Skeletons and Social Composition

Bahrain 300 BC - AD 250

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Abstract

This book proposes a model of skeletal analysis aimed at the reconstruction of past social composition. The model comprises five basic steps: recording of age, sex and pathology; analysis of demographic structure using life tables; identification of pathological conditions through differential diagnosis; analysis of the causes of death; and finally, interpretation of these results in terms of the experience of each age class. The end result is an analysis aimed beyond the comparison of percentages between skeletal samples to a reconstruction of the living society. This picture is then open to comparison with ethnographic as well as archaeological sources of evidence.

In order to test this analytical model and its ability to address questions of biocultural adaptation, two groups of skeletons from Bahrain, the Arabian Gulf, were examined. These date from c 300 BC to AD 200 and come from two cemeteries: DS3 and Saar. Analysis of palaeodemography and palaeopathology indicates that these two populations faced difficulties due to the agricultural environment in which they lived. Levels of morbidity and mortality were high during early childhood and later again in the young adult ages. The older age groups were affected by skeletal fluorosis. This particular combination of disease and causes of death determines a population structure which, despite higher mortality levels (particularly amongst adults) does not vary greatly from small agricultural populations today. Such a population, however, is extremely vulnerable to economic and environmental crises suggesting that the evident regional continuity is based upon discontinuous local histories.

The application of the model demonstrates that a joint palaeopathological and palaeodemographic analysis upon a single skeletal population can result in a reconstruction of the past living society; that skeletal analysis can present a dynamic model of past social and environmental networks.

Chapter 1

Introduction

Humans are constrained by biology and environment. A large proportion of human history is the result of individual and social adaptations to these constraints. Two factors, in particular, have been the focus of numerous studies in physical anthropology: disease and demography.

These studies have begun with a biocultural viewpoint. Simply stated, each individual responds to biology and culture by a variety of adaptative mechanisms. Change in either sphere (stress) forces an individual response, aimed at adapting to the new conditions. In examining past societies the aim is to document what changes in biology and culture occurred, how these changes affected individuals, and how people coped in response to the changed conditions.

Physical anthropology and archaeology have addressed aspects of these questions. Much physical anthropology, however, has been directed at chronological or spatial comparisons between societies and hence with environmental reconstruction, rather than examining individual groups in detail. Despite the reliance of physical anthropology upon biocultural models, the field lacks a middle range theory allowing us to move from essentially observation of the dead to a living past society. The lack of such a mode of analysis means that much work is, of necessity, restricted to the comparison of percentages between populations.

There has been very little sense of what these results meant in terms of the past life of the people we study: what does it mean on a social level if 90% of children who died had cribra orbitalia, or, economically, if 50% of the population died before 15 years of age? In attempting to answer these questions we have the power to assess our results, make these results accessible to others, and begin to assemble an accurate picture of the constraints of past societies. In short, to ask more anthropological questions about the past.

This work aims to present a model of analysis which bridges this gap. Necessarily, any such analysis is aimed at writing a 'particular history' (Trigger 1978): a history of the interaction of population, human biology and society. Particular histories, rather than generalizing formulae, are the appropriate format for addressing issues of adaptation in this instance. As Trigger states:

> The aim of any historical discipline is not only to describe but also to interpret specific events... The particularizing nature of such a task does not imply a lack of concern with theory, but indicates that within prehistory theoretic formulations should be sought in order to explain events, rather than as ends in themselves (Trigger 1978:36).

Of central importance to this analysis is the realisation that the reconstruction of past disease and population conditions cannot proceed without paying some attention to the nature of the society and the living conditions our analyses imply; that if there is no mechanism for our results to be validated by, and, in turn, to test others (as by historical or ethnographic comparison) then their validity for research both on prehistoric and modern groups is limited. Unless there is a linking mechanism between the skeletal group and the living population it represents, our analyses will always be limited to the comparison of percentages between skeletal samples.

In order to write such particular histories, and to address the nature of human adaptation, a model of analysis is proposed. The frame of the model is partly determined by the issues outlined by Howell in concern to demography. As she states, an issue analytical models need to address is:

> how to measure or estimate the parameters from existing information, how to assess the plausibility or accuracy of findings, and how to correct for under-reporting, misestimation, or random fluctuations in the small numbers we have to work with in order to turn out useful results... to assess how much simplification of demographic methodology is possible in this kind of situation, while at the same time, retaining a level of accuracy high enough that the studies are worthwhile doing (Howell 1973:250).

Palaeopathologic methodology could equally well be substituted in this statement.

The analysis proposed here consists of five basic steps or proposals:

1. Assessment of the sample and recording of basic skeletal attributes: age, sex, metric characteristics and palaeopathological lesions.
2. Establish social composition from life table methods (Howell 1982).
3. Pathological conditions can be identified by careful consideration of the skeletal lesions (Buikstra 1977).
4. The combined results of demography and pathology can identify underlying disease processes (Buikstra 1977) and the age structure of the population can be explained by examining the effect of pathology (Palkovich 1978).
5. The results of these analyses can be assessed in terms of the experience of each age class and compared with ethnographic data and archaeological inference.

In sum, the process is to (1) record, (2) analyse a life table, (3) attempt differential diagnosis of any lesions, (4) analyse the cause of death and (5) interpret the results via a consideration of survivorship.

