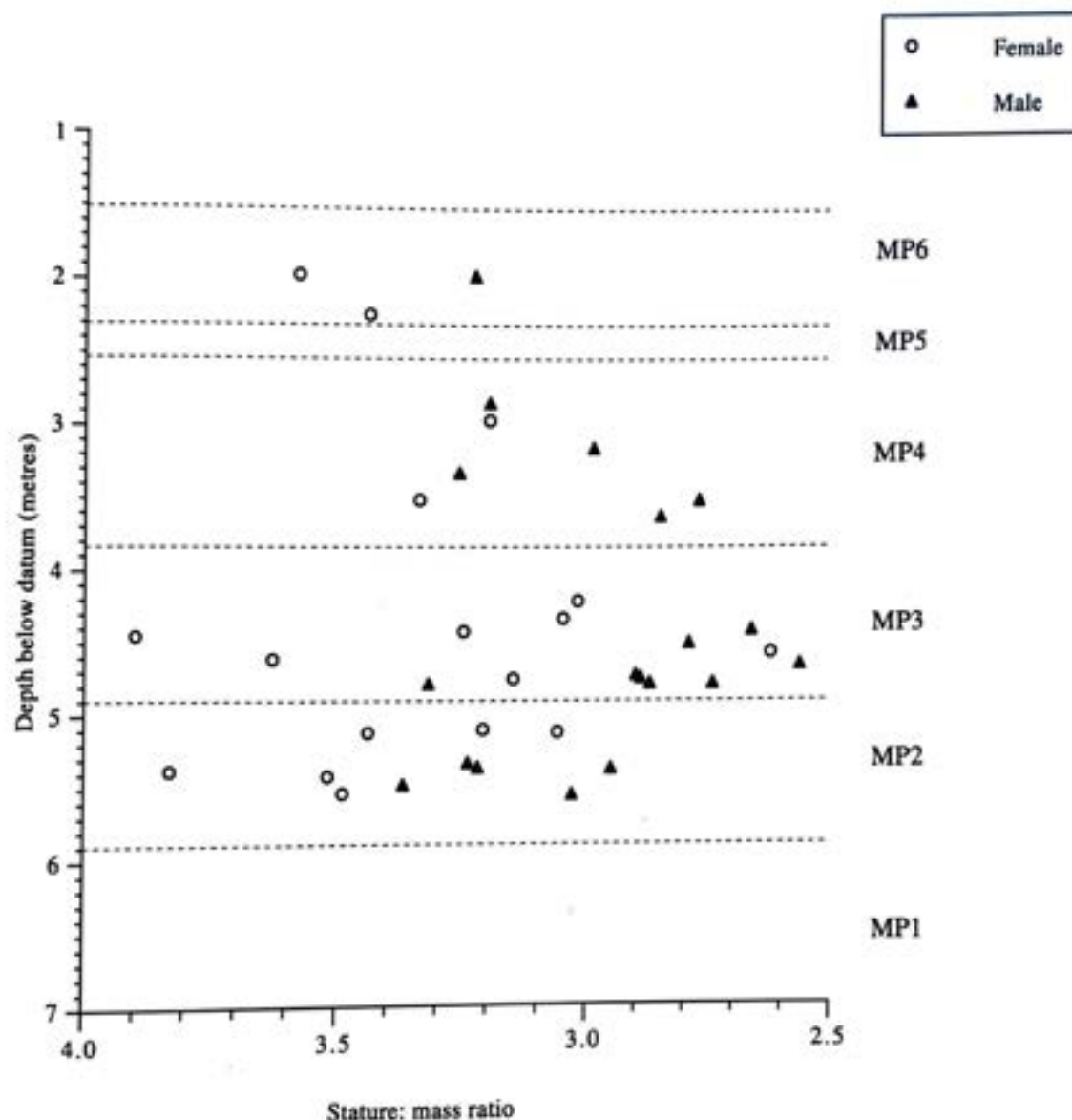


FIG. 86. Stature:mass ratio of adults shown against depth of burial and mortuary phase

Harris lines

The mean number of lines recorded on the distal tibiae is listed in table 123 by sex and mortuary phase. The prevalence of line formation in males appears to vary less over time than in females but regression analysis shows there was no statistically significant difference.

TABLE 122: *Variation over time in mean sexual dimorphism in stature and mass*

	% of male stature		% of male mass	
MP1	4.2		-	-
MP2	4.3	**	13.5	**
MP3	6.5	***	15.3	**
MP4	3.0	*	12.8	*
MP5-6	3.9	ns	10.4	ns

significance *** $\leq .001$ ** $\leq .01$ * $\leq .05$

If B56 is excluded from the female mean stature, the degree of dimorphism is reduced to 5.5 per cent. The level of statistical significance does not change

TABLE 123: *Variation over time in the mean number of Harris lines in adult distal tibiae*

	Total				Females				Males			
	n	Mean	s. d.	Range	n	Mean	s. d.	Range	n	Mean	s. d.	Range
MP1	-	-	-	-	-	-	-	-	-	-	-	-
MP2	13	2.46	1.51	1-6	6	3.00	1.90	1-6	7	2.00	1.00	1-3
MP3	12	1.92	1.38	1-5	5	1.60	1.34	1-4	7	2.14	1.46	1-5
MP4	16	2.69	2.94	0-11	8	3.88	3.68	1-11	8	1.50	1.31	0-3
MP5-6	3	1.00	0.00	1-1	1	1.00	-	1-1	2	1.00	0.00	1-1

Enamel hypoplasia

The variation over time in the proportion of adults with each type of developmental defect of the dental enamel is summarised in table 124. A majority of individuals in all mortuary phases had either linear enamel hypoplasia, non-linear hypoplasia or both. There were no statistically significant trends over time.

The variation over time in the number of children with dental enamel defects in permanent teeth is listed in table 125. No detailed conclusions can be drawn because of the small numbers involved, but the absence of any defects in the single individual in MP2 and two of the four individuals in MP6 and 7 and the presence of non-linear enamel hypoplasia only in the two individuals with defects in the latter phases is noted. The prevalence of different types of defects in the larger sample in MP4 is similar to that observed in the adult teeth.

The variation over time in the proportion of children with different types of developmental defects of the enamel in deciduous teeth is summarised in table 126. The discrete hypoplastic defects

TABLE 124: *Variation over time in the mean number of developmental defects of adult dental enamel*

	N	Linear ¹	Non-linear ²	All ³	Mean LEH ⁴
Females		n	n	n	
MP1	1	1	1	1	3.00
MP2	8	7	5	8	1.56
MP3	7	7	7	7	1.44
MP1-3	16	15	13	16	
MP4	7	6	5	6	1.22
MP5-6	3	3	1	3	1.00
MP4-6	10	9	6	9	
Males					
MP1	1	1	0	1	3.50
MP2	7	6	7	7	1.63
MP3	9	7	6	7	1.70
MP1-3	17	14	13	15	
MP4	7	6	7	7	1.71
MP5-6	2	2	2	2	0.67
MP4-6	9	8	9	9	

¹ Number of individuals with at least one tooth with linear enamel hypoplasia (codes 1,2 - refer to Page 101)

² Number of individuals with at least one tooth with non-linear enamel hypoplasia (codes 3-5)

³ Number of individuals with at least one tooth with hypoplasia of either type

⁴ Mean number of linear defects per individual, based on the defects appearing on the canines

occurred in infants in both early and late mortuary phases but the staining of teeth occurred only in MP1-2.

Infant and child mortality

Figure 87 illustrates the pattern of infant and child burials over time and table 127 summarises the data. These illustrate a marked change in the age composition of the child burials between MP1-3 and MP4-6. In the earlier period, the pattern is similar in each mortuary phase, with a majority of the deaths occurring at birth or during the first year. There are very few older children. In the later burials, the deaths are more evenly spread over all age groups. In MP7 there are no neonates and three of the five burials are of children over five years old.

The proportion of total mortality represented by infants and children in each mortuary phase is discussed in chapter II in the context of variation in the demography of the group over time.

TABLE 125: *Variation over time in the numbers of children with different types of developmental defects of the enamel of permanent teeth*

	N	Linear ¹	Non-linear ²	All ³	No EH ⁴
MP2	1	0	0	0	1
MP4	5	1	2	2	0
MP6	2	0	1	0	1
MP7	2	0	1	0	1
MP4-7	9	1	4	2	2

¹ Number of individuals with at least one tooth with linear enamel hypoplasia (codes 1,2 - chapter IV)

² Number of individuals with at least one tooth with non-linear enamel hypoplasia (codes 3-5)

³ Number of individuals with teeth with both linear and non-linear hypoplasia

⁴ Number of individuals with no enamel hypoplasia

TABLE 126: *Variation over time in the numbers of children with different types of developmental defects of the enamel of deciduous teeth*

	N	Discrete EH ¹	Stain ²	Both ³	No defects
MP1	1	0	0	1	0
MP2	8	4	2	0	2
MP3	1	0	0	0	1
MP4	4	1	0	0	3
MP5	1	1	0	0	0
MP6	3	2	0	0	1

¹ Number of individuals with at least one tooth with enamel hypoplasia (code 4 - refer to chapter IV)

² Number of individuals with at least one tooth with discoloured enamel (code 5)

³ Number of individuals with teeth with both types of defect

Adult mortality

The mean ages at death of adults in each mortuary phase, in total and for each sex, are listed in table 128. Although the mean age at death is higher for females in all mortuary phases than for males except in the small sample in MP1, these sex differences are not statistically significant. Mean age at death for both sexes was higher in MP2 and 3 than in the subsequent mortuary phases but again

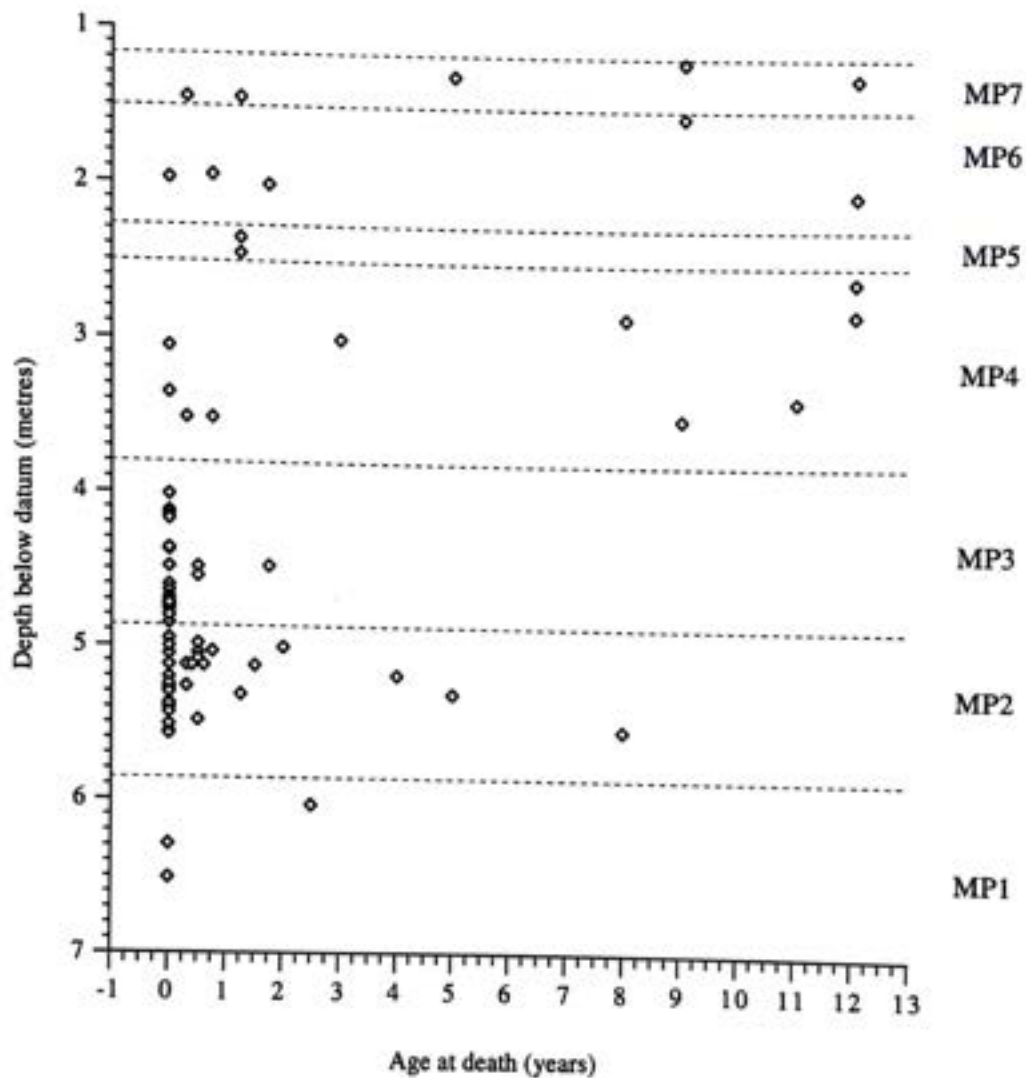


FIG. 87. Infant and child mortality shown against depth of burial and mortuary phase

the difference is not statistically significant. Figure 88 illustrates the composition of the samples and shows lack of males in the older age groups in MP4-6, where no males survive beyond the age of 35 years.

Cortical thickness

Figure 89 illustrates individual cortical indices in relation to depth of burial. Changes over time are not significant in either sex, although the two males in MP1 have cortical indices which are

TABLE 127: *Variation over time in infant and child mortality*

	Total	Age at death (years)															
		Neonates		≤ 1		1-4		≤ 5		5-9		10-14		5-14		≤ 15	
		n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	%
MP1	6	2	33	-	-	1	17	3	50	-	-	-	-	-	-	3	50
MP2	56	21	38	10	17	4	7	35	62	2	4	-	-	2	4	37	66
MP3	42	20	48	2	5	1	2	23	55	-	-	-	-	-	-	23	55
MP4	29	2	7	2	7	1	3	5	17	2	7	3	10	5	17	10	34
MP5	4	-	-	-	-	2	50	2	50	-	-	-	-	-	-	2	50
MP6	12	2	17	1	8	1	8	4	33	1	8	1	8	2	17	6	50
MP7	5	-	-	1	20	1	20	2	40	2	40	1	20	3	60	5	100
Total	154	47	31	16	10	11	7	74	48	7	5	5	3	12	8	86	56
(MP5-7	21	2	10	2	10	4	18	8	38	3	14	2	10	5	24	13	62)

TABLE 128: *Variation over time in adult mean ages at death*

	Females			Males			All Adults		
	n	mean	s.d.	n	mean	s.d.	n	mean	s.d.
MP1	1	25.0	-	2	28.0	12.7	3	27.0	9.2
MP2	11	34.0	9.7	8	29.4	9.4	19	31.9	9.6
MP3	9	32.9	11.4	10	30.4	7.3	19	31.6	9.3
MP4	10	29.1	9.5	9	26.8	5.9	19	28.0	7.9
MP5	1	35.0	-	1	30.0	-	2	32.5	3.5
MP6	4	32.0	8.0	2	22.5	10.6	6	28.8	9.2

higher than average. One of these, B152, has been considered pathological and was discussed in chapter V.

Skeletal pathology

The miscellaneous infections and injuries documented in chapter V are scattered throughout MP2-4. The lack of any pathology in MP1 and MP5-6 may reflect both the small samples and the relatively poor condition of the skeletons. There is no good evidence for a concentration of any one disease during a particular time period.

The temporal distribution of the anaemic individuals is of more relevance to understanding changes in the society. The data are summarised in Table 129. Neonates (n=47) are excluded. In

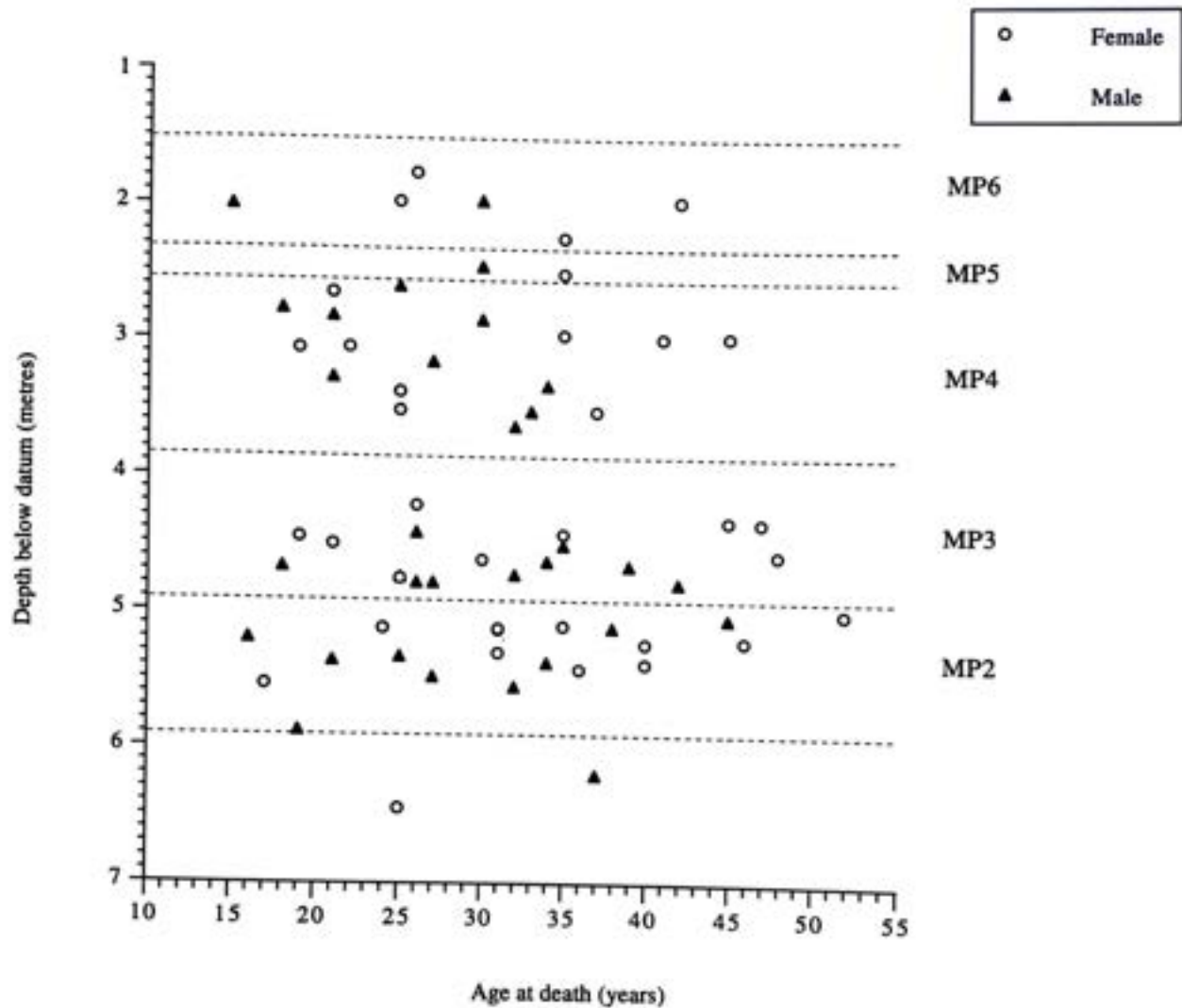


FIG. 88. Adult age at death shown against depth of burial and mortuary phase

the early phases, infants were affected. From MP3 there were relatively few infants in the sample but they were not affected and the few cases indicate that individuals with anaemia were surviving at least into childhood.

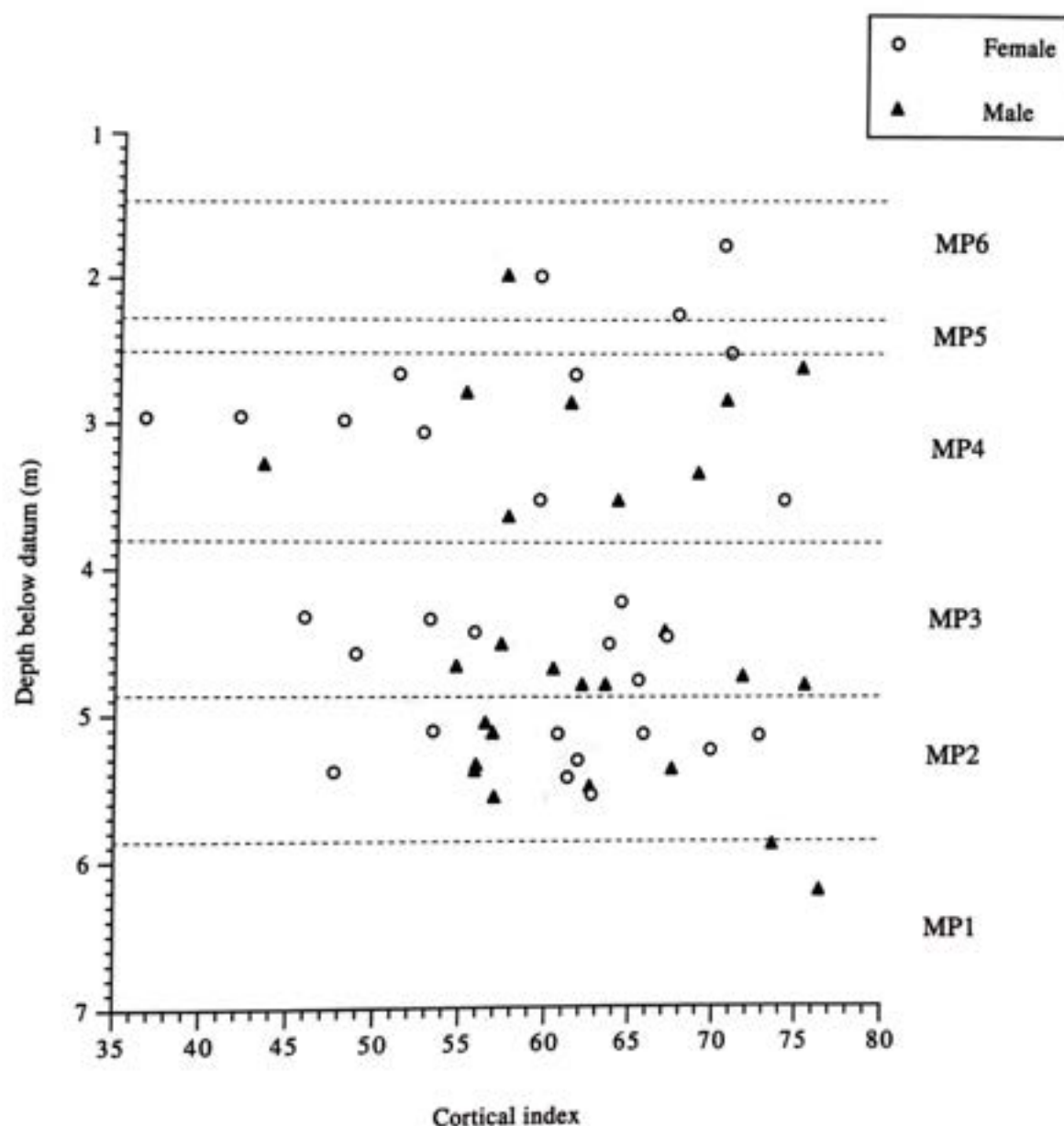


FIG. 89. Individual adult cortical indices (Nordin scores) shown against depth of burial and mortuary phase

Arthritis and osteophytosis

Tables 130 and 131 summarise the prevalence of vertebral degeneration in each mortuary phase. Because of the small numbers in each phase the statistical models used in assessing overall prevalences of degeneration are not valid. Data are provided for the number of individuals in each phase and the evidence is considered on this basis. Osteophytosis of the lumbar vertebrae was the most

TABLE 129: *Variation over time in the proportion of individuals with anaemia*

MP	Infants			Children			Adults			Total		
	N	n	%	N	n	%	N	n	%	N	n	%
1	1	1	100	0	0	0	3	0	0	4	1	25
2	14	4	29	2	0	0	19	0	0	35	4	11
3	3	0	0	0	0	0	19	1	5	22	1	5
4	3	0	0	5	1	20	19	1	5	27	2	8
5	2	0	0	0	0	0	2	0	0	4	0	0
6	2	0	0	2	0	0	6	0	0	10	0	0
7	2	0	0	3	0	0	0	0	0	5	0	0
Total	27	5	19	12	1	8	68	2	3	107	8	7

prevalent degenerative condition of the spine in the females in all mortuary phases. There is no clear pattern of change over time in the prevalence of type or location of degeneration other than in the porosity of the bodies of the cervical vertebrae, which affected a higher proportion of individuals in MP2 than in later phases. The patterns of prevalence of arthritis did not vary over time in the males. There was a higher proportion of individuals with phytosis and porosity of the cervical spine in MP2 and phytosis of the upper thoracic spine in MP3 than in other phases. In the lower back, both lower thoracic and lumbar spine were affected in all phases, although there was a relatively high prevalence of collapse or compression of the vertebrae in MP3.

Table 132 summarises variation over time in the number and proportions of individuals with degeneration in appendicular joints. In MP2 the joints most commonly affected in the females are the shoulders, hips and knees. In MP3 all joints are affected to a similar degree except the hips. In the later phases joint degeneration is less common overall. In the males, the temporal differences are more marked, with high proportions of individuals in MP1-3 having degeneration at the shoulders and the knees, ankles and feet in particular. In contrast, in the later phases almost no males suffered from joint degeneration.

Dental pathology

Table 133 summarises the caries by mortuary phases. In the females, caries affected a higher proportion of individuals in MPs 2 and 5-6 than in other phases but overall the highest rates were in MPs 5-6. In the males, rates were highest in MP3 but overall the females were affected more than the males.

The numbers of children in each mortuary phase are too small to draw any conclusions about caries rates, although the data (Table 134) do show that caries was not confined to any single mortuary phase. In all except the child from MP6, the caries in permanent teeth were associated with defective enamel.

TABLE 130: *Variation over time in degeneration of the vertebral synovial joints. Data represent all grades of degeneration*

	Cervical		Thoracic				Costal		Lumbar	
	N ¹	n ²	Upper N n		lower N n		N n		N n	
Females										
MP1	1	-	-	-	-	-	1	-	-	-
MP2	11	4	9	5	11	4	11	5	9	4
MP3	7	4	8	3	9	3	9	2	7	2
MP5-6	5	2	3	-	3	1	3	1	3	1
Males										
MP1	1	-	-	-	1	1	1	1	1	-
MP2	7	5	7	4	7	3	7	5	7	4
MP3	10	4	9	4	10	5	10	4	8	2
MP4	8	4	8	3	8	3	8	4	8	4
MP5-6	1	1	1	-	1	1	1	-	1	-

¹ Number of individuals with at least one of the joints present² Number of individuals with degeneration of at least one joint

The numbers and proportions of individuals with at least one tooth excessively worn are summarised for both sexes by age at death and mortuary phases in table 135. These data indicate that, although comparatively more females than males were affected by excessively worn teeth in most mortuary phases, comparatively few teeth per individual were involved. The males with worn teeth in MP1-3 had a high proportion of their teeth affected. The pattern changed in MP4 with relatively few individuals of either sex affected and by MP5 no-one had excessively worn teeth.

Table 136 summarises the variation over time in the prevalence of periodontal disease by sex. In both sexes, the proportion of individuals with evidence of periodontal disease is higher in MP 1-3 than in the later mortuary phases. Both porosity and resorption, and pocket formation, are particularly common in MP2. The proportions are similar in both males and females.

The temporal variation in formation of periapical cavities in the alveolar bone is summarised by sex in table 137. Apart from the males in MP5-6, a high proportion of individuals of both sexes suffered from periapical inflammation in all mortuary phases, with the highest proportion in MP2-3 in the females and MP3-4 in the males. The mean number of periapical cavities per individual was highest in MP3 in the females and lowest in MP5-6 and in the males was highest in MP2 and 3 and lowest in MP5-6. The numbers of periapical cavities were similar in both sexes.

The underlying pathology which provides the route for the infection is summarised, by age at death and mortuary phase, in table 138. In female dentitions, caries was the most common cause of periapical inflammation in MP2, superseded by wear in MP3 and 4. In the male dentitions, the

TABLE 131: *Variation over time in the degeneration of vertebral cartilaginous joints. Data represent all grades of degeneration*

	Cervical		Osteophytosis						Compression/Collapse					
			Phytosis		Porosity		Upper		Thoracic		Lower		Lumbar	
	N ¹	n ²	N	n	N	n	N	n	N	n	N	n	N	n
Females														
MP1	-	-	1	-	-	-	-	-	-	-	-	-	-	-
MP2	10	2	11	8	10	1	10	5	11	9	11	1	11	2
MP3	7	3	7	3	9	5	9	7	9	7	9	2	9	2
MP4	10	6	10	2	10	4	10	5	10	7	10	1	10	1
MP5-6	5	3	5	-	5	1	4	2	4	2	5	1	5	-
Males														
MP1	1	-	1	-	-	-	1	1	2	1	1	1	1	1
MP2	7	5	7	6	7	2	7	5	7	5	7	4	7	3
MP3	10	4	10	6	10	7	10	8	10	8	10	7	10	8
MP4	8	4	8	3	8	1	8	6	8	5	8	3	8	1
MP5-6	1	-	1	-	1	-	1	-	2	2	1	-	1	-

¹ Number of individuals with at least one of the joints present² Number of individuals with degeneration of at least one joint

pattern is the reverse, with wear the most common cause of periapical inflammation in MP1-3 and caries in MP4.

Table 139 summarises tooth loss during life for each mortuary phase. Loss rates were higher in females than in males in all phases. Not only were more females affected but the proportion of teeth lost by each individual was higher. Differences over time were not significant.

SUMMARY OF VARIATION IN HEALTH BY MORTUARY PHASE

Mortuary phase 1

This mortuary phase is represented by six individuals. There was no defined burial area and the distribution of the graves did not provide any evidence as to social relationships among the individuals. The earliest burial, 17 cm above the natural deposits, was a newborn infant. This was followed by the burial of an adult female (B154), a second neonate, an adult male (B152) and a 2 year-old infant (B150). The last burial in the phase, 63 cm above the first, was a young adult male (B147).