The following work is concerned with explaining this model, that is, its place within current approaches to

palaeodemography and palaeopathology, and with demonstrating its applicability to skeletal research by means of a case study. The skeletal sample used as an example is the result of two cemetery excavations on Bahrain, Arabian Gulf, dating to c300 BC–AD 250.

The power of this model of analysis is demonstrated by its ability to explain the nature of agriculture life on Bahrain and the constraints upon development, and to challenge and expand current archaeological hypotheses concerning settlement on the Island.

The organisation of the following chapters follows this process. In Chapter 2 the evolution of the theoretical model is traced. The model is then applied: Chapters 3 and 4 deal with the archaeology and Island and the nature of the samples; Chapter 5 is an outline of the processes involved in recording the skeletal information (Step 1); Step 2, life table analysis, is undertaken in Chapter 6; palaeopathological lesions are analysed in Chapters 7 and 8 (Step 3 of the model); in Chapter 9 the causes of death are examined (Step 4); while the conclusion to the case study (Chapter 10) is an interpretation of the results via a consideration of survivorship (Step 5). In Chapter 11, the value of the analytical model and the questions its use provokes will be addressed.

Chapter 2

The analytical model and its basis

Introduction

The proposed analysis consists of five basic steps. The result is a model of social organization which can be used to address such issues as the nature of the economy, the interaction between population and environment, and the reasons for cultural or economic change.

Broken into its constituent parts, no single aspect of this analysis is totally new. Each step is based upon a number of studies dealing with both theoretical and methodological issues. The aim of this chapter is to examine these bases and explain the model fully. General methodology will be dealt with in this chapter but more specific techniques (such as the recording of hypoplasia) will be discussed later as the model is actually applied.

Recording of age, sex, and pathology

Much of the debate in both palaeodemography and palaeopathology has concentrated upon the development of appropriate methods. Even now there is still no accepted corpus of methodological techniques to which one automatically turns when confronted with a skeletal sample. Given the proposed analytical model, it is necessary to outline the baseline assumptions the model rests upon and examine the basic principles behind age and sex estimation and their application to skeletal samples.

Baseline assumptions

Behind any work lies the assumption that the basic processes of human biology such as ageing, maximum reproductive life span, and generalized disease response, have remained constant within *Homo sapiens*. Any demonstrated variation is seen as response to environmental factors. This theory, as it applies to palaeodemography, was first explicitly outlined by Howell (1976) under the title of uniformitarianism.

Unless we accept this assumption there is no basis for estimating age, sex or any demographic parameters; without it we cannot apply standards of age or sex developed from modern populations to those in the past. Similarly in palaeopathology we could not assume that skeletal lesions are caused by the same biological mechanisms seen in the present.

A cautionary note needs to be added, however. Some parameters have been known to change. For instance, life expectancy has increased while the age of menarche has decreased. We therefore need to be careful to highlight areas of our estimations where extrapolation from the present may not be entirely applicable. There is, however, the understanding that the basic biological processes of *Homo sapiens* have not significantly changed till now.

Representativeness

The second issue is representativeness of the sample. Does the collection accurately reflect the living population who used the cemetery? It needs to be remembered that there are three stages prior to archaeological recording of skeletons and at each stage intervening factors may operate:

1. Pre-interment—selection of individuals for burial, and prior processing (eg. exposure).
2. Interment—preservation, disturbance.
3. Disinterment—archaeological methods, the extent of excavation etc.

Each of these factors needs to be taken into account and their examination constitutes an import part of the first phase of analysis.

Pre-interment factors which may bias a skeletal sample are those decisions made by the living on treatment of the dead. For example, preferential burial may operate as a biasing factor, as when only adult males are buried (eg. Haglund 1976). The issue is whether the full range of burial patterns within a society is known, and whether the skeletal sample represents this full range. It is less important to demonstrate that it does, than to demonstrate what part of a living population the skeletal sample represents: to know the shortcomings of the sample.

Interment factors are those affecting skeletal preservation after burial. These are primarily taphonomic issues such as differential preservation within different areas of the cemetery, decay, and its relationship to age, sex, and other personal characteristics. Again it is important to identify possible biasing factors and attempt to account for them.

The final stage at which bias can occur prior to recording is during excavation. The factors to be accounted for here are the extent of excavation (is it sufficient to warrant a claim of representativeness); sampling techniques (all areas of the cemetery or only some); recognition of remains; and recovery which can damage fragile small bones or may operate in favour of recognisable parts such as crania.

The discovery that any of the above factors operate may not necessarily invalidate the analysis (see Buikstra 1981). Yet it does set limits upon what inferences can be drawn, particularly in terms of palaeodemography (eg. Lukacs 1994). Therefore, implicit within the first stage of the proposal is a consideration of the context of the skeletal sample and its representativeness. This is one of the primary tasks.

Ageing and sexing

The reliability of any palaeodemographic study depends on the accuracy of methods used for ageing and sexing, as well as representativeness of the sample. Ageing and sexing methods, in particular, have been discussed in several publications, most noticeably those by Bocquet-Appel and Masset (1982, 1985), who claim that the resultant age and sex distributions mirror the reference sample upon which the method was based. More recently work by Koningsberg and Frankenberg (1994) and Jackes (1992) confirms these criticisms. They make very different suggestions, however, on mitigating the issue. Koningsberg and Bocquet-Appel suggest statistical iterative techniques for age estimation in order to obtain both a measure of accuracy and estimates less dependent on the reference sample. Their methodology at the moment, however, requires samples for which there can be either perfect use of a single method of age. Alternatively baseline data for the correlations between multiple indicators is needed. Currently this is not available. Jackes (1992), on the other hand, suggests using ratios in order to identify outlying populations.