B150 was the earliest infant in the sample to die with anaemia. The three adults in the sample were relatively tall, although two of them had evidence of growth disruption in childhood. The

TABLE 132: *Variation over time in the degeneration in the appendicular joints of adults*

	Shoulder		Elbow		Wrist		Hand		Hip		Knee		Ankle		Foot	
	N ¹	n ²	N	n	N	n	N	n	N	n	N	n	N	n	N	n
Females																
MP1	-	-	1	-	-	-	-	-	-	-	1	1	1	-	1	1
MP2	8	5	10	5	10	4	8	3	8	4	10	7	10	3	7	1
MP3	9	6	9	7	8	5	8	4	8	1	9	5	9	6	7	5
MP4	9	3	10	2	8	2	7	3	7	2	9	3	10	3	10	4
MP5-6	5	1	4	1	4	-	4	1	3	-	3	2	3	1	4	3
Males																
MP1	2	1	2	-	1	-	2	1	1	-	2	1	1	-	1	-
MP2	7	7	7	3	6	3	7	4	5	-	7	4	7	5	6	5
MP3	8	7	10	6	8	5	8	5	10	4	10	8	8	5	8	6
MP4	8	2	9	2	8	2	8	2	8	1	5	-	7	3	7	5
MP5-6	-	-	1	1	1	-	1	-	1	-	1	1	1	-	1	-

¹ Number of individuals with at least one of the joints present² Number of individuals with degeneration of at least one joint

stature suggests that the episodes of growth disruption were short term and recovery was good with no resulting long-term effect on growth. Both males had very good cortical thickness, although the thickness of B152 bones may be pathological. The long bones of the young B147 were not robust. The female had borne a child or children. Despite this picture of good health, B147 and the female died at a relatively early age. The older male had degeneration of the shoulders, lower spine and knees, although his muscle attachments were not well marked. Dental problems started early in life. All three adults had calculus on their teeth and evidence of periodontal disease, conditions which are often related. Caries were a minor problem. The female had already lost teeth from an unknown cause. The teeth of the older male had worn to a level that exposed the pulp and periapical inflammation had resulted.

Mortuary phase 2

This was the first mortuary phase with graves in the discrete areas (clusters) which continued through to MP4. Each cluster includes infants and adults of both sexes. The individuals within the clusters are possibly related.

The sample totals 56 individuals in deposits of a similar depth to MP1. It is the largest sample from any mortuary phase and the most densely concentrated. It has the highest level of subadult mortality, with two-thirds dying in infancy or childhood. A large proportion (21) died at or soon after birth and a further 10 within the first twelve months, making a total of over half (31 = 55 per cent) who did not survive the first year of life. This is an extreme level of early infant mortality. In

contrast the deaths of older infants or children are comparatively low. Of the 19 adults, eleven are females and eight males.

TABLE 133: *Variation over time in caries in adults*

	Individuals ¹		Teeth ²			Means per individual ³		
	N	With caries n	N	Carious n	%	Present	Carious	%
Females								
MP1	1	0	24	0	0	-	-	-
MP2	11	10	216	38	18	21.5	3.8	18
MP3	9	6	187	25	13	21.2	4.2	20
MP4	10	4	161	13	8	16.0	3.3	20
MP5-6	5	4	69	20	29	13.5	5.0	37
Males								
MP1	2	1	38	1	3	14.0	1.0	7
MP2	8	5	192	9	5	26.8	1.8	7
MP3	10	6	227	25	11	24.8	4.2	17
MP4	8	3	119	8	7	12.7	2.7	21
MP5-6	3	0	49	0	0	-	-	-

¹The number of individuals included in the sample and the number with one or more carious teeth

²The number of teeth assessed for caries and the number and proportion of teeth with caries

³The mean number of teeth present and the mean number of teeth which were carious in those individuals with one or more caries

Four infants have evidence of anaemia. Growth disruption was common in the deciduous dentition, with the defective enamel resulting in decay in several cases.

The adults had all suffered growth disruption during childhood, with infancy and the age of 10–14 years the most susceptible periods. More of the females had episodes during adolescence than the males and the females were affected more often. A higher proportion of the males had non-linear dental enamel defects. The long-term effect on stature of the growth disruption appears to have affected males more than females, with a relatively low level of sexual dimorphism. Bone cortices were good in the females but comparatively thin in the males. Overall, the evidence is that adults in MP2 had suffered some illness in childhood but this had less effect on the females than the males. All the women bore children, with the exception of a 17-year-old, B140. On average, females lived longer than the males and longer than in subsequent phases.

Joint degeneration in the women was most prevalent in the upper limb, the knees and the discs of the cervical spine. A smaller proportion had problems in the upper thoracic spine and the shoulders. A high proportion of the males also had joint degeneration involving the neck, upper back and shoulders. In many cases, the degeneration involved the entire shoulder girdle. In several, the lower back was also affected, with compression and collapse of vertebrae in almost half of the males. The

hands and the major joints of the lower limbs were also commonly affected. It is clear that a proportion of individuals, particularly males, were engaged in an activity which involved the whole body with particularly vigorous use of the upper body and spine. The different pattern of degeneration in the females suggests sexual division of labour.

Degeneration of the dentition affected most of the females. A majority had caries, many of which resulted in periapical inflammation, and rates of antemortem loss of teeth were high. Periodontal disease was also common. In contrast the males had low rates of caries but their teeth were more worn. Despite the abrasive diet suggested by the tooth wear, periodontal disease was common. It included periapical inflammation which probably accounted for moderate rates of tooth loss during life. The sex differences suggest males and females had different diets from childhood.

TABLE 134: *Variation over time in caries in children*

	Deciduous		Permanent	
	N ¹	n with caries	N	n with caries
MP1	1	1	0	0
MP2	8	3	1	0
MP3	1	0	0	0
MP4	4	1	5	3
MP5-6	4	1	2	0
MP7	0	0	2	1

¹ Number of individuals with teeth erupted

Mortuary phase 3

Graves were again clustered as in MP2. One cluster (Cluster E) is not represented in this mortuary phase but as it is located by the southern baulk, it is likely to have continued with graves outside the excavation area. Another cluster (Cluster B) is represented by two infant burials only and does not continue beyond this mortuary phase. Although the group it represents may have died out, the graves are again by the baulk and the cluster may continue beyond the excavation area.

The burials are less densely concentrated than in the previous phase, with 42 individuals in deposits which were one third deeper than those of MP2. Almost half (20) are newborn babies, and a further three died in infancy but there were no older children. The neonatal mortality rate is the highest in any phase at 48 per cent, but the rate for older infants is low, indicating that almost all who survived birth also survived the exigencies of infancy and childhood. The sample has the same number of adults as the previous phase, comprising nine females and ten males.

No evidence of anaemia was found on any of the infants but three adults have *cribra orbitalia*. The only female classified as anaemic, B56, is buried in this mortuary phase. Despite having anaemia, she survived to the age of 45 years and was fertile.

All adults had at least one episode of growth disruption during infancy, but during adolescence the patterns of Harris lines and non-linear enamel hypoplasia is the reverse of that in the preceding phase. Sexual dimorphism in stature was higher during this mortuary phase than at any other time, mainly because the males were relatively taller and more robust than at other times. This was also reflected in the highest index of cortical thickness of any period in the males. Mean ages at death of both sexes were similar to the previous phase. All indications are that the health of the males was relatively good in this mortuary phase but the health of the females did not vary greatly from that in other phases.

Joint degeneration in both sexes was similar in frequency and distribution to that in the previous phase, suggesting a continuation of the patterns of physical activity. In contrast with the previous mortuary phase, both sexes suffered similar patterns of periodontal disease. There were moderate-high rates of all forms of dental pathology, resulting in loss of teeth during life.

TABLE 135: *Variation over time in excess tooth wear*

	Individuals ¹ With excess wear		Teeth ² Worn			Means per individual ³ Teeth		
	N	n	N	n	%	Present	Worn	%
Females								
MP1	1	0	23	0	0	-	-	-
MP2	11	5	200	7	3	10.8	1.4	13
MP3	9	4	188	17	9	15.5	4.3	28
MP4	10	3	167	11	7	12.0	3.7	31
MP5-6	5	0	66	0	0	-	-	-
Males								
MP1	2	1	39	11	28	15.0	11.0	73
MP2	8	2	199	24	12	18.5	12.0	65
MP3	10	3	224	39	17	19.3	13.0	67
MP4	8	2	134	3	2	19.5	1.5	8
MP5-6	3	0	32	0	0	-	-	-

¹The number of individuals included in the sample and the number with one or more worn teeth

²The number of teeth assessed for excess wear and the number and proportion of teeth excessively worn

³The mean number of teeth present and the mean number of teeth which were worn in those individuals with one or more excessively worn teeth

Mortuary phase 4

The burials continue to be concentrated in the clusters, although the density of graves is again lower than in the previous phase. There are 29 individuals in one metre of deposits. Cluster B is not represented.

Infant mortality is lowest in this phase. There were two neonate deaths, two other infants who did not survive the first year, and one who died before the age of five. This is a total of 17 per cent infant mortality, about one third of the levels in the previous phases. This is very low for a prehistoric society. In contrast, there are five deaths of children aged 5–14 years, representing a further 17 per cent of the sample. This is a high rate of child mortality and compares with a total of two deaths in this age group of the 104 burials in the previous three phases. The total subadult mortality is lowest of any phase. The possibility of sampling error or a demographic change as an explanation has been discussed briefly in chapter II. There is no change in mortuary ritual or morphology which could indicate an alteration in the genetic composition of the group through immigration. An alternative possibility is a change in the attitude towards the disposal of infant bodies, resulting in culturally determined sampling error in the burials recovered from the excavation. Very young infants may have been buried in a separate area, or disposed of without ceremony. The presence of at least some infants, including neonates, among the adult burials, shows that if such a practice was acceptable it was not universally adopted.

There is also the possibility that there was a dramatic change in the health of neonates with a resulting alteration in the pattern of child mortality. An amelioration in the cause of neonatal death could result in the least fit becoming able to survive birth and infancy, only to succumb to ill health later in childhood. This would result in a secondary demographic factor contributing to the drop in deaths of infants through a reduction in fertility. The longer survival of children would have resulted in longer birth intervals and lower birth rates, with a compounding effect on the reduction of the potential infant mortality rate. One of the children had evidence of severe anaemia, which may have been the ultimate cause of death.

In the adults, the relative health of the sexes reverses compared with MP3. Sexual dimorphism in stature is at its lowest level, mainly because of a reduction in male stature. This is the only phase in which there are individuals without skeletal evidence of growth disruption in infancy, which strengthens the suggestion that the low level of infant mortality reflected a genuine improvement in infant health. A low proportion of the males had Harris lines formed at that age, although there was no associated reduction in dental enamel hypoplasia. The presence of enamel hypoplasia suggests that the paucity of Harris lines reflects chronic, low-level ill health with relatively few periods of recovery, rather than good health. A relatively high proportion of females had evidence of growth disruption in mid-childhood (5–9 years) compared with other mortuary phases. As in MP3, more males than females had Harris lines formed during adolescence, but the pattern of a negative correlation with non-linear dental defects no longer holds as more males were affected than females. Childhood health appears to have had less long-term effect on the stature of the females than the males, possibly reflecting the length of periods of growth disruption and the ages at which growth was disrupted. Despite this, cortical thickness was good in the males but low in the females. Both sexes died on average at an earlier age than in earlier phases, although this was more obvious in the males than in the females. Overall, male health declined compared with the previous mortuary phase but females were as healthy as previously. One male (B24) had signs of one of the haemoglobinopathies.

Joint degeneration affected fewer individuals than in earlier mortuary phases and was concentrated in the joints of the lower back with relatively little involvement of major limb joints. Degen-

eration was still more common and more complex in the males than in the females. Dental degeneration was also less prevalent. Caries rates and the prevalence of periodontal disease were relatively low in both sexes compared with earlier phases. Periapical inflammation and loss of teeth during life were at moderate levels in females but low levels in males. The infections more commonly the result of exposure of the pulp cavity through wear than because of caries as in earlier phases.

Mortuary phases 5-7

The burials in these mortuary phases were no longer distributed in clusters and the grave density was lower than in MP4. Some other aspects of the mortuary practices varied from the earlier pattern but there were also continuities. Despite the changes, there is no reason to believe that the cemetery was not in virtually continuous use from MP4 by the same group. The sample from MP5-7 contains 21 individuals. There are four burials in MP5, two infants, a female and a male. The placement of the graves and treatment of the burials suggests that the infants were related to the female. The male was buried some distance away. All graves were elaborate and wealthy. In MP6 there are twelve burials, four females, two males, four infants and two children. Three of these, two females and a child, are buried within an elaborate mortuary structure. Eight (four infants, two females and two males) are buried in a row of parallel graves and the remaining burial, a child, is in the south-east corner of the square. In MP7 there are five burials, two infants and three children. Two are in a single grave above the mortuary structure and three form a small cluster with one burial from MP6 in the south-eastern corner. Census totals are eight infants, five children, five females and three males.

Total infant and child mortality returns to the high levels of MP1-3 but the age composition of the sample is very different from the early phases. As in MP4, there are relatively few neonates or very young infants. The mortality rate of older infants and children is even higher than in MP4, with this age group forming almost half of the sample. Two of the five children have evidence of anaemia, the probable primary cause of death.

Adults of both sexes were shorter and smaller than in earlier phases. Sexual dimorphism in stature was higher than in MP4, a reflection of a greater reduction of size in the females than in the males. All adults had dental evidence of growth disruption, although the number of episodes was low compared with previous phases. Both males and one of the three females had non-linear enamel hypoplasia. On the evidence of stature, childhood health in MP5-6 appears to have declined in both sexes relative to the earlier phases, although this assessment is on the basis of a small sample. Despite this, the females had significantly thicker cortices than in MP4. The females survived to a similar age as in earlier phases but the males died earlier.

Skeletal and dental degeneration was minimal in the males, although this would be partly a reflection of their early deaths. In the females, joint degeneration was similar to previous phases, affecting the spine, knees and feet. Dental caries and loss of teeth during life were at high levels in the females. None of the adults had excessively worn teeth and periodontal disease was minimal.

TABLE 136: *Variation over time in periodontal disease*

	n ¹	Porosity/resorption ²		Pockets ³	
		n	%	n	%
Females					
MP1	1	1	100	0	0
MP2	11	11	100	6	55
MP3	9	7	78	2	22
MP4	10	3	30	2	20
MP5-6	5	1	20	0	0
Males					
MP1	2	2	100	1	50
MP2	9	8	89	5	56
MP3	10	8	80	3	30
MP4	8	2	25	0	0
MP5-6	3	1	33	0	0

¹Number of individuals included in the sample

²Number and proportion of individuals with evidence of periodontal disease in the form of porosity and/or resorption of the bone on the alveolar crest

³Number and proportion of individuals with evidence of periodontal disease in the form of infrabony pockets and the mean number of teeth affected per individual

IMPLICATIONS OF THE CHRONOLOGICAL VARIATION IN HEALTH

The sample from MP1 suggests that the haemoglobinopathy was present at Khok Phanom Di from the beginning of the settlement. It was the cause of death of at least one child in infancy in this phase. Despite the anaemia, other aspects of health were good during this early period of the settlement. Nutrition during childhood was more than adequate. The anaemia may have been implicated in the early death of two of the three adults. Adults were engaged in physical activities which were similar to those in later mortuary phases, although probably not as vigorously pursued. Both sexes were eating a diet which was abrasive but not cariogenic.

In MP2 the group coped with a high rate of neonatal mortality, the deaths of first-year infants with haemoglobinopathy, and universal periods of childhood illness. Individuals who survived the first year of life almost all reached adulthood, although the episodes of illness in childhood had a greater long-term effect on males than females. This did not prevent adults leading a vigorous, active life. The infant mortality would have represented a high cost to the females, particularly in the case of infants who survived birth only to die after a few months during which their mothers would have made a significant input into lactation. Despite this, females survived longer than the males on average. Physical activities appear to have been particularly vigorous with degeneration

TABLE 137: *Variation over time in periapical cavities*

	Individuals with pc		Teeth with pc			Means per individual Teeth		
	N	n	N	n	%	Present	with pc	%
Females								
MP1	1	0	28	0	0	-	-	-
MP2	11	8	333	27	8	30.5	3.4	11
MP3	9	7	288	34	12	32.0	4.9	15
MP4	10	4	308	15	5	29.0	3.8	12
MP5-6	5	2	146	2	1	30.0	1.0	3
Males								
MP1	2	1	58	2	3	26.0	2.0	7
MP2	8	3	254	11	4	32.0	3.7	11
MP3	10	7	319	23	7	32.0	3.3	10
MP4	8	5	232	8	3	27.4	1.6	5
MP5-6	3	0	81	0	0	-	-	-

¹ periapical cavities

common in the spine and limbs in both sexes. Nevertheless there appears to have been some sexual division of labour with degeneration of the upper spine and shoulder girdle in particular in several males. Sex differences in dental pathology suggest that diet also differed between the sexes, with the females eating food which was more cariogenic and less abrasive than that eaten by the males.