Age estimations of subadults and the identification of sex among adults are the least problemmatic areas of estimation. Numerous methods have been developed for adult ageing (Iscan and Loth 1989) and all subsequently criticised. The necessity, apart from obtaining the most accurate estimate is to avoid any systematic bias inherent within the method.

One way of minimising this problem has been to use multiple methods: estimating a final age by means of weighted averages (Acsadi and Nemereski 1970; Lovejoy et al. 1985). Supposedly, by systematically incorporating more than one method both these multifactorial techniques should avoid the systematic bias present in any single method, unless its effect is cumulative.

It is claimed that multifactorial methods can account for differential preservation. If all four indicators cannot be recorded, a different formula using only those indicators available may be used. Yet with fewer indicators the degree of accuracy declines. Since the present sample consists of a mixture of complete to very fragmentary material, it was realised that many age estimates would have to rely upon a single method thereby rendering the formal application of a multifactorial approach useless. While some methods are more accurate than others, and while the best solution is to use as many methods as possible, which methods are finally used is always determined by the practical constraints of skeletal completeness, time, and equipment.

There are three general points to skeletal ageing. Firstly the use of multiple methods is preferable. Yet, as recommended by Steele and Bramblett (1988), the full range of possible ages needs to be used rather than an exact age. Finally accuracy is a factor of sample size. While ageing of individual skeletons may be inaccurate, the larger the sample size the close the fit between estimated age and real age of the sample.

Obviously, while it is desirable to have age and sex estimates for every individual within a sample, spurious estimates are useless. Rather a conservative approach is desirable. In addition, the more standardized the approach, the better the degree of comparability between standards.

The principles that need to be adhered to in this first phase are:

1. assess the representativeness of the sample and take account of its analytical limitations;
2. not to underestimate the range of possible ages which apply in each case;
3. use multiple methods of age estimation if feasible;
4. it is better to be conservative than to obtain spurious results.

Obtaining a schedule of age and sex for all individuals within a sample is merely the first stage of analysis. At this same recording phase pathological lesions need to be noted. The recording of pathology is again determined by the sample itself although general principles may be observed. The reason for these will be discussed further in step 3.

Establish social composition from life table methods

Palaeodemography vs demography

Palaeodemographers have similar concerns to demographers. After all, palaeodemographers constantly work with incomplete data. The current trend of papers demonstrates the concern with accurate recording above all else. Yet there are major differences. Demographers work from a series of data sources: censuses of the living, fertility surveys, birth and death registrations. Palaeodemographers work with a single set of data—the age at death. Any other population parameters must be estimated either from the age at death data itself, or from accompanying archaeological or skeletal data. Compared to the demographer, the palaeodemographer has a much narrower range of methods available.

The other major difference is that demographers tend to work with large national or regional populations where the next decimal point is actually meaningful. The populations in palaeodemographic studies are small local groups liable to extinction or rapid growth merely through stochastic variation. This combination of incomplete data and small populations make palaeodemography as one researcher put it:

> a subject which is simultaneously intensely interesting and devilishly difficult (Howell 1976:25).

Yet the restrictions of palaeodemography do not exclude the use of demographic methods. Rather the methods must be modified to accommodate the data restrictions imposed by using dead rather than living people.

Demographic theory states that a population is the end result of three processes: fertility, mortality, and migration, which operate upon the original age distribution (Coale 1972). A population with constant fertility and mortality rates is demographically stable; although it may be growing or declining, the various proportions of age classes will remain the same. This means that it is possible to estimate the remaining parameters given a combination of any two of the following: fertility rate, mortality rate, growth rate, age distribution of the living, or age distribution of the dead. It is this stable population theory, which forms the basis of the life table (Coale 1972).

A stable population is not necessarily stationary. Since in palaeodemography there is only one parameter—age at death—stable population theory is inapplicable unless it is assumed that the growth rate is zero. Assuming a stationary case, it is then possible to construct a life table and hence estimate other population parameters.

Life table analysis in palaeodemography uses two techniques not commonly employed in modern demography. One is a synthetic abridged cohort. Life tables generally are constructed using either the experience of a single birth cohort (a so-called generation table) or using a synthetic cohort, i.e. all those alive in one particular year. The latter assumes that all members of a population have undergone the same mortality and fertility experiences and can therefore be congregated into a single table. Unless one is using gravestone data, the former course is not possible, therefore palaeodemographers generally use the concept of a synthetic cohort where it is assumed that all individuals represented in the sample were born in year 0 (Weiss 1973). This assumption is untenable in conditions where the population numbers are unstable from year to year.

The other device used by palaeodemographers is that the table is constructed using a D_x series (i.e. the number of deaths/ age), rather than a L_x series (the number alive at each age). As a result, the derivation of formulae are slightly different. Using D_x as the basis of a life table is a device of biologists studying animal populations (Caughley 1966).

Palaeodemography, therefore, is not linked with demography solely through common concerns. Rather, there is a borrowing of several baseline assumptions, most importantly stable population theory. The techniques for using the theory then become a mixture of standard demographic techniques and those modified for skeletal work.

The life table method

The adoption of these methods is not without controversy and the approach has been criticised on several grounds:

1. The assumption of stability for small populations.
2. A persistent tendency in skeletal groups to under-enumerate certain age classes.
3. The assumption of no net migration.
4. The assumption of a zero rate of growth.

Small sample size

The small sample size of groups being studied is a primary problem common to most methods of analysis. Stochastic fluctuations affect small populations; mathematical models have demonstrated that, unless a population maintains a slightly positive rate of growth, it will head toward extinction (Weiss and Smouse 1976). The question is whether this translates in practice, since it is possible for a group (at least on an individual basis) to consciously ameliorate demographic trends. To some extent the question of stochastic fluctuations remains unsolved since it is unknown whether changes in a group's demographic behaviour are in response to underlying rates or to these stochastic occurrences. So far, since skeletal samples can rarely be isolated into narrow time frames for comparative purposes, the issue of stochastic change has had to be largely ignored or dealt with by simulation techniques (Roth 1992).

The assumption of stability has, on the other hand, been studied with the use of computer simulation. Weiss, modelling such fluctuations, has demonstrated that a population, preferably not smaller than 100, can recover from a periodic episode within approximately 10 to 20 years depending upon which age group is primarily affected (Weiss 1975). Similar results are seen in ethnographic work (Howell 1979). The conclusions of Weiss (1975) are that, if the original population was sufficiently large and if the basic patterns of mortality and fertility had prevailed for several decades, then the assumption of stability may be safely made. The proviso is that the population is not known to have suffered any systematic disruptions in its recent past. Such an assumption requires assessment of the archaeological record for evidence of any disruption. As one researcher points out:

> The implication is that, if used in isolation from a study of other factors—ecological and cultural—a life table treatment of osteological data may prove misleading. (Hall 1978:728)

Sample under-enumeration

Other problems with the life table include a tendency in skeletal samples for the under-enumeration of certain age classes. Most commonly it is the youngest age group that is missing. The primary reason for concern with under-enumeration is that many life table functions, most noticeably mortality, survivorship, and life expectancy, are cumulative and therefore subject to errors due to loss of the youngest age group (Moore et al. 1975). One solution is to avoid comparison on the basis of these parameters; this, however, seriously limits the value of the life table (see also Roth 1992).

Methods of testing for under enumeration have been tried (Brothwell 1981, Jackes 1992). Alternatively, reference model life tables can be used and the missing infants estimated from tables that match parameters such as the mortality rate of higher age groups. It needs to be remembered though that this may serve to mask any real differences in mortality patterns. Preferably the burial

practices and recovery techniques should be examined closely to assess whether there is justification in assuming infants are underrepresented (as recommended in Step 1).

Net migration

No net migration has been the most readily accepted of all the assumptions concerning life table usage. In small settlements it is accepted that the most common form of migration is reciprocal. This assumption may well fail to hold in urbanized areas where young people migrate in, or in economically depressed or militarized areas where young men tend to be mobile.

In the case of out-migration, the population will tend to show high infant and old adult mortality relative to young adult mortality (eg. Buikstra 1981). This can probably be differentiated from a high fertility population where mortality only amongst the youngest cohorts is elevated. In in-migration the reverse is true: mortality in the young adult categories should be elevated relative to mortality of the youngest and oldest age groups. In addition, there may be supporting pathological evidence since migration into urbanized areas, at times, occurred because the workforce could absorb sufficient labour: evidence that the population is either not replacing itself or not growing fast enough. Obviously, if a population has an unexpected age pattern, then the possibility of migration needs to be seriously considered.

Stationarity

The final assumption, a stationary growth rate, is the most problematic. The simplifying assumption, that population growth rate (r) is nil, means that using the formula births equal deaths enables direct estimation of the age distribution of the living from the dead. Yet in a population that is either systematically growing or declining the formula must incorporate the rate of positive or negative growth. Failure to account for a growth rate leads to misestimation of the survivorship functions of the life table (Bennett 1973, Moore et al. 1975).

More recently Wood et al. (1992) following the work of Sattenspiel and Harpending (1983) point out that demographic parameters are more overtly fertility rather than mortality driven. For instance mean age at death is correlated to fertility. This seemingly contradictory effect can only be adequately dealt with by some form of estimating either the growth rate or fertility rate.

From the beginning inability to account for population growth has been identified as a major problem is the use of life tables. Nevertheless life tables are still constructed using a stationary growth rate (eg. Mensforth 1990). The commonly used explanation is that population rates prior to the industrial revolution were seldom more than 0.01% (Acsadi and Nemeskeri 1970; Hassan 1981). Yet the aim of palaeodemography is to examine change over time; analysis cannot be satisfactorily conducted by making stationarity an *a priori* assumption. In addition, comparisons between life tables become meaningless if they are wrongly assumed to have a zero growth rate.

Methods of estimating the rate of population growth are fraught with problems. Bennett (1973) attempted two methods of estimating population growth. One, suggested by Carrier (1958), relied upon comparison with the proportions of age at death and survivorship in model life tables to estimate a rate of growth. Bennett found this unsatisfactory since the shape of the mortality curve which forms the basis of the UN tables failed to correspond to his prehistoric sample (Bennet 1973). (Carrier, himself, warned of this possibility; Carrier 1958) In the second method, Bennett attempted with limited success to estimate a growth rate on the basis of the archaeological record.