In MP3, the extreme level of neonatal mortality would again have placed a high physiological demand on the females, although without the cost of lactation. A high proportion of the women appear to have had to contend with the drain of multiple pregnancies. Nevertheless they survived as long as the men. In this phase the pattern of evidence for anaemia changes, with no evidence on infants but at least one adult affected and possibly three others. Despite this, and universal childhood growth disruption, this phase represents a high point in the health of the men at Khok Phanom Di. They were tall and robust and appear to have led a vigorous, active life. Physical activities were similar in pattern to the previous phase. Dental health was similar in both sexes in this phase, suggesting that both were eating the same diet. The diet was less abrasive and moderately cariogenic.

The apparent reduction in neonatal and early infant mortality between MP3 and MP4 remains a matter of conjecture. The increase in deaths of older children in MP4 represents a substantial loss to the community, with both the physiological cost to the mothers and the community cost of nurturing children who did not reach the age of productivity. Adults appear to have been less physically active than previously, although they died at an earlier age, giving less opportunity for skeletal degeneration. The condition of adult dentitions suggests a change in diet to one which was more abrasive, with lower caries rates and less periodontal disease in both sexes.

TABLE 138: *Variation over time in the causes of periapical cavity formation*

	n	Females			n	Males		
		C ¹ %	P ² %	W ³ %		C %	P %	W %
MP1	0	0	0	0	2	0	0	100
MP2	13	92	8	0	11	9	18	73
MP3	19	32	5	63	18	39	6	55
MP4	9	33	0	67	5	60	40	0
MP5-6	2	50	0	50	0	0	0	0

¹ caries, ² periodontal disease, ³ wear

By MP5-7, the community was bearing a high cost of raising individuals who died before reaching maturity. As this period represents the end of the use of the area of the excavation as a cemetery, the high proportion of child deaths is partly the effect of the absence of any adult burials in the final 95 cm of deposits. The parents of these children were either buried elsewhere on the site or had left Khok Phanom Di. As stated in chapter I, there is no sedimentological evidence of a hiatus in the occupation of the site at the end of MP7. With the change in burial practices represented by the discontinuation of clustering of interments in groups of mixed ages, the segregation of the cemetery cannot be discounted. Nevertheless, despite the apparent wealth in material terms, health was relatively poor with adults of both sexes shorter and less robust than earlier. The decline in health may have reflected a change which provided the motivation for at least some of the occupants to move away from the site. The decline affected the females less than the two males, who died at a relatively early age. Females continued to have been engaged in physical activities similar to those in earlier phases but the males were relatively inactive. The poor dental health of the females suggests a sexually differentiated diet, although this and the sex differences in skeletal degeneration may be sampling error.

Interpretation of variation in health

The major features of change over time, the variation in morbidity and mortality patterns of infants and children, the changes in diet suggested by dental pathology and in physical activities suggested by degenerative joint disease, and the overall deterioration of health, exemplified by the males, largely coincide with the end of MP3 and the beginning of MP4. The environmental changes discussed in chapter I are most evident within the period of deposition of MP4 (Mason 1991, Thompson 1996). Although the environmental changes would have taken some time to have a visible effect on the people, the coincidence with the changes in health and 'lifestyle' is persuasive. The potential consequences for health of the environmental change could be through the diet, exposure to pathogens, and physical activity patterns. The possible effects are discussed in the following sections.

TABLE 139: *Variation over time in antemortem tooth loss*

	Individuals ¹ With tooth loss		Teeth ² lost			Means per individual ³ Teeth		
	N	n	N	n	%	Present	lost	%
Females								
MP1	1	1	28	3	9	32.0	3.0	9
MP2	11	9	333	59	22	30.4	6.6	22
MP3	9	6	288	42	22	32.0	7.0	21
MP4	10	4	308	20	17	28.8	5.0	17
MP5-6	5	4	146	18	15	30.5	4.5	14
Males								
MP1	2	0	58	0	0	-	-	-
MP2	8	3	254	14	5	31.7	4.7	14
MP3	10	3	319	11	3	32.0	3.7	11
MP4*	8	3	232	15	6	26.7	5.0	19
MP5-6	3	1	81	1	1	23.0	1.0	4
if B28 is excluded:								
MP4	7	2	216	3	1	32.0	1.5	5

¹The number of individuals with one or more teeth lost²The number of teeth assessed for loss and the number and proportion lost³The mean number of teeth assessed for loss and the mean number lost in those individuals with one or more teeth lost

Diet and nutrition

The changes over time in the archaeological evidence for diet and the consequences for nutrition were discussed in chapter VII. The possible deterioration in nutritional quality could have contributed to the general deterioration in health. High protein needs during weaning in particular may not have been met, with the interaction of reduced nutrition with increased exposure to pathogens at this age contributing to the relatively high mortality rate of older infants during MP5-7. The relatively short stature of adults may also reflect the long-term effects of this interaction.

The changes over time in the dental evidence of diet are firstly from a relatively abrasive, often cariogenic, diet which also caused periodontal disease in MP1-3, to one which was equally abrasive but less cariogenic and less likely to cause periodontal disease in MP4. In MP5-7 the diet was no longer abrasive and did not cause periodontal disease but was cariogenic. This is ignoring sex differences for the moment and assuming that the dental conditions reflected dietary changes rather than any change in dental hygiene. The detailed analysis of dental evidence for diet is not part of this study, but in very general terms the pattern is consistent with the MP1-3 diet including relatively abrasive material such as sand with shellfish or fibrous vegetable foods together with plaque-forming foods which were rich in carbohydrates such as rice and other starchy foods or fruit. In MP4

the diet continued to include the abrasive foods but less of the plaque-forming foods. In MP5-7 the diet was softer, perhaps reflecting the lower quantity of shellfish. The reduction in abrasive foods over time correlates with the reduction in shellfish shells in the excavation area deposits, although the association can only be verified through further research such as analysis of microwear on the teeth or chemical analysis of bone composition.

Disease

The postulated environmental change would have had the possibility of altering the epidemiology of malaria. The habits of different species of mosquito, including the range, biting habits and preferred host species, vary greatly. The change in the proximity and nature of standing bodies of water around Khok Phanom Di could have affected either the number of mosquitoes present through a reduction in breeding area or resulted in a change in the species of *Anopheles*. The suggestion that the numbers of terrestrial mammals were higher in the later mortuary phases also raises the possibility of a change in exposure of humans from a mosquito species with an attraction to other mammals. There may also have been a change in the species of *Plasmodium* with a change in *Anopheles*.

The complexity of the interaction of the parasite, vector and host means that there are multiple scenarios which could be suggested, with the effect of either increasing or reducing the endemicity or virulence of malaria. There is no point in considering all the possibilities in detail, but a reduction in the exposure of the population to the disease is consistent with the improvement in the infant mortality rate from MP4. It can be speculated that this increase in survivorship to childhood included haemoglobinopathics who then succumbed to the primary or secondary effects of the disease as older children or young adults, accounting for the change in the mortality pattern of subadults.

The effect of an environmental change on the less significant pathogens affecting the population is also complex and does not warrant further discussion, given the degree to which evidence of their presence, as discussed in chapter VII, is circumstantial.

Physical activity - 'lifestyle'

The postulated deterioration in accessibility of the river with the consequent loss of easy access to the coast and the interior would have resulted in a gradual but extreme change in the activities of the community. The reduction from MP4 of the prevalence of upper body joint degeneration in particular is consistent with the reduction in opportunities for canoeing, although life appears to have been less physically active overall. To stretch the speculation further, it is also consistent with a reduction in the relative status of the males and, consequently, their well-being.

TABLE 140: *Variation in cranial and facial measurements over time*

	MP1-3			MP4-6			Regr.*	Nonp.*
	n	Mean	s.d.	n	Mean	s.d.	p-value	p-value
Females								
Maximum cranial length	12	170.33	5.76	3	163.67	13.05	-	-
Maximum cranial breadth	11	135.36	4.72	4	133.25	4.79	-	-
Maximum cranial height	10	134.70	5.89	2	131.00	2.83	-	-
Minimum frontal breadth	15	93.03	5.17	8	95.28	4.97	-	-
Bizygomatic width	11	131.55	4.82	4	128.00	6.38	-	-
Bimaxillary width	13	104.35	5.07	5	99.04	3.23	-	.034
Inter orbital width	14	25.18	2.33	7	25.61	2.71	-	-
Upper facial height	13	67.76	3.22	1	63.00	-	-	-
Nasal height	15	49.53	2.39	3	49.27	2.06	-	-
Nasal breadth	13	27.55	1.33	5	25.46	1.46	<0.01	.018
Palatal breadth	11	65.54	3.89	4	63.92	3.31	-	-
Palatal length	11	42.46	1.89	2	44.20	1.13	-	-
Basion-prosthion length	9	97.32	5.67	0	-	-	-	-
Cranial base length	10	96.10	4.75	2	94.00	5.70	-	-
Males								
Maximum cranial length	5	179.00	5.10	2	183.50	2.12	-	-
Maximum cranial breadth	5	145.00	0.71	2	147.00	4.24	-	-
Maximum cranial height	4	144.25	4.27	1	147.00	-	-	-
Minimum frontal breadth	9	101.82	5.00	5	99.30	2.96	-	-
Bizygomatic width	4	145.25	4.27	1	150.00	-	-	-
Bimaxillary width	6	108.67	4.97	3	114.33	0.58	-	-
Inter orbital width	5	27.68	1.61	3	27.93	0.12	-	-
Upper facial height	4	73.38	1.16	0	-	-	-	-
Nasal height	4	54.18	1.05	4	52.08	3.71	-	-
Nasal breadth	6	27.93	1.08	4	28.00	2.03	-	-
Palatal breadth	8	69.50	1.76	3	67.13	3.59	-	-
Palatal length	6	46.13	1.13	3	46.93	0.31	-	-
Basion-prosthion length	4	101.25	0.96	0	-	-	-	-
Cranial base length	3	101.33	2.89	2	102.00	8.50	-	-

Regr.*: univariate regression; Nonp.*: nonparametric Mann-Whitney U-test

TABLE 141: *Variation over time in mandibular measurements*

		MP1-3			MP4-6			Regr.*	Nonp.*
		n	Mean	s.d.	n	Mean	s.d.	p-value	p-value
Females									
	Length	16	104.19	4.35	14	102.71	5.21	-	-
	Bicondylar breadth	14	122.44	5.24	13	123.87	4.45	-	-
	Bigonial breadth	17	92.30	4.72	14	92.89	4.08	-	-
	Ramus breadth	19	35.88	2.45	15	34.06	2.66	-	0.066
	Symphyseal height	14	31.62	2.37	2	29.50	2.83	-	-
	Body height	14	30.59	2.40	11	30.48	2.57	-	-
	Bimandibular foramen breadth	17	80.38	3.85	14	83.41	3.62	<0.05	-
	Bimental breadth	18	47.13	2.22	13	49.22	1.78	<0.02	0.0073
	Ramus height	19	59.64	5.67	15	60.16	5.34	-	-
	Gonial angle	19	116.58	6.24	14	119.43	5.33	<0.05	-
Males									
	Length	16	109.12	3.63	7	105.86	4.49	-	-
	Bicondylar breadth	16	131.48	6.78	6	127.62	7.17	-	-
	Bigonial breadth	16	101.26	7.24	6	103.95	6.14	-	-
	Ramus breadth	17	37.13	2.90	7	34.46	2.08	<0.05	0.0361
	Symphyseal height	15	34.01	2.44	5	34.42	3.35	-	-
	Body height	17	32.20	2.64	7	31.24	1.64	-	-
	Bimandibular foramen breadth	16	87.57	6.39	7	84.97	5.56	-	-
	Bimental breadth	16	49.31	1.74	7	49.44	2.72	-	-
	Ramus height	16	66.74	5.48	7	63.11	4.14	-	-
	Gonial angle	16	113.19	5.49	7	118.00	2.31	-	0.045

Regr.*: univariate regression; Nonp.*: nonparametric Mann-Whitney U-test

TABLE 142: *Variation over time in dental measurements
(mean, left and right sides mesio-distal x bucco-lingual index)*

		MP1-3			MP4-6			Regr.*	Nonp.*
		n	Mean	s.d.	n	Mean	s.d.	p-value	p-value
Females									
Maxilla	Central incisor	7	62.66	6.80	0	-	-	-	-
	Lateral incisor	7	43.78	1.86	2	55.15	2.02	<0.02	0.0001
	Canine	6	58.05	2.81	9	65.23	6.99	<0.05	0.034
	First premolar	11	69.99	6.97	7	69.93	8.74	-	-
	Second premolar	9	64.20	9.38	8	65.60	9.60	-	-
	First molar	8	120.92	8.75	9	126.99	12.50	-	-
	Second molar	16	113.43	13.55	9	116.08	15.97	-	-
	Third molar	11	96.37	17.44	7	98.64	14.21	-	-

TABLE 142: *Variation over time in dental measurements (mean, left and right sides mesio-distal x bucco-lingual index) (cont.)*

		MP1-3			MP4-6			Regr.*	Nonp.*
		n	Mean	s.d.	n	Mean	s.d.	p-value	p-value
Females									
Mandible	Central incisor	4	33.10	4.97	1	36.30	-	-	-
	Lateral incisor	7	37.59	3.79	2	36.05	5.09	-	-
	Canine	7	52.01	5.05	8	53.00	7.34	-	-
	First premolar	15	58.11	4.37	9	58.53	6.47	-	-
	Second premolar	17	63.28	6.93	8	62.10	9.05	-	-
	First molar	12	126.20	9.95	9	133.66	10.57	-	-
	Second molar	13	112.85	12.26	10	113.52	12.22	-	-
	Third molar	10	116.02	17.23	6	119.11	18.78	-	-
Males									
Maxilla	Central incisor	6	68.38	7.92	0	-	-	-	-
	Lateral incisor	2	52.78	3.57	2	53.62	9.51	-	-
	Canine	9	69.31	4.90	4	63.52	7.07	-	-
	First premolar	11	69.96	7.02	5	72.34	6.68	-	-
	Second premolar	11	63.07	6.14	4	62.42	6.12	-	-
	First molar	11	127.60	10.06	3	140.43	5.91	<0.05	0.0603
	Second molar	13	111.74	9.30	5	121.35	10.98	-	0.0793
	Third molar	10	101.47	10.41	8	109.82	11.35	-	-
Mandible	Central incisor	8	34.24	3.25	3	36.86	1.08	-	-
	Lateral incisor	10	40.08	4.75	3	45.32	1.69	<0.05	0.0946
	Canine	11	59.22	4.49	5	56.73	6.97	-	-
	First premolar	14	60.74	6.49	8	64.47	7.37	<0.05	-
	Second premolar	12	60.66	5.11	8	64.44	6.25	-	-
	First molar	9	127.16	6.94	5	135.37	7.17	<0.10	0.0576
	Second molar	13	115.92	9.00	7	116.04	9.67	-	-
	Third molar	12	116.41	9.22	10	116.62	11.71	-	-

*Significance: Mann-Whitney U-test

TABLE 143: *Variation over time in a selection of postcranial dimensions*

		Right						Left					
		MP1-3			MP4-7			MP1-3			MP4-7		
		n	Mean	s.d.	n	Mean	s.d.	n	Mean	s.d.	n	Mean	s.d.
Females													
Humerus													
Head max. diameter		16	40.1	2.0	10	40.4	2.3	18	39.7	1.7	13	39.8	2.4
Length		19	287.4	10.4	10	286.8	15.8	16	285.9	12.4	10	285.3	15.7
Shaft circumference		18	61.3	3.8	4	61.0	4.6	17	61.7	4.8	4	60.9	4.8
Radius													
Length		18	227.3	10.4	9	226.0	11.4	16	226.8	11.2	12	228.7	10.8

IX. CONCLUSIONS

IT would be satisfying to be able to answer the question 'were the the people of Khok Phanom Di healthy?' with a simple 'yes' or 'no' but that would deny the complexity of the evidence, including changes over time. Firstly, ignoring the temporal changes, the conclusions are considered for the group as a whole. By some measures their health was poor. They had high levels of infant mortality and childhood morbidity. Many failed to achieve their growth potential. In adulthood they had poor dental health, joint degeneration was common and life expectancy was short. Anaemia was common in children and in some cases severe. They appear to have led a physically active life from an early age and to have been exposed to a variety of debilitating and often fatal pathogens. This gloomy picture suggests that survival was difficult for them, both individually and as a group, and that life was hard. Yet despite all these factors, adult bone structure was good and women were fertile. It appears that, for most of the period represented by the seven mortuary phases, sufficient natural resources were available to help counteract the difficulties of survival. Their diet appears to have been balanced and the food to have been available in sufficient quantities, perhaps with the exception of seasonal shortages, to have provided them with good nutrition. This may have been the factor which enabled enough individuals to withstand the effects of episodic poor health during childhood to reach adulthood and provide for the survival and maintenance of the group. Good nutrition may also have been the explanation for the maintenance of good bone structure in adulthood.

The picture becomes more complex when changes over time are taken into account. The very high levels of neonatal and infant mortality during the early mortuary phases (MP1-3) have already been discussed in some detail. However, during this period those who survived beyond early infancy were particularly successful in enduring the episodes of childhood morbidity to reach adulthood. The adult females were healthy enough to maintain fertility despite the high cost of infant mortality. The adult males were robust and muscular and appear to have been physically very active. The population was able to continue their occupation of the site and had sufficient resources, beyond the energy required for subsistence, for other activities such as the development of the skills and investment of time in the manufacture of high-quality ceramics. In the later mortuary phases (MP4-7), there was an apparent improvement in the survival of infants but this was counteracted by higher levels of child mortality. The children almost all suffered from anaemia. Childhood morbidity continued to be almost universal and appears to have not only been more often fatal but also to have had a greater long-term effect. There is also a suggestion of a decline in adult health which is statistically unconfirmed but nevertheless evident in some measures of health. Males were less physically active and lived shorter lives. Nevertheless, the manufacture of pots continued and the interpretation of the distribution of grave goods, as discussed in chapter I, suggests that the pots and the individuals involved in their manufacture may have become more important in the survival of the community (Bannanurag 1989). The correlation of the timing of the change in health between the early and the late mortuary phases with the postulated environmental change has been discussed

in chapter VIII. In MP7, the final phase of the cemetery, there were no burials of adults. As also discussed in chapter VIII, the reason for this and for the subsequent discontinuation of burials in the area of the excavation is a question which remains unresolved. Whether there was a relationship with the decline in health, indicating a deterioration in the quality of life to the extent that some or all of the group made a decision to move from the site, cannot be confirmed without further evidence.

It is clear that the health status of the people of Khok Phanom Di cannot be classified simply. Tropical environments such as that of the Gulf of Thailand have high biological productivity. For the people of Khok Phanom Di, this would have been reflected in a high level of availability of natural resources, although this apparent wealth would have been tempered by an accompanying high level of potential pathogens. The evidence for these environmental features at Khok Phanom Di has been discussed in chapter VII. Both the richness of resources and the virulence of pathogens could have been moderated annually by seasonal variations in natural productivity reflecting the monsoonal climate, and modified in the long-term by the postulated major changes in the local environment. The successful adaptation of the people of Khok Phanom Di therefore required them to make best use of the wealth of resources to cope with the both the pathogens and the short- and long-term environmental changes.

On one level, it appears axiomatic that the survival of the people at Khok Phanom Di for the period represented by the seven mortuary phases is indicative of a successful adaptation by the population for that period. As discussed in the previous section, their subsequent fate remains unknown. Whether the environmental changes proved so overwhelming that they abandoned the site altogether for a time, meaning that they were no longer successfully adapted, requires speculation on several levels. Beyond the evidence of health in the later mortuary phases already discussed, nothing further can be inferred from the skeletal evidence about the adaptation of the people subsequent to the final mortuary phase.

Beyond the bare fact of survival of the population for the period represented by the burials, the success of their occupation of the site during that period as reflected in health is the point at which the complexity of biological mechanisms involved becomes apparent. The most obvious of these is the presence of the haemoglobinopathy in the gene pool. This suggests that the population was able to survive in an environment which also supported malarial parasites. In doing so, they paid the biological cost of both the blood disorder and the parasitic infection. This is likely to have been implicated in the infant and child mortality and morbidity and their long-term consequences for both the individuals and their parents in reproductive 'wastage' and the failure to achieve growth potential. There may also have been a reduction in energy and consequently work capacity of those with anaemia. The apparent presence of anaemia in the population from the earliest mortuary phase suggests that the first occupants of the site may have brought the gene with them. They were therefore already adapted to malaria and had migrated to the site from a malarial environment. The importance of its identification has wider significance, discussed in the next section.

The maintenance of good cortical thickness in adulthood, despite the apparent failure to achieve growth potential in stature, was particularly evident in males. They may have suffered episodes of ill health during the critical growth period of infancy which had a long-term effect, whereas any loss in cortical thickness was restored during recovery. There is a correlation between body size

and climate which reflects the significantly better heat tolerance of a small body size because of a relatively smaller surface area compared with large body size (Harrison *et al.* 1988). In the tropical climate of Central Thailand there would be a physiological advantage in small body size. The comparatively rapid recovery from illness in females allowed them to maintain growth to reach a stature similar to that of the females from the sites in North East Thailand as well as maintaining or recovering cortical thickness and fertility. The high level of infant mortality in the early mortuary phases may again be explained as a mechanism of adaptation. The virtual absence of child mortality during this period may indicate that the cause or causes of the neonatal and infant deaths was sufficiently virulent to ensure that no weaklings survived only to die later in childhood after effectively wasting the resources of the community. Carrying this suggestion further, the change in infant and child mortality rates later in the occupation of the site is an indication of weakening ability to survive, perhaps in the face of the changing environment. The almost universal fertility of the females from an early age suggests that the length of the adult lifespan was not a reproductive disadvantage but long enough to allow the population to maintain itself.

ASSOCIATION OF THE HAEMOGLOBINOPATHY, MALARIA AND AGRICULTURE

Studies which have considered the relationship of the distribution of genetic anaemia and malaria have drawn a link between these factors and the large-scale human modification of the environment associated with the introduction of agriculture. One study is based on the distribution of the sickle-cell gene in West Africa. Livingstone (1967) suggested that malaria became hyperendemic in the region when large areas of tropical forest were cleared for agricultural land *c.* 2000 years ago, with the result that the breeding places for the species of mosquito which are major vectors of malaria were multiplied. He suggested that the present-day distribution of the sickle-cell gene was determined by the pattern of adoption of forest clearance and the subsequent redistribution of both the human population and the mosquito vectors of malaria. The interaction of the reproductive advantage of sickle-cell trait carriers with the redistribution and expansion of the malarial parasite population over the time since the forest clearance has been demonstrated mathematically by Wiesenfeld (1967) to account for the present distribution pattern of the sickle-cell gene. In a similar association of genetic anaemia with human modification of the environment, Angel (1966, 1971) suggests that the relatively high prevalence of porotic hyperostosis in early farmers living in marshy areas in the Eastern Mediterranean compared with their hunter-gatherer ancestors or their contemporaries living in dry or rocky environments indicates that there is a correlation between the pathology and the environment. He suggests that the early farmers, who preferred soft and marshy soils, modified drainage patterns and this, along with the increase in human population density, encouraged an increase in the mosquito population. After a time-lag, this resulted in an increase in the prevalence of the genetic anaemia because of the preferential survival and fertility of heterozygotes for the gene.

The possibility also exists of an association of the human modification of the environment in the vicinity of Khok Phanom Di with the prevalence of mosquitoes. The clearing of the forest suggested by Maloney (1991) to date from *c.* 5000–4000 B.C. to the time of occupation of the site could have

modified the drainage pattern to the extent that one or more of the known Anopheline carriers of malaria were able to breed. The survival of the malarial parasite, as discussed in chapter VII requires not only a suitable vector but also a suitable human host population. Whether the pathogen would have been able to survive in the area prior to the occupation of Khok Phanom Di depends on the pattern of human inhabitation. Small, mobile groups of people are unlikely to have maintained the parasite. The existence of a sedentary population would have been a necessary prerequisite. If the early episodes of forest burning do reflect the modification of the environment in preparation for rice cultivation, the chances are high that such a population did exist.

CONCLUSION

The people of Khok Phanom Di were living in a fertile, resource-rich tropical environment. They used both aquatic and terrestrial resources in obtaining food and material items. They grew or had access to domesticated rice and probably exploited a variety of wild plant foods. They caught fish and collected shellfish and crustacea. Despite the seasonal, monsoonal climate, they may have been well-nourished for most of the year. They were technologically skilled and energetic manufacturers of pottery. They traded or otherwise acquired a variety of exotic goods such as shell jewellery and stone tools. They would have had access to many of these resources by boat. The river would have afforded a means of transport of both food and other items in bulk, as well as a means of communication.

There were also disadvantages in living in such a rich environment, which also allowed a variety of parasitic organisms to flourish. These included one of the malarial protozoa, *Plasmodium* sp. This had a profound effect on their health, both directly through infection and indirectly through the maintenance in the population of genes for one of the haemoglobinopathies. There was probably also some infection with less virulent intestinal parasites. Infant mortality was high, particularly about the time of birth. Illness in childhood was almost universal, perhaps as a consequence of parasitic infection, although seasonal malnutrition cannot be absolutely discounted as a contributor. Adults, and males in particular, were short but nevertheless maintained good bone structure. Females were fertile from an early age and may have experienced extra pregnancies because of the high neonatal mortality rate. A large proportion of those who died in childhood had anaemia and some of the adults had suffered from it at some time in their lives. Degeneration of the limb joints and the spine was common and probably a consequence of a physically active life. Dental pathology was also common and this source of infection may have contributed to a short adult lifespan. Another disadvantage of life at Khok Phanom Di was the major environmental change which occurred during the period of use of the cemetery. The change appears to have the most profound effects on children, with fewer neonatal deaths but higher child mortality, and on males. Prior to the change, males had been comparatively tall and well-built. In the later period they were shorter and smaller and lived shorter lives. There were no adults buried in the latest levels of the cemetery. Whether that indicates emigration of some or all of the community or a cultural change in the disposal of the dead is not known. Further excavation at the site would be extremely valuable in increasing the size of

the skeletal sample and perhaps clarifying some of the general issues of demography and sampling as well as answering specific questions such as the fate of the parents of the children buried in MP7.

Although the suggestion of the possible presence of one or more of the haemoglobinopathies in prehistoric South East Asians is not new, the people of Khok Phanom Di have provided the first firm evidence of its presence in a population, as well as the opportunity to examine the implications for their health and quality of life. This, together with the explanations for other characteristics of the health of the people of Khok Phanom Di, has offered a dynamic picture of them and their lives which goes some way towards describing a prehistoric Thai population "with confidence".

X. APPENDIX: INDIVIDUAL PROFILES

THESE profiles do not attempt to provide all the detailed data for each individual but are a summary of the skeletal representation and preservation, together with a brief outline of the most important aspects of each person's morphology and health. All ages given are approximate.

Burial 1 MP7, 9y. The bone is moderately well preserved, with the axial skeleton complete, the cranium present but distorted, and some of the long bones present, although damaged. Although there is no definite evidence for growth disruption, the child was short for its age and was probably anaemic. The teeth were healthy.

Burial 2 MP6, 9m. The skeleton is represented by fragments of the axial skeleton and some long bones. The child was buried with one pot. The cranium and mandible are crushed and fragmented. Apart from areas of defective enamel on the deciduous canines, no observations as to health can be made.

Burial 3 MP7, 4m. This infant is represented by fragments of ribs and vertebrae. The grave was disturbed, but had contained pots and jewellery.

Burial 4 MP6, female, 25y. The skeleton is almost complete but the bone is poorly preserved. She was one of the smallest individuals in the sample. She had suffered growth disruption in infancy and the short stature suggests childhood health problems, although she had thick bone cortices which is at variance with this. She had borne a child or children. She had dental problems with caries and periodontal disease, and had lost some teeth during life. There is evidence on the clavicle and metacarpals of moderate use of the upper limbs and hands.

Burial 5.1 MP6, neonate. This infant was buried in a crouched position in a scooped grave. The interment disturbed B5.2 which was buried in the same grave. The skeleton is represented by fragments of the cranial and postcranial skeleton.

Burial 5.2 MP6, neonate. This infant was buried in the same grave as B5.1, and was disturbed by the later interment. The skeleton is represented by the pelvis and lower limb bones.

Burial 6 MP6, 9m. This child was buried on the mortuary platform, above B18 but possibly in the same grave. The bone is well preserved, but the skeleton had been disturbed by a later prehistoric pit and only the upper body, with the exception of the right arm, is present. The cranium is present, but incomplete. The child had light cribra orbitalia but there is no other evidence of ill health other than a single carious deciduous molar.

Burial 7 MP6, 21m. The skeleton is represented by fragments of some long bones and an almost complete cranium. Apart from a patch of defective enamel on one canine, there were no observations as to health.

Burial 8 MP6, 15y, probably male. The skeleton is almost complete, including the cranium, which is distorted. As he was an immature individual, no size estimations have been made. There is spondylolysis of the fifth lumbar vertebra.

Burial 9 MP6, 30y, male. The skeleton is almost complete, although the bone is encrusted and there is some damage. The cranium is distorted and fragmented. He was small, and there is minimal evidence on the bones of growth disruption during adolescence. The right elbow and knee joints show some evidence of degeneration and the cortices are thinner than average for males. There is minor evidence of physical activity involv-

ing the upper arms or shoulders, and the legs. The upper central incisors appear to have been ablated and he suffered from periodontal disease.

Burial 10 MP7, 15m. The skeleton is represented by fragments of vertebrae and long bones. No observations were recorded.

Burial 11 MP6, 12y. The grave was isolated at the edge of the excavation. The bone is very well preserved, and the skeleton almost complete. The child had medium grade cribra orbitalia, and had suffered episodes of growth disruption. The blood disorder is likely to have been the underlying cause of death.

Burial 12.1 MP7, 12y. The bone is moderately well preserved, but the skeleton is incomplete and represented only by some long bones, vertebrae and part of the cranium. The grave had disturbed B12.2. It was isolated at the edge of the excavation. The child had severe cribra orbitalia, suggesting anaemia, but there is no other skeletal evidence for the aetiology of the condition. The illness is reflected secondarily in poor dental enamel and short stature for the age of the child and may have contributed to the early death.

Burial 12.2 MP7, 5y. These bones were found with B12.1 with the grave possibly disturbed by that burial. The bone is well preserved, but there are only a few fragments of the skeleton, including part of the mandible. No other observations were made.

Burial 13 MP6, 35y, female. The skeleton, including the cranium, is almost complete, although the bone is in poor condition with some postmortem damage. She was of average-large size for the group, and was the largest female in MP6. The teeth suggest some childhood growth disruption. She suffered from extensive dental caries, with periapical cavities and loss of teeth, and had minor spinal bone degeneration, but was otherwise healthy with strong cortices. There is some evidence for vigorous use of the upper limbs.