Carrier points out:

> The theory is sound, but it is shown that more often than not, the conditions necessary for the theory to be applicable do not hold. The result of a single calculation should be deemed as carrying no weight, but if the same result is obtained from several calculations it may, with reserve, be advanced tentatively as a working hypothesis. (Carrier 1958:153)

In a surprising number of cases it is also possible to work out from the archaeological data limiting assumptions of growth, especially for samples covering long periods of time. As with Carrier's method, this then allows for a working hypothesis which is certainly an improvement on pretending that the issue does not exist.

Ways of coping with or without life tables

Certainly many studies, while admitting the above problems, still adopt life table methods. Angel has suggested an alternative approach (Angel 1969). Distrusting the assumptions inherent in life tables, he used a combination of the age at death ratios and fertility estimates based on pelvic morphology. From such data he estimated the average number of births per female, generation length, birth and death rates, household and population size. The approach, while simple, has been invalidated by the problems of estimating fertility on the basis of trauma on the pelvis (Holt 1978). Nevertheless, Angel's use of age at death ratios and interpretation of the results in terms of family structure are important indications of where the field could be heading.

His work has not been followed by similar approaches. The majority of researchers have chosen to work with life tables. Generally such studies have used reference model life tables which present the average mortality experience of groups of populations having various levels of life expectancy and survivorship. These tables are used with three aims: to identify bias in the samples, to estimate shortfalls from life

tables, or else to use those parameters suggested by each life table.

The model life table approach

The approach is not, of course, without problems. One is the truncation of older age groups since most methods of ageing skeletons do not allow estimates beyond 50 or 60 years of age. Above this age there is a lack of correspondence between the skeletal group and model life tables unless some estimation is made of the maximum life span. A variety of methods, of varying success, have been suggested to cope with this (eg. Asch 1976, Mobley 1980). Wherever possible it could well be better to ignore this final age class rather than apply an unknown value. This is not alway possible, thus alternative values are frequently used (eg. 50–70 yrs, 50–80 yrs) or the ages are simply truncated (50+ years).

A more important issue is that three separate groups of reference life tables have been used in palaeodemography studies: the UN life tables (UN 1982), Coale and Demeney's model life tables (Coale and Demeney 1983), and Weiss's tables of anthropological populations (Weiss 1973). Each collection of life tables is constructed from the average experience of large numbers of populations and is based upon different methods of construction. There are advantages and disadvantages to each. For instance, in the UN model life tables, life expectancy does not go below 30 years of age, making them largely inappropriate for comparison with anthropological or skeletal groups (UN 1982).

The Coale and Demeney tables are more commonly used since, by linear extrapolation, tables have been constructed to the lower life expectancy of 15 years (Coale and Demeney 1983). In addition the tables have been subjected to different rates of growth to form a series of model stable populations. The tables are grouped into four regions, north, south, east and west, the first three matching characteristics of particular areas. for example, the North pattern is similar to that of Scandinavian countries. West, in contrast, is based on the largest number of populations who do not correspond to any of the other peculiarities. These tables are more applicable to skeletal data due to the lower life expectancy. Yet the basis of the model 'West' life tables are primarily western populations and their applicability to high mortality populations may be questioned.

The final set of life tables have been constructed by Weiss from a series of anthropological, prehistoric and historical populations (Weiss 1973). To enable greater flexibility Weiss combined different patterns of juvenile mortality (based on survivorship to age 15) with patterns of adult mortality (based on life expectancy at age 15). His tables have a distinctively different mortality curve to those of Coale and Demeney, having greater adult mortality. It is noteworthy that Weiss' tables are based on at least some populations which have the same problems with ageing and bias as any skeletal group. Model life tables are only as good as the data they are based upon.

The point is that, while model life tables are a useful device for identifying disparities in the data, it is questionable how such data should be interpreted. Different sets of life tables have different mortality curves. All correspond to the basic 'U' shape of mortality but, as noted earlier, curves presented by Weiss differ significantly from the Coale and Demeney tables on the basis of adult mortality. If a skeletal group shows evidence of deviation from a normal curve should this be interpreted as a stochastic fluctuation or evidence of a different mortality structure?

Other uses of model life tables

Palkovich, in an analysis which avoids many of the above problems, used model life tables to identify age peaks in the mortality curve (Palkovich 1978). She analysed pathological data to determine whether the pattern of skeletal pathology mirrored this age distribution. Correspondence would suggest that the deviations were reflections of a population specific cause of death structure. The method was used to analyse depopulation in Amerindians. Such analysis avoids many of the problems inherent in life table approaches since it is reliant on the structure of death and pathology. It is, however, less useful in terms of investigating population composition which requires analysis of the hypothesized live age distribution.

Two other approaches have been used to avoid the problems of estimating population growth and of directly using model life tables. One is the use of ratios based on age at death data. This approach was first demonstrated by Bocquet and Masset who isolated the age groups 5–15 and 20+ as the most easily identified and least subject to under-enumeration (Bocquet and Masset 1977, Bocquet-Appel and Masset 1996). They then produced a series of regression equations based on a selection of historical populations, to estimate mortality at age 1, age 5, and the expectation of life. These regressions were calculated for various rates of growth. In a further article Bocquet demonstrated the relationship between fertility and this same ratio given a stationary population (Bocquet-Appel 1979).