Burial 14 MP5, 15m. The body was interred in two urns, the only such burial recovered, and was close to B15. Jewellery and pots were included in the burial. The bones are fragmentary, and the cranium present but partially complete. Two teeth have areas of defective enamel formation, but no other observations were made.

Burial 15 MP5, 35y, female. The skeleton is almost complete, although the cranium and mandible are in pieces. The bone is poorly preserved. This was the richest burial in the sample with much jewellery, many pots and pot-making equipment and a deep, elaborate, grave. She was among the shorter individuals from the site. There is evidence from the teeth of some growth disruption in infancy, although cortical thickness suggests that she was generally healthy. There is specific evidence of degeneration in the neck and lower spine, and one knee. She had some dental problems with caries and loss of teeth during life. There is evidence of vigorous use of the upper limbs, and of the hands and forearms in particular.

Burial 16 MP5, 15m. The burial included much jewellery, many pots and a miniature clay potter's anvil, in a scaled-down version of the goods buried with B15. The skeleton is represented by some of the long bones and fragments of the axial skeleton. The cranium is virtually all present, but crushed. No observations could be made as to health.

Burial 17 MP4, 12y. The bone is moderately well preserved, and the skeleton, including the cranium, almost complete, although some long bones are damaged. The child had suffered periods of growth disruption and had medium grade cribra orbitalia.

Burial 18 MP6, 42y, female. The skeleton, including the cranium, is complete apart from the distal tibiae, fibulae, and feet, which were disturbed and removed in prehistory. The bone is in good condition. The grave was on the mortuary platform. She was one of the smallest in the group and had suffered growth disruption.

tion during early childhood. She was in poor health at death, with severe degeneration in all regions of the spine, including several collapsed vertebrae. The spinal degeneration and thin cortices suggest she was osteoporotic. She also suffered from severe dental pathology. There is evidence of physical use of the shoulders.

Burial 19 MP, 25y, female. The skeleton is almost complete, although the bone is in poor condition, with some long bones and the cranium damaged. The grave was on the mortuary platform. She had suffered minor growth disruption during infancy and was of average size for the group. She was healthy during adulthood.

Burial 20 MP4, 12y. The bone is very well preserved and the skeleton almost complete, although the cranium is distorted. There is evidence of growth disruption at about the time of birth and again later in childhood. There are multiple caries in the teeth, which is unusual for such a young individual. No more specific observations were made.

Burial 21 MP4, 8y. This child was buried in the same grave as B23, with no grave goods. The bone is very well preserved, and the skeleton virtually complete apart from the left forearm and damage to some other long bones. The child had been anaemic, almost certainly as a result of one of the haemoglobinopathies. Some of the metatarsals and hand phalanges have hypertrophied shafts, with marked porosity of the cortical bone and enlarged foramina. Radiographs show the cortical bone to be disorganised. Similar features are noted on the spine of the scapulae, clavicular shafts and the nasal bone. The child had suffered episodes of growth disruption and the haemoglobinopathy was most likely to have contributed to the early death.

Burial 22 MP4, 21y, male. The postcranial skeleton is almost complete, but the head and the first cervical vertebra were removed in prehistory. The presence of the rest of the neck vertebrae suggests that the head was removed sometime after interment rather than decapitation being the cause of death. The bone is in good condition. He was of average size for the group, with well-developed muscular attachments on the clavicle and lower limb in particular. He had had a healthy childhood. Despite his youth, he suffered from neck problems, although the sacralization of the fifth lumbar vertebra may account for the degeneration. There is evidence of vigorous physical activity involving the shoulders and the lower limbs.

Burial 23 MP4, 30y, male. The bone is in good condition and the skeleton is virtually complete apart from the cranium, which is also distorted. B21 was interred in the same grave. He was one of the taller men in the group. He was the tallest in MP4, and although relatively light when compared with other males in that mortuary phase, both upper and lower limb bones suggest well-developed muscularity. There is evidence of growth disruption in infancy and again in adolescence. The cortical thickness suggests that he was reasonably healthy. He suffered from spinal degeneration, although unusual morphology of the first sacral vertebra may account for this. The robusticity of the bones and pattern of degeneration suggest vigorous physical activity involving both the upper and lower limbs.

Burial 24 MP4, 25y, male. The skeleton, including the cranium, is virtually complete, and the bone in good condition. Although he was the shortest male in the group, the bones show evidence of well-developed muscles. He had multiple episodes of growth disruption in infancy and adolescence and had probably suffered from the haemoglobinopathy. At variance with the short stature and growth disruption, the bone cortices were well developed. In the dentition, caries have developed in a band of enamel hypoplasia in the third molars. He had asymmetrical humeri, with one misshapen at the proximal end. This probably reflected premature fusion of the epiphysis. He suffered from arthritis in this shoulder. The shafts of the metacarpals and lower limb bones are bowed. The development of muscle attachments suggests that he was very active physically, particularly using the hands, especially the right. He appears to have survived through the exigencies of the blood condition in childhood to lead a short but active adult life.

Burial 25 MP4, 19y, female. The skeleton is virtually complete, with the bones in good condition. The cranium is complete, but badly distorted. She had borne a child or children, despite her young age. She was of small-average size for the group, and was the smallest female in MP4. The bones and teeth show evidence of a period of growth disruption in infancy. There is some evidence on the clavicle and the humeri of use of the upper limb and shoulders.

Burial 26 MP4, mid thirties, female. The skeleton is almost complete, and the bone in good condition. The cranium is complete but distorted. She was average size for the group. There was evidence on the bones and teeth of multiple episodes of growth disruption from infancy to later childhood. She was not healthy, with thin cortices, moderate to severe lower spinal degeneration and extensive dental problems with extreme wear, caries, severe periodontitis, periapical cavities and loss of teeth during life. There is evidence of physical activity involving the upper limbs.

Burial 27 MP4, 40y, female. The bone in this skeleton was poorly preserved, with considerable damage. Parts of most bones are present, including the cranium, although the left maxilla and many teeth were lost postmortem. She was average size, but gracile, with thin cortices. She suffered from degeneration in almost all joints, but it was particularly severe in the elbows and knees, and all regions of the spine. Her teeth showed evidence of considerable infection and tooth loss during life. There is evidence of physical activity involving the upper limb.

Burial 28 MP4, 21y, probably male. The skeleton, including the cranium, is almost complete, although some bones were damaged and the bone fabric is only moderately well preserved. The mandible is missing. The sexing and assessment of age at death of the individual posed problems, as the sexually-dimorphic features of the pelvis and cranium were equivocal, and the degree of dental and skeletal degeneration appeared to be at variance with the state of epiphyseal fusion (the iliac crest fusing). He was amongst the shorter males, and was underweight, with minimal muscular development. There is evidence on the bones of periods of growth disruption in infancy and adolescence, and thin cortices suggest severe osteoporosis. The lumbar vertebrae are porotic, with osteophytosis and collapse which may have been partially a consequence of spondylolysis and possibly spondylolisthesis of L5. He suffered from extreme dental pathology, with almost all maxillary teeth lost during life, the remaining teeth carious and the periodontal tissues infected. The lack of distinctive secondary sex characteristics, the possibility that the fusion of the iliac crest epiphysis was delayed, and the evidence on both the bones and the teeth of ill health suggest that this individual may have been one of the sufferers of the haemoglobinopathy. Although he survived into adulthood, he may not have been able to contribute significantly to the community.

Burial 29 MP4, 27y, male. Most bones are present, but some are incomplete, including the cranium. The bone is well preserved but was heavily encrusted with calcareous deposits, which made cleaning difficult. He was of average size and muscular. There was evidence on the dentition of some growth disruption. The lower spine shows evidence of some degeneration. Both upper and lower limbs show evidence of vigorous physical activity.

Burial 30 MP4, 34y, male. The skeleton is complete, including the cranium, and the bone in very good condition. He was small-average size, although the cranium and mandible were robust. He suffered multiple growth disruptions in infancy, with enamel hypoplasia sufficiently severe on the mandibular canines to result in a break on the crown at the level of the defect. Despite the growth disruptions, the cortices were thick and he appears to have been healthy in adulthood. Evidence of degeneration was minimal. There is evidence of vigorous use of the shoulders and the lower limbs.

Burial 31 MP4, 25y, female. The skeleton was almost complete, although the cranium was partly missing. The body was interred in the same grave as B32. The bone is in very good condition. She had suffered mul-

multiple episodes of growth disruption from infancy to later childhood but was of average size for the group. There are no other indications of ill health in adulthood. Her upper limbs were well developed.

Burial 32 MP4, 11y. This child was buried with a turtle carapace and an object which is possibly a figurine, in the same grave as B31. The skeleton is in very good condition, and the skeleton almost complete, although the cranium is distorted. The bones show evidence of growth disruption in infancy, and the canines of disruption during later childhood. Medium grade cribra orbitalia suggests that the child was anaemic, although the muscle attachments on the bones were relatively well developed for such a young individual. The presence of a turtle carapace in the grave suggests that the child was a male, who may have been contributing physically to the community despite his youth. He may have been small for his age because of the illness.

Burial 33 MP4, 3y. This infant was buried in a relatively deep grave with rich grave goods. The grave was isolated, at the edge of the excavation. The bone is moderately well preserved, and the skeleton represented by the cranium, which is crushed, some long bones and fragments from the axial skeleton. Many of the teeth have enamel hypoplasia in the form of discrete areas of missing enamel, some with associated caries. The metacarpals have enlarged foramina.

Burial 34 MP4. This infant died at birth. The skeleton is represented by some long bones, vertebrae, and the cranium, which is virtually complete but fragmented.

Burial 35 MP4, 21y, female. This woman revealed evidence of pregnancy. The skeleton is almost complete, including the cranium, but the bone is not well preserved, with some damage to the pelvis and some vertebrae. B36 was interred at her feet. She was among the larger females. There is evidence on the bones of multiple growth disruptions during the ages of 6–10 years, but no evidence on bones or teeth of problems in infancy. She appears to have been healthy in adulthood, with no evidence of pathology. The upper limb and hands show evidence of vigorous muscle use.

Burial 36 MP4, 21y, female. This female had a pregnancy. The skeleton is almost complete, although there is some damage to long bones and the cranium is distorted. The bone is moderately well preserved. She was buried at the feet of B35. The bones and teeth show evidence of growth disruption in infancy. She was of average size, but gracile with thin bone cortices. Apart from some periodontitis, there is no other evidence of pathology or degeneration. The hands show evidence of vigorous use.

Burial 37 MP4, 9y. The bone is moderately well preserved, and the skeleton, including the cranium, almost complete. The child had suffered episodes of growth disruption, medium grade cribra orbitalia and enlarged nutrient foramina which indicates possible anaemia.

Burial 38 MP4, early thirties, male. The skeleton is virtually complete, including the cranium, and the bone fabric is in good condition, although some long bones are damaged. He was average-large size, but muscular, with thick cortices. The teeth show evidence of multiple growth disruption in infancy, and the bones show an episode at about the age of 10. There is some evidence of degeneration of the upper spine, and of the loss of some teeth during life. The left ulna shows evidence of a healed lesion on the arm. There is evidence of vigorous physical activity involving the whole body, with strong neck and upper and lower limb muscles.

Burial 39 MP4, 25y, female. The skeleton is almost complete, and the bone in moderate condition, with some damaged, including the cranium. She was amongst the shorter females and was the shortest in MP4. There is evidence on the bones and teeth of growth disruption in infancy, but thick cortices nevertheless suggest relatively good health. The only pathology is on the dentition, where there were some caries and loss of third molars during life.

Burial 40 MP4, late thirties, female. The skeleton is almost complete, with the bone in moderate condition, although there is damage to some of the long bones and the cranium, and extensive postmortem loss of teeth. She had borne a child or children and was buried in the same grave as an infant, B46. She was of average size for the group. Enamel hypoplasia on the canines suggests some growth disruption at about age five. The cortex of the second metacarpals is thick, but as the cortices of other bones are not well developed, this may reflect vigorous use of the hands rather than systemic good health. She had degeneration of the spine and lower limbs and some dental pathology. The attachments of the dorsal interosseous muscles on the metacarpals are very well developed, and the forearms show evidence of muscularity. These factors, together with the degeneration of the vertebral discs in the neck and the development of costal facets on the vertebrae, suggest vigorous physical activity involving the upper limb and shoulders, and in particular the hands.

Burial 41 MP4, 9m. The skeleton is represented by fragments of the cranium and axial skeleton, and some long bones. No observations were possible.

Burial 42 MP4, early thirties, male. The skeleton is almost complete, including the cranium, although the bone is only moderately well preserved and some long bones are damaged. He was among the largest males in the group, and the largest in MP4. Although he was slightly shorter than B23, he was heavier, and the cranium is robust. The teeth suggest growth disruption in infancy, but the bones show no lines of arrested growth. The metacarpals show slight cortical thinning. There is unusual remodelling of the lower thoracic spine, with T11 compressed on the right, and asymmetric degeneration of the body and the costal facets on T12. Spinal degeneration otherwise is minor, in the cervical and lumbar vertebrae, suggesting that the thoracic degeneration may have been in response to trauma. The dentition shows extreme wear, few caries and loss of teeth during life, and severe periodontitis. There is evidence of physical activity involving the upper body.

Burial 43 MP5, 30y, male. Parts of most of the major bones of this skeleton were recovered but preservation of the bone is very poor, most are crushed and many are cemented together with calcareous deposits. The grave was isolated at the edge of the excavation. He was amongst the smaller males of the group. His teeth show evidence of extensive hypoplasia, suggesting growth disruption during infancy. Minimal observations could be made on the bones, there was no pathology visible on the teeth, and the only skeletal degeneration recorded was on the lumbar spine. The clavicles and humerus suggest that the shoulders were muscular and that he was involved in vigorous physical activity.

Burial 44 MP4, 18y, male. The skeleton is virtually complete, although some of the long bones were damaged and the bone fabric is not well preserved. The cranium is complete, but there was considerable postmortem loss of teeth. He suffered some growth disruption during childhood, but despite being not yet skeletally mature, he was already taller than some mature males, although the cortical structure was not strong. The robusticity of the mandible and upper limb suggested good muscular development. He has cribrotic porosity of the outer table of the parietal, of unknown aetiology.

Burial 45 MP4, mid forties, female. The skeleton is virtually complete, and despite some damage to the ends of some long bones as a result of brittleness after cortical thinning during life, the bone is well preserved. There was considerable postmortem loss of teeth. The individual was one of the older and taller females. Along with B47, she was the largest in MP4. The bones showed evidence of episodes of growth disruption in early infancy and later childhood. The bone cortices are very thin. Remodelling and pathology of bones and the dentition suggest extensive age-related degeneration involving the lower spine and the major joints of both upper and lower limbs. The dental alveolar bone had several periapical cavities, and the third molars were impacted, with adjacent periostitis. The development of the muscle attachments on the bones suggest that she had engaged in vigorous physical activity involving the upper limbs and hands.

Burial 46 MP4 4m. This infant was buried in the same grave as B40, an adult female. The skeleton is represented by fragments of the axial skeleton, the cranium, and long bones. No health observations were possible.

Burial 47 MP4, early twenties, female. The skeleton is virtually complete, including the cranium, and the bone is in very good condition. The burial was in the same grave as an infant, B48. She was one of the largest females. The dentition and bones show evidence of growth disruption during infancy, but there is no evidence of pathology other than thin cortices. Development of muscle attachments suggest that she was involved in physical activities using her hands.

Burial 48 MP4. This infant died at birth, and is represented by fragments of the cranium and postcranial skeleton. The burial was in the same grave as B47, an adult female.

Burial 49 MP3. This infant died at birth and was buried prone. It is represented by fragments of the cranium and post cranial skeleton.

Burial 50 MP3. This infant died at birth, and the skeleton is represented by fragments of the cranium and axial skeleton and some of the long bones.

Burial 51 MP3 This infant died at birth, and is represented by fragments of the cranium and postcranial skeleton. The lower body was disturbed.

Burial 52 MP3. This infant died at birth, and is represented by fragments of the cranium and the postcranial skeleton. The grave was disturbed.

Burial 53 MP3. This infant died at birth, and the burial was disturbed by B54, which was buried in the same grave. The skeleton is represented by fragments of ribs and vertebrae.

Burial 54 MP3. This infant died at birth, and is represented by fragments of the cranium and the postcranial skeleton. It was buried in the same grave as B53.

Burial 55 MP3. This infant died at birth, and is represented by fragments of the cranium and the postcranial skeleton.

Burial 56 MP3, 45y, female. Parts of almost all bones were present, but the bone was not well preserved and there is damage to the ends of long bones. The cranium is crushed. She was exceptionally small, by far the smallest individual in the group. The dentition shows evidence of multiple episodes of growth disruption, with the enamel defects on mandibular molars sufficiently severe to result in caries. The bones are gracile, with no evidence of muscular development, and the cortical bone is very thin. There is no evidence of degeneration on the spine, but the left shoulder, hands and knees are arthritic. She also suffered from severe and multiple dental pathologies with extreme wear, caries and infection of the periodontal tissues. There is bony evidence that she suffered from anaemia, probably the haemoglobinopathy. She appears to have been unhealthy during childhood, probably reflecting the blood disease, but once she reached adulthood to have lived a long, full and active life, including bearing children.

Burial 57 MP3, 26y, male. The skeleton is almost complete, including the cranium, and although the bone is well preserved, it is heavily encrusted and some damage resulted from cleaning. He was one of the larger males in the group, both in stature and mass, and was muscular with strong cortical bone. He had minimal spinal degeneration. He had was one of the big, physically active men in this mortuary phase and there is no evidence as to the cause of his early death.

Burial 58 MP3, 35y, female. The skeleton is almost complete apart from some vertebrae and the right clavicle. The bone is well preserved, although the ends of some long bones are damaged. She was one of the largest

females in the group, and the tallest in MP3. Despite this, the cortical bone was not thick, perhaps reflecting her age. She suffered from degeneration of the lower back and elbows and severe dental degeneration with caries and infection. Her shoulders and lower limbs were muscular.

Burial 59 MP3. This infant died at birth and was buried in a scoop grave. The skeleton is represented by some of the upper limb bones and fragments of the vertebrae, pelvis and cranium.

Burial 60 MP3, 19y, female. The skeleton is almost complete, but the bone is in poor condition, with some damage including fragmenting of the cranium. She had borne children, despite her youth. The dental enamel suggested multiple episodes of growth disruption in infancy, including distinctive pitting, opacity and staining of the entire crowns of the maxillary third molars. She had light cribra orbitalia. She was of average size for the group, and the cortical bone is thick, suggesting that the growth disruption did not have a long-term effect. There is evidence of physical activity involving the forearms and possibly the hands.

Burial 61 MP3, late forties, female. The skeleton is almost complete, including the cranium, although the bone is not in good condition and some long bones are damaged. She was of average stature for the group, but the heaviest female. There was evidence of extensive, probably age-related, pathology, including osteoporosis and degeneration of all regions of the spine and most limb joints. The teeth were extremely worn, with some consequent periapical infection and loss of teeth during life. She had suffered a fracture to the left clavicle early in life, but this had healed satisfactorily. The development of muscle attachments on the bones suggested that she had been involved in vigorous physical activity involving both upper and lower limbs.

Burial 62 MP3, 6m. This infant was buried with two pots. The skeleton is represented by fragments of the axial skeleton, the cranium, and long bones. No health observations were possible.

Burial 63 MP3, 21m. The skeleton is represented by fragments of the axial skeleton, the cranium, and long bones. The bones show evidence of growth disruption at about 12 months.

Burial 64 MP3, early twenties, female. The bone is in excellent condition, and the skeleton complete, except for most of the foot bones. The bones and teeth suggest multiple episodes of growth disruption in infancy and during childhood, including irregularity, staining and pitting on the maxillary third molars. She was of average size for the group. She had borne a child or children.

Burial 65 MP3. This infant died at birth, and is represented by fragments of the cranium and the postcranial skeleton. The body was buried on its back, with the legs flexed, in the same grave as B66.

Burial 66 MP3. This infant died at birth, and is represented by fragments of the cranium and the postcranial skeleton. The body was buried on its back, with the legs flexed, in the same grave as B65.

Burial 67 MP3, 35y, male. The skeleton is almost complete, including the cranium, and the bone is well preserved, although heavily encrusted with calcareous deposits. He was of average size for the group, with moderate thickness of the cortical bone. Many joints show evidence of degeneration, including all regions of the spine, and major joints of the upper and lower limbs. The degeneration was particularly severe on the right shoulder, the neck and the upper thoracic vertebrae, and the lumbar spine. There was some ossification of the vertebral costal cartilages. The pattern of degeneration and the robusticity of the clavicles and the humeri suggests that he was involved in vigorous physical activity using the shoulders. There was a non-active lesion on the shaft of the right humerus, suggesting a trauma involving primarily the soft tissue of the upper arm. The dentition showed extreme wear with associated periapical infection, some periodontitis and loss of teeth during life. He appears to have lead a physically active life, using the upper body in particular, and to have paid the price of consequent joint degeneration.

Burial 68 MP3. This infant died at birth, and is represented by fragments of the cranium and the postcranial skeleton. The interment was in the same grave as B69.

Burial 69 MP3. This infant died at birth, and is represented by fragments of the cranium and the postcranial skeleton. It was buried above B68, in the same grave.

Burial 70 MP3. This infant died at birth, and is represented by fragments of the cranium and the postcranial skeleton.

Burial 71 MP3. This infant died at birth, and is represented by fragments of the cranium and the postcranial skeleton.

Burial 72 MP3, early thirties, male. His skeleton is almost complete, including the cranium, and the bone in very good condition. There is evidence of growth disruption in infancy and adolescence. The morphology of the clavicle and scapula suggests that he had wide shoulders. He was one of the large males in MP3, and one of the largest in the group. The bone cortices are very thick. There is slight degeneration in all regions of the spine, and arthritis in both upper and lower limb joints. The bones of both upper and lower limbs suggest well developed muscularity. He appears to have been a big man, who had a healthy childhood, developed to full genetic potential physically, and worked hard in adulthood.

Burial 73 MP3, mid twenties, female. Her skeleton is almost complete, including the cranium, and the bone well preserved, although heavily encrusted. The bones suggest minimal growth disruption during infancy, but the dentition shows that numerous episodes occurred. She was amongst the smaller individuals in the group, and one of the smallest in MP3, although the thickness of the cortical bone does not suggest long-term ill health. The lumbar spine showed some degeneration. Caries and antemortem tooth loss affected the third molars, principally in association with enamel hypoplasia. Muscle development suggests physical activity patterns using the arms.

Burial 74 MP3, 40y, male. The skeleton is virtually complete, including the cranium, and the bone well preserved, although encrusted with deposits which made cleaning difficult. Many teeth were damaged or lost postmortem. He was one of the largest males in the group, and among the group of large males in MP3. All limb bones showed evidence of extreme muscle development and there was degeneration of the spine, particularly the upper right side, both shoulders, and the lower limbs. The robusticity of the bones and the pattern of degeneration suggests a lifetime of vigorous physical activity, particularly involving the shoulders and upper body. The dentition shows extreme wear with associated periapical cavities and some periodontitis. He is an example of the big males in this mortuary phase who lived to a good age after a vigorous, healthy life.

Burial 75 MP3. This infant died at birth, and is represented by long bones, fragments of the cranium and ribs. The grave had been disturbed in prehistory.

Burial 76 MP3, 34y, male. The bone was heavily encrusted with calcareous deposits, making it difficult to clean. Parts of most bones were present, but some of the long bones were damaged. The cranium was crushed and is incomplete. He was one of the smaller males in the group, and the second smallest in MP3. The bones and teeth show evidence of growth disruption during infancy and adolescence. Degeneration was severe on the neck and less so on major limb joints. The bodies of a few lower thoracic and lumbar vertebrae showed slight asymmetry, resulting in slight scoliosis. The upper arms suggests vigorous use of the shoulders, although the pattern of degeneration of the spine suggests a different physical activity from that of other MP3 males. He had relatively good dental health.

Burial 77 MP3, mid twenties, female. The skeleton is almost complete, including the cranium, and the bone is in very good condition. She was among the larger females. The dentition has evidence of multiple episodes

of childhood growth disruption, but she had thick cortical bone, and this, together with her size, suggests that there were no long-term effects. She suffered from joint degeneration in the spine and major limb joints. Despite the pattern of degeneration, there is no evidence of particular physical activities. She had some periodontal infection, with one abscess draining into the maxillary sinus. She had lost a few teeth during life with consequent alveolar resorption.

Burial 78. This infant died at birth, and is represented by long bones, fragments of the cranium and ribs. The grave had been disturbed.

Burial 79 MP3, late forties, female. The skeleton is virtually complete, including the cranium, and the bone is well preserved, although heavily encrusted. She was of average size for the group and the cortical bone was relatively thin, probably a reflection of age-related involution. She suffered from degeneration of the spine and major limb joints. The pattern of degeneration on the right upper body and remodelling of the arm bones in response to muscular development suggest that she was involved in vigorous physical activity involving that part of the body. She also suffered from dental degeneration, with extensive tooth loss during life, resorption of the alveolar bone, and a chronic dental abscess which connected with the maxillary sinus was continuing to drain through a cloaca in the alveolar bone.

Burial 80 MP3. This infant died at birth, and is represented by long bones, fragments of the cranium and ribs. The body was lying on its back, with the legs flexed, in the same grave as B81.

Burial 81 MP3. This infant died at birth, and is represented by long bones, fragments of the cranium and ribs. The body was interred on its back, with the legs flexed, in the same grave as B80.

Burial 82 MP3 6m. The grave was partly disturbed by a posthole, and contained miniature pottery working equipment. The skeleton is represented by fragments of the axial skeleton and long bones. No health observations were possible.

Burial 83 MP3, 30y, female. The skeleton is almost complete, including the cranium, and the bone well preserved, although heavily encrusted with calcareous deposits. She was one of the smaller women in the group and had suffered episodes of growth disruption during infancy. There is no evidence of particular physical activity.

Burial 84 MP3. This infant died at birth, and is represented by long bones, fragments of the cranium and ribs.

Burial 85 MP3. This infant died at birth, and is represented by fragments of the cranium and postcranial skeleton. The burial was in a scoop grave, and was disturbed.

Burial 86 MP3, mid twenties, male. The skeleton is largely complete, although there is damage to the ends of some long bones, and the cranium is crushed and fragmented. The bone is not well preserved, and was heavily coated which made cleaning difficult. He was of average height but was heavy. He had minor spinal degeneration and the shoulders and right knee were arthritic. The pattern of degeneration and development of muscle attachments at the shoulders suggest physical activity involving the upper body. He was otherwise in good health.

Burial 87 MP2, 24y, female. The skeleton is almost complete and the bone fabric is well preserved but, apart from the cranium, heavily encrusted and difficult to clean. She had suffered growth disruption in infancy and again in adolescence but was of average height but was heavy. Spondylolysis and possibly spondylolisthesis of the third and fourth lumbar vertebrae caused some lower spinal degeneration. Apart from dental problems in the form of caries and infection in the third molars, possibly resulting from defective enamel formation, she appears to have been in good health and had borne children.

Burial 88 MP2, 9m. The grave was disturbed. The skeleton is represented by fragments of the axial skeleton, the cranium, and long bones. The child had suffered episodes of growth disruption, and had caries in defective enamel on the deciduous teeth. The shafts of the long bones of both the upper and lower limbs and the clavicles have reactive subperiosteal bone, with growth extending radially from the shafts. This is most likely to reflect a haemoglobinopathy which would have contributed to the death of the child.

Burial 89 MP2. This infant died at about six months. The skeleton is represented by fragments of the axial skeleton, the cranium, and long bones. No health observations were possible.

Burial 90 MP3, 27y, male. The skeleton is almost complete, and the bone well preserved but heavily coated. The cranium is distorted and partially fragmented. He was one of the larger males. Both the teeth and bones showed evidence of growth disruption in childhood, but his size and the thickness of cortical bone suggests that catch-up growth was more than adequate to overcome any problems. All limb bones showed evidence of well developed musculature, but both the right arm and leg were particularly robust. Both clavicles had grooves at the point of contact with the first rib, this was particularly marked on the left. He had some degenerative problems concentrated on the right upper body, including the spine and the joints of the right arm. The pattern of muscle development and degeneration suggests that he was physically very active, with particular use of the upper body. He had dental caries, principally in the third molars. He was one of the big, healthy, vigorously active men in this mortuary phase.

Burial 91 MP2, 45y, male. Parts of most of the skeleton is present, but many bones are damaged and cemented together. The bone fabric is not well preserved, is brittle and heavily coated, especially on the lower limb. The cranium is crushed and cemented together, and many teeth were lost or damaged postmortem. This individual was one of the large men in the group and the tallest in MP2. The cortical bone was thinner than the other large males, which probably reflects his age. He suffered from multiple, moderate-severe degeneration in all regions of the spine and both upper and lower limbs. These also showed evidence of well developed musculature. His teeth were very worn, with multiple associated periapical cavities and lost teeth, suggesting a coarse diet. The pattern of development and degeneration suggests he had a very healthy childhood and during adulthood led a life of vigorous physical activity, especially using the upper body, but was beginning to suffer from the degeneration of advanced years.

Burial 92 MP3, 18y, male. The skeleton is virtually complete, although the cranium is crushed and fragmented. He had light cribra orbitalia and was amongst the shorter males. Despite his youth, he had spinal problems with slight arthritis on the upper vertebrae, particularly on the right side, and collapse of lower thoracic and lumbar vertebrae, with evidence of disc degeneration on the bodies of the fourth and fifth lumbar vertebrae.

Burial 93 MP3, 42y, male. The skeleton is almost complete, including the cranium, and the bone is well preserved although heavily coated. Many teeth were lost or damaged postmortem. The bones and teeth show episodes of growth disruption in infancy and in adolescence, but he was one of the larger males in the group, and one of the large men in MP3, which suggests that catch-up growth was adequate. He also had thick cortical bone. He suffered from extreme joint degeneration in all regions of the spine. Spondylolysis and possible spondylolisthesis of the fifth lumbar vertebra is a possible partial explanation for the degeneration. All the joints of the upper limb and the knees also showed degenerative changes. The pattern of degeneration and evidence of muscular development on the upper body suggest that he was physically very active, using his arms and shoulder in particular. His teeth were in poor condition, with associated periapical cavities. He was one of the big men from this mortuary phase, with degeneration after an active life.

Burial 94 MP2, 31y, female. Parts of most bones are present, and the cranium is complete apart from the maxillary dentition. The bone is not well preserved, with damage to the ends of long bones and erosion of

bone surfaces. She was of average height and gracile, but with good cortical bone structure. She had degenerative problems in the lower spine, which suggests a physically active person, although there is minimal evidence of muscle development. She had caries associated with enamel hypoplasia in the third molars. She had periostitis of the distal tibiae and fibulae which may have been a result of ulceration.

Burial 95 MP3. This burial was identified as neonate at excavation, but no skeleton was received in the laboratory. The grave had been disturbed by a posthole.

Burial 96 MP2, 4y. The grave was heavily disturbed in prehistory, and the skeleton is incompletely represented by fragments of the cranium and a few long bones. The bone is moderately well preserved, although encrusted. The bones show evidence of a growth disruption at about two years of age. No other health observations were made.