A similar approach has been used by Buikstra and coworkers, based on the relationship between fertility and the pattern age at death (Buikstra et al. 1986). Basically, mean age at death is fertility driven: the higher the fertility, the lower the mean age at death. The use of this relationship was initially espoused by Sattenspiel and Harpending (1983) who suggested that it was possible to predict the number of births from life expectancy. Horowitz et al. (1988) have, however, demonstrated that the value of this predictive relationship only holds in certain situations. Buikstra and coauthors (1986), rather than using the relationship for prediction, suggest that the ratio of deaths at 30+ to deaths 5+ (or 20+ to deaths 5+) can be used on a comparative basis between

skeletal groups. This seems to be useful to identify instances of change in fertility over time, in populations with similar mortality curves. It is noticeable, though, that the relationship between the crude birth rate and the inverse of mean age at death is not linear, and becomes less predictable with higher life expectancy. In addition, without independent identification of possible changes in mortality, it may be very difficult in practice to distinguish between a minor change in fertility and a major change in mortality (Ryan Johannson and Horowitz 1986). This leads us back again to the need for a full exploration of other data sources.

A more serious problem is that these perceived relationships are initially the product of the reference model life table. Age at death series which fall outside known parameters of the model life tables used for the original predictions cannot be used in the same way since the underlying regressions are solely the result of which life tables were used. The tendency amongst workers appears to be to accept model life tables as unassailable limits. In reality, each set of model life tables incorporates differing regression coefficients to construct mortality curves. In addition the designers of these tables make no claims that their tables constitute the total of mortality experience; how could they when currently the mortality curve is changing in many places from the basic U shape to a more characteristic J? Given this, we need to be careful of making our uniformitarian assumptions too general.

Finally the relationship between fertility and mortality in these groups is in fact a product of the model itself; the model is a simplifying assumption. While in life tables the mortality and fertility schedules are in a fixed and predictive relationship, the extent to which this is mirrored in biological reality is unknown (see also Wood et al. 1992). Weiss has demonstrated that a small population can, after suffering demographic fluctuations, return to stable rates which hold this predictive relationship (Weiss and Smouse 1976), but still palaeodemographers pay insufficient attention to the possible nature, extent and timing of such relationships.

Nevertheless there are outside limits for human populations imposed by biology (such as the maximum fertility rate given mortality). These rates can be calculated via a life table. In addition the life table itself must be internally consistent. Keeping this in mind, model life tables can be useful if they are treated with the caution they deserve.

Alternative approaches

Some of the problems associated with comparing model life tables characterize alternative approaches to palaeodemography. This is the use of reference model life tables to generate age at death estimates which can then be compared with untransformed skeletal data. The aim, in common with more traditional approaches to model life tables, is to identify bias found in skeletal samples. Secondly it is used to identify demographic parameters without manipulating the skeletal data: to avoid the assumptions inherent in life table construction. To some extent the argument is circular since rather than directly manipulating skeletal data, the work is conducted by analogy: the basic assumptions are still the same.

Possibly a more useful approach is that proposed by Milner and coworkers (Milner et al. 1989) who compare age at death distributions generated from high (called Yanamano) and low (called !Kung) fertility models (based on Coale and Demeney tables). Their comparison is suggestive. It avoids the problems of model life tables which, by averaging the experiences of several populations, are removed one step from reality and which also, by means of their construction, impose certain assumptions of mortality and fertility. This may be the only way in which eventually we shall be able to examine stochastic fluctuation in skeletal groups. Certainly Milner's idea of applying the simplest hypothetical model and then attempting to falsify results has the advantage of sticking closely to the actual data rather than applying methods which impose artificial constructs (Milner et al. 1989, see also Roth 1992).

In cases where it is not possible to estimate a rate of growth this approach is probably the most useful. Alternatively, Ryan, Johansson and Horowitz (1986) suggest that where a rate of growth can be estimated, the life tables constructed by Coale and Demeney for varying rates of growth can be used to reconstruct mortality conditions, while fertility can be indirectly estimated by use of age at death ratios suggested by Buikstra and co-workers (1986). The untransformed age at death data are used as the basis of comparison.

A new attempt

The interesting point about all these approaches, with the exception of Milner (Milner et al. 1989) and Palkovich (1978), is the emphasis placed upon methods to allow comparison between skeletal samples (see also Wood et al. 1992). It is frequently seen as sufficient to present a life table perhaps with a statement on changing fertility or mortality (more commonly now fertility). Skeletal samples are appraised solely on two bases: other skeletal groups or model groups (one of which is based on skeletal samples). Howell noted this in 1982:

> While life tables are commonly produced and presented in palaeodemographic studies, the implications of the life table for the comparison of the living population which produced the cemetery are rarely spelled out in detail (Howell 1982:263).

Since then little has altered. Yet one of the values of demography is its ability to highlight the age distribution of the living population. In addition, a match between a model life table and skeletal sample proves very little since not only are Weiss's tables based on possibly flawed data, but Coale and Demeney constructed their life tables with life expectancies lower than recorded in any of their datum

populations. In fact the only satisfactory method to validate a skeletal sample is by examining the population structure implied by the age at death distribution and comparing this to other sources of data—notably the pathological data, ethnographic comparisons, and archaeological data.

By reconstructing the social composition implied by the demography, the true benefit of the life table approach is realised. It is, indeed, the only way of approaching the once living population. Otherwise, we are left with only comparative studies of change over time.

This does mean that it is necessary to deal with the limitations of life table analysis, especially the issue of population growth. This can be done by estimating, firstly, whether a population is growing or declining, and what are the outside limits of that movement. Until other methods are devised, this can only be done by estimation. The fact that many samples cover several hundred years means that generally any movement in either a negative or positive direction is probably not far removed from zero.

Once an outside limit of growth and decline is estimated the traditional life table methods can be used, based on the series of ages at death (D_x). This allows estimations of the living population's age distribution. In turn it is possible to estimate measures of social composition such as dependency ratios, the amount of orphanhood, widowhood etc. For palaeodemographers the link to social composition is the real advantage of a life table approach. The hypothetical model of social composition can tied in with other sources of skeletal data, primarily the palaeopathological data, in order to validate the model and to add further elaboration.

It is proposed that, in cases where it is possible to estimate outside limits for the population growth rate:

1. The representativeness of the sample is tested (checking for bias due to archaeological processes).
2. It is determined whether conditions satisfying life table construction are present (stability, no net migration).
3. The outside limits of population growth are estimated.
4. Construct a life table with varying rates of growth.
5. Comparison with model groups to compare the mortality curves and highlight anomalies.
6. Estimation of functions using a combination of ratios and life table extrapolation (weaning deaths, kinship, fertility, dependency ratios).

The result is a hypothetical model of social organisation to be tested against other sources of data, especially those from palaeopathology.

Specific methods of palaeodemography

A normal age at death distribution?

Prior to any demographic analysis it is necessary to test the assumption of an unbiased population. Initially the archaeological background of the sample needs to be examined for possible biases due to differential burial for various age/sex groups, preservation and/or recovery. Also, the age at death distribution needs to be checked for possible under or over enumeration. Various methods have been suggested, three of which will be adopted in the current work: the ratio of deaths (Brothwell 1981); comparison of the mortality curve (Buikstra and Miekle 1985; Waldron 1994); and comparison with model life table distributions, bearing in mind the reservations already outlined.

Primarily, any comparison should be based upon either the curve of mortality, since this function is least affected by under-enumeration, or the distribution of ages at death which has the advantage of being untransformed data. The most common method of comparison is by visual curve fitting. Alternatively the ratio of age at death of the least biased age classes (determined above) can be compared (Jackes 1992). Additionally Weiss suggests the use of an index of dissimilarity to test this fit (Weiss 1973) although essentially this is a measure of effective sample size only (Koningsberg and Frankenberg 1994).

The aim of the comparison is to determine which age classes appear biased. Identifying dissimilarities points out areas in the age distribution which need to be explained since they go beyond the common experience. It is, however, most useful to first demonstrate that the population is biologically capable of survival. Reasons for the peculiar age at death structure may then be sought from the pathological and archaeological data.

The ratios based on age at death

Prior to this, the age at death schedule can be the source of useful information.

The ratio of weaning deaths i.e. deaths 0–1 : deaths 0–4 is indicative of relative child mortality (Wills and Waterlow 1958). In high mortality situations the number of children dying during weaning increases proportionally to those dying between 0–1 year. This is a reflection of the effect of malnutrition which is most severe after the first six months of life. The ratio can also be calculated by using the probability of dying between 0–1 compared to 1–4 years (Garenne 1982).

This ratio and in fact the calculation of a rate of growth can be further explained by the derivation of fertility. The ratio of D_{30+}/D_{5+} reflects fertility more strongly than mortality (Buikstra et al. 1986). In a high fertility population the ratio will be low, higher in a low fertility population. In a low mortality population, the ratio will generally be fairly stable and slightly lower than in a stationary case, while in a high mortality population it will be slightly higher. This is on the proviso that changes in mortality apply to all age groups. Use of this measure, apart from allowing comparisons between groups, helps to indicate whether, in the population studied, fertility may have exceeded mortality.

The importance of the actual distribution of deaths itself has to some extent been undervalued. As Angel pointed out, psychologically, death (and the patterns of age at which it is experienced) is important to the society as a whole (Angel 1969). As part of this, the ratio of female to male deaths at varying age groups needs to be studied. In developed countries male mortality during adult years is in excess of female mortality. This is in stark contrast to the majority of skeletal populations, and some underdeveloped areas (eg. Lopez and Ruzicka 1983). The sex ratio of deaths during adulthood is an important reflection of differential mortality.

The estimation of r

The estimation of population growth for archaeological samples is difficult. There are, however, two methods worth attempting and which are probably most effective when used in tandem.

Carrier's (1958) method of selecting a growth rate (restated by Pollard et al. 1974) is presented in Appendix 2. This is based on the assumption that the difference between the quotient of deaths and of survivors reflects r since by rights in a stationary case $l_{40}-l_{30}$ is equivalent to $d_{30\text{-}40}$, any difference therefore represents population growth or decline. This formula is inapplicable in cases where the mortality curve of the model life table does not fit the actual data (eg. Carrier 1958). Yet it may provide a useful comparison to archaeologically derived methods.

Methods based on the archaeological data differ according to the exact nature of that data. One useful way of estimating outside limits of growth or decline is to estimate parameters from the following questions:

1. Was the settlement already established?
2. What is the average population size derived from the cemetery?
3. What length of time did the cemetery cover?
4. Is there a nearby cemetery postdating the cemetery being studied?
5. In this time could the population have doubled...increased tenfold? (In 500 years a tenfold increase is only a 0.004 or 0.4% growth rate).

Given the period of use of the cemetery, settlement survival and average population size, it is possible to begin to look at outside parameters, remembering that a tenfold increase is a major increase of a small population particularly if it is weathering environmental fluctuations.

Finally, it is relevant to compare the resultant age at death distribution with that obtained from the best fitting model life table subjected to the same growth rate. If the fit is better than that of the two stationary populations, or somewhere in between the two, it is suggestive that by estimating a growth rate, a closer approximation of the living population has been achieved.

Life table construction

The methods used in life table construction are based upon those used by Weiss (1973) and Acsadi and Nemeskeri (1971). Detailed formulae are presented in Appendix 1.

The formulae are for use with a stationary population. Given a growing population, the number of survivors in each age group is proportional to the number of deaths adjusted by r (Asch 1976; Appendix 1). To calculate this it is necessary to convert the age distribution of a burial population into the age distribution of deaths of a cohort born during the same interval. The two are not identical since each cohort in a growing population is successively larger, although the actual shape of age distribution does not change over time (Preston and Coale 1982).

Survivorship and mortality are both calculated in the same manner as for the stationary population. Age structure of the living population, however, has to take account of the fact that older age groups come from smaller initial cohorts in a growing population than a stationary case where all cohorts are of the same initial size. Life expectancy is no longer identical to the mean age of death.

Suggested method of proceeding

The suggested method of proceeding through Step 2 is, after assembling the data and establishing representativeness (Step 1), to:

1. Search for bias.
2. Construct a life table based on the assumption of zero population growth.
3. Estimating possible growth rates and construct applicable life tables thereby allowing comparison of parameters.
4. Construct a sex-specific life table and compare mortality.
5. Compare the life table with model groups to establish points of similarity and contrast.
6. Estimate social indicators on the basis of the life table and age at death ratios such as mortality, dependency, fertility, family size, orphanhood, widowhood, and other measures of kinship.

The results will form the basis for the analysis of pathology and the first stage of a reconstruction of social organisation.

Pathological conditions can be identified by careful consideration of the skeletal lesions

Palaeopathology: the biocultural approach

As emphasized in the introduction the two areas of pathology and demography are closely intertwined. Analysis in palaeopathology should be capable of both testing and further refining palaeodemographic models. To explain how this can be achieved, it is necessary first to describe the theoretical and analytic approaches of the field that are used in this study.

In its narrowest description, palaeopathology is the study of disease in past populations. In recent years this definition has been broadened to incorporate skeletal markers of stress which are indicative of a more general physiological disturbance (Huss-Ashmore and Goodman 1982). The aims of the discipline have been seen as:

1. elucidating the history of disease;
2. identifying change in disease patterns through time;
3. reconstructing the environmental conditions of past populations;
4. reconstructing the processes of response to changes in these conditions (Ortner 1991).

The majority of studies follow a biocultural approach in which environment and culture are seen as interacting with human biology. The results of this interaction in turn affect environment and culture. Where in palaeodemography there has been constant debate over the role of demography, the ability to interpret past conditions from limited data and the applicability of methods, there has not until recently been a parallel movement in palaeopathology (Wood et al. 1992). Rather, the emphasis in palaeopathological studies has been upon increasingly complex methods without any corresponding debate on the initial formulation of models or the interpretative frameworks.

The results of this narrow emphasis can be seen in any survey of palaeopathology literature. Firstly there are a core group of studies which form the theoretical basis of palaeopathology, and secondly, there are a plethora of studies which follow, with increasingly sophisticated methods, the same basic idea. In its simplest form this comes down to a comparison of percentages of skeletal conditions between groups, and interpretation of changes in percentages in terms of changing environmental conditions.

While such studies are important, and indeed essential, they go only so far, especially in looking at the processes by which human biology and environment interact. Thus their ability to generate hypotheses that can be tested by other forms of evidence is limited, while few studies incorporate any way of verifying the results. In part this has led to a lack of unifying link between such studies on prehistoric and on modern populations. A large amount of evidence is presented solely in the form of percentages without interpretation of what this means in terms of living populations. Goodman's claim that palaeopathologists have quit too early is certainly supported by any survey of the current literature (Goodman et al. 1988).

The problem is that a collection of skeletons is not equivalent to a group of living individuals. Rather these are the people that failed to survive (Wood et al. 1992). This factor, added to the uncomfortable realisation that diseases have evolved over time in both manifestation and impact, is probably behind the tendency of many studies not to go beyond the skeletons themselves in terms of interpretation. Nevertheless there have been attempts to circumvent the paradox of skeletal collections. Cook, for instance, used measures of health status (namely mortality ratios) directly comparable with modern populations (Cook 1981).

Differential diagnosis

At the same time, Buikstra has emphasised the importance of differential diagnosis in interpreting pathological lesions (Buikstra 1977, Buikstra and Cook 1978). This method operates on the underlying assumption that diseases in past populations were similar to modern diseases. Therefore, if it can be proved that the pattern of lesions is similar, they represent a related disease. Diagnosis necessarily incorporates a level of uncertainty which can only be partially overcome by the development of appropriate models based on recent experience (before effective treatment), careful observation of all possible skeletal elements, and detailed comparison between the two.

Problems include a lack of comparability between clinical samples (based on living people) and skeletal material (Ortner 1991) and uncertainty regarding the evaluation of disease. The ongoing debate