Burial 97 MP2, 6m. The grave was disturbed in prehistory, and the bones scattered. The skeleton is represented by fragments of the cranium, and a few long bones.

Burial 98 MP2. This infant died at birth, and is represented by long bones, fragments of the cranium and ribs. The grave had been disturbed.

Burial 99 MP2, 6–9m. The skeleton is represented by fragments of the cranium and a few long bones.

Burial 100 MP2, 32y, male. The bone is very well preserved, although stained black by the surrounding matrix. The skeleton is almost complete, although the cranium is partly damaged. He was of average size and, apart from degeneration of the upper right spine, appears otherwise healthy.

Burial 101 MP2, 18m. Fragments of the cranium and mandible, and a few long bones are present. The child was anaemic, with hypertrophy of the zygomatic bones which suggests the maxillary hypertrophy typical of severe thalassaemia.

Burial 102 MP2, 35y, female. The skeleton, including the cranium, is almost complete and the bone very well preserved. She had suffered growth disruption in infancy and was one of the shorter females, with thin cortical bone. There is evidence on the pelvis that she had borne children and an infant, B104, was interred in the same grave. Apart from minor degeneration of the spine and knees, her only obvious health problem was numerous dental caries with associated chronic periapical inflammation. The musculature of her legs was well developed but her upper body was gracile.

Burial 103 MP3, 26y, male. The skeleton is almost complete, although the cranium is fragmented and the bone not well preserved. Apart from the immature B92, he is the smallest male in MP3. Although he had light cribra orbitalia, he had very thick cortical bone. His cranium was robust, and the muscle attachments of his arms and hands were well developed. He had little skeletal or dental degeneration, except in association with defective enamel on the third molars.

Burial 104 MP2. This infant died at birth, and is represented by long bones, fragments of the cranium and ribs. The interment was in the same grave as B102, an adult female.

Burial 105 MP2, 3–4m. The skeleton is represented by fragments of the cranium and some of the long bones. Despite the early age at death, the sphenoid and frontal cranium were hypertrophied, suggesting the first stages of bony response to severe anaemia.

Burial 106 MP2, 6m. The grave was badly disturbed and the skeleton represented by fragments of the cranium and a few long bones.

Burial 107 MP2, 40y, female. The skeleton is almost complete, although the cranium is damaged. The bone is in good condition but heavily coated. She had evidence on the bones and teeth of multiple episodes of growth disruption during childhood, was of average stature but light in weight. Very thin cortical bone, minimal muscular development and little joint degeneration suggest a relatively inactive, probably unhealthy adult life. She suffered from severe and multiple dental problems.

Burial 108 MP2. This infant died at birth, and is represented by long bones and fragments of the cranium. The body was buried on its back, with the legs flexed.

Burial 109 MP2, 31y, female. The bone is not well preserved and was heavily encrusted. There is considerable postmortem damage, although parts of most bones are present and some bones are complete. She had suffered growth disruption during infancy and adolescence but was of average size for the group, with good cortical structure. There is minimal joint degeneration, and she was not muscular. Apart from some dental problems, she appears to have been healthy in adulthood.

Burial 110 MP2, 36y, female. This skeleton is almost complete, and the bone well preserved. The cranium is complete. She had suffered growth disruption during infancy and childhood and was one of the smaller women, although the cortical structure was good. Degeneration of the joints was minimal, apart from the right shoulder. The left clavicle had been fractured at some time in the past, and had healed but was misshapen, possibly with a slight chronic infection. The arthritis in the right shoulder may have been in response to adjustments required to compensate for the broken left clavicle. She had comparatively minor dental problems. She was not muscular and may not have been physically very active.

Burial 111 MP2. This infant died at birth, and is represented by long bones and fragments of the cranium. The grave had been disturbed.

Burial 112 MP2, 40y, female. The skeleton is largely complete, although some long bones are damaged and the cranium is crushed and fragmented. The bone fabric is moderately well preserved but heavily encrusted, which caused problems with cleaning. She was one of the larger women, and the tallest in MP2 with strong cortical structure. She suffered from degeneration in the upper spine and the lower limbs. There is evidence for muscle development in the upper limbs and this, together with the pattern of spinal degeneration, suggests physical activity involving the upper body. She had extensive dental caries with associated periapical inflammation and tooth loss. She appears to have had a healthy childhood but was developing the degenerative problems of advanced years.

Burial 113 MP2, 46y, female. This woman was among the longest-surviving in the group. Parts of most bones are present, although damaged, and the cranium is crushed and fragmented. The bone is very badly preserved, is brittle and heavily coated. She was of average stature. She suffered from degeneration in the spine, and both upper and lower limbs. The knees were severely affected. There is minimal evidence of muscular development. She had severe dental problems, had lost many teeth during life, with subsequent extensive alveolar resorption, and there were caries in her remaining teeth. The degeneration is a reflection of her advancing years, rather than a necessarily active life.

Burial 114 MP2. This infant died at birth, and is represented by long bones and fragments of the cranium. The grave had been disturbed.

Burial 115 MP2, 38y, male. The skeleton is almost complete, although the cranium is crushed and fragmented and many teeth were lost postmortem. The bone fabric is very well preserved, but heavily coated. The bones and teeth show evidence of growth disruption during infancy but as he was one of the large males in the group, and second tallest in MP2, catch-up growth was adequate. Good cortical structure supports this hypothesis. All regions of the spine showed severe degeneration, particularly on the upper right where the costal artic-

ulations were severely affected and the costal cartilages had ossified, to the extent that two of the ribs were fused to the spine. The limb joints showed arthritic degeneration in the shoulders, elbows, wrists, hands and knees. The pattern of degeneration and muscular development of the shoulders, upper and lower limbs suggest a physically very active life, using the upper body in particular. The dentition shows he had been eating an abrasive diet and had relatively few caries.

Burial 116 MP2. This infant died at birth, and is represented by long bones and fragments of the cranium. The legs were flexed.

Burial 117 MP2, 16y, male. The skeleton is almost complete, but many of the bones, especially in the area of the thorax, have been cemented together. The cranium is complete but distorted. The bone fabric is well preserved but partly remineralised. The bones and teeth show evidence of growth disruption in infancy and early adolescence. Little else can be said about this individual because of his immaturity. There is no indication of ill health which may have precipitated his early death.

Burial 118 MP2. This infant died at birth, and is represented by long bones and fragments of the cranium. The grave was disturbed.

Burial 119 MP2. This infant died at birth, and is represented by long bones, fragments of the cranium and ribs. The legs were flexed.

Burial 120 MP2, 21y, male. The skeleton is almost complete, although the cranium is fragmented. The bone is very well preserved and partly remineralised. The lower legs have been stained by the surrounding matrix. He was of average size for the group, and had strong cortical bone. There is no evidence of bony degeneration. The muscular attachments of the forearms and hands are well developed, despite his youth, suggesting an active life using the upper limbs.

Burial 121 MP2, 15m. The grave was largely disturbed by a later pit. The anterior teeth have dark brown linear stains reflecting pre-natal growth disruption, with caries subsequently formed in the stained area on one tooth. The bone cortices are very thin, suggesting juvenile osteoporosis. Hypertrophy of many of the cranio-facial bones and severe cribra orbitalia suggest that the child was anaemic, probably haemoglobinopathic, and this was almost certainly the primary cause of death.

Burial 122 MP2, 52y, female. This woman was the longest-surviving person of the group. The bone is poorly preserved and very brittle, with a heavy coating. Despite this, parts of most bones are present. The cranium is fragmented and distorted. She was of average size, and had borne a child or children. The cortical bone is thin, possibly reflecting age-related osteoporosis, but the poor preservation limits other observations of degeneration. Those observations which were made showed severe degeneration of the lumbar spine, including collapse of the second lumbar vertebra, and arthritis of several major limb joints. The few observations of muscular development suggest that she had been physically active, using her shoulders, forearms, and legs. She was almost edentulous, and the few remaining teeth were very worn. She had suffered from periapical inflammation.

Burial 123 MP2, 2y. The skeleton is represented by some of the long bones and fragments of the cranium and axial skeleton. The bone is in good condition, although some bones are cemented together. The bones show evidence of growth disruption at about age one, and there is a discrete area of defective enamel on one canine. The child had severe cribra orbitalia. There is an area of reactive bone on the right maxilla, which appears to be an infection associated with the fully erupted first molar. The infection may have been the ultimate cause of death.

Burial 124 MP2. This infant died at birth, and is represented by long bones and fragments of the cranium.

Burial 125 MP2. This infant died at birth, and is represented by long bones and fragments of the cranium. The legs were flexed.

Burial 126 MP2. This infant died at birth, and is represented by long bones, fragments of the cranium and ribs. The grave was partially disturbed.

Burial 127 MP2. This infant died at birth, and is represented by long bones, fragments of the cranium and ribs. The grave was very shallow, and the skeleton had been disturbed.

Burial 128 MP2. This infant died at birth, and is represented by long bones, fragments of the cranium and ribs. The grave had been disturbed.

Burial 129 MP2, 27y, male. The skeleton is virtually complete, although the cranium is fragmented. The bone is partly remineralised and very well preserved, although stained by the surrounding matrix. Together with B131, he was one of the shortest males, although of average mass for his height. The bones and teeth suggest growth disruption during infancy and adolescence, although the cortical bone is well developed. He had degeneration of the thoracic spine, especially on the right. He had arthritis in the left shoulder, and severe arthritis of the left ankle, which may have been secondary to an earlier trauma. There was evidence of a healed lesion on the anterior left tibia, possibly from the same event. The evidence of vigorous activity involving the upper body is similar in pattern to Bs 131 and 132. He had impacted third molars which would have caused dental problems had he lived longer.

Burial 130 MP2, 6m. The grave was slightly disturbed, and the skeleton is represented by a few long bones and cranial fragments.

Burial 131 MP2, 25y, male. The skeleton is almost complete, although the cranium is fragmented. The bone is well preserved, but coated with calcareous deposits. He had suffered multiple episodes of growth disruption during infancy and later childhood and was one of the shortest males, although of average mass for his height. The cortices were not well developed. There is slight degeneration on all regions of the spine, except the lower thoracic where two vertebrae almost fused on the right side. He also had some arthritis in the shoulders and hands. The pattern of degeneration in the upper body suggests vigorous physical activity in a similar pattern to that in other adult males, and there is some muscular development in his shoulders which corresponds with this. His teeth were in good condition.

Burial 132 MP2, 34y, male. The skeleton, including the cranium, is almost complete. The bone is well preserved, but heavily coated. He was of average size for the group and the mortuary phase. The bones and teeth show evidence of multiple episodes of growth disruption during infancy and the cortices were not well developed. He suffered from extreme degeneration of the neck and the lower right thoracic spine. He also had arthritis in the shoulders, knees and ankles (extreme on the left ankle). The pattern of degeneration and the development of muscularity on the humerus and lower limb suggests he had led a life of vigorous physical activity.

Burial 133 MP2, 3–4m. The grave was slightly disturbed, and the skeleton is represented by some of the long bones and cranial fragments.

Burial 134 MP2, 6m. The grave was slightly disturbed, and the skeleton is represented by some of the long bones and cranial fragments.

Burial 135 MP2, 3–4m. The grave was disturbed, and the skeleton is represented by cranial fragments.

Burial 136 MP2. This infant died at birth, and is represented by long bones, fragments of the cranium and ribs. The burial was over the legs of B137, in the same grave. The lower body had been disturbed.

Burial 137 MP2, 5y. Parts of most bones are present. The bone fabric is very well preserved but encrusted, the cranium is crushed and bones are cemented together. An infant, B136, was interred in the same grave. The long bones show evidence of multiple episodes of growth disruption between the ages of one and three years, but the dentition has no visible enamel hypoplasia. The child appears to have been healthy during early infancy but to have had a chronic health problem prior to death.

Burial 138 MP2. This infant died at birth, and is represented by long bones, fragments of the cranium and ribs. The grave was very shallow, and the body interred with the legs flexed.

Burial 139 MP2. This infant died at birth, and is represented by long bones, fragments of the cranium and ribs.

Burial 140 MP2, 17y, female. The skeleton is almost complete, including the cranium. The bone is very well preserved and partly remineralised. She had borne a child, and was almost skeletally mature. She was of slightly below average size for the group. The teeth showed evidence of episodes of growth disruption during infancy but she was healthy during later childhood.

Burial 141 MP2. This infant died at birth, and is represented by long bones, fragments of the cranium and ribs.

Burial 142 MP2, 31y, female. The bone is well preserved and partly remineralised, but heavily coated. The skeleton is almost complete but the cranium is very distorted and heavily encrusted. She was of average size for the group. There is no evidence for pregnancy, although the pubic bones are damaged so this is not conclusive. She suffered from minimal joint degeneration and was healthy during adulthood.

Burial 143 MP2. This infant died at birth, and is represented by long bones, fragments of the cranium and ribs.

Burial 144 MP2. This infant died at birth, and is represented by fragments of the cranium and postcranial skeleton.

Burial 145 MP2, 8y. The skeleton, including the cranium, is virtually complete. The bone is very well preserved, and is partly remineralised. There is light cribra orbitalia and evidence of growth disruption during early infancy. The deciduous canines have areas of defective enamel.

Burial 146 MP2. This infant died at birth, and is represented by fragments of the cranium and postcranial skeleton.

Burial 147 MP1, 19y, male. Parts of most bones are present. The bone fabric is well preserved, although coated and stained by the surrounding matrix. Despite episodes of growth disruption during infancy, he was the tallest person of the entire sample. He was not muscular, although this may reflect his youth to some extent. There is no evidence to explain his early death.

Burials 148, 149. Two infant burials just visible in the section. No bone was recovered.

Burial 150 MP1, 2.5y. This infant was buried in a crouched position in a shallow grave. The skeleton is represented by cranial fragments and a few long bones. The bone fabric is well preserved, although some bones are broken. The infant had been severely anaemic, the bones were porotic and there had been episodes of growth disruption. A number of the teeth have areas of defective enamel, in some cases with associated caries. All the evidence of ill health is potentially attributable to the anaemia, which was most likely to have reflected one of the haemoglobinopathies. This would have contributed to the short life span of the child.

Burial 151 MP1. This infant died at birth, and is represented by fragments of the cranium and postcranial skeleton. The grave was shallow and had been disturbed. No evidence of cause of death was observed.

Burial 152 MP1, 37y, male. This skeleton is largely complete, although the cranium is fragmented and heavily encrusted, with many teeth lost or damaged postmortem. The bone fabric is well preserved, apart from the lower limbs which were coated and damaged. He was shorter than the other two adults in MP1 but of average size for the group with exceptionally thick cortices, which may have been pathological but remain undiagnosed. He had suffered from moderate degeneration in the lower spine, the shoulders and one knee. His teeth were very worn, and he had caries and periapical inflammation. Although the pattern of joint degeneration was similar to that seen in males in subsequent mortuary phases, he was not muscular, suggesting a comparatively less active life.

Burial 153 MP1. This infant died at birth, and is represented by fragments of the cranium and postcranial skeleton. The grave was shallow and had been disturbed. It was the earliest burial in the excavation square. No other observations could be made.

Burial 154 MP1, 25y, female. Parts of most bones are present, although the long bones are represented by broken shafts and the cranium and vertebrae are fragmented. The bone is brittle and not well preserved, with postmortem damage and black staining from the surrounding matrix. She had suffered from growth disruption during infancy but was nevertheless one of the tallest females in the group. She showed evidence of pregnancy. From the few observations which were possible, she did not have strong muscular development but did have one arthritic knee. She was otherwise healthy apart from dental caries with periapical inflammation and loss of teeth during life.

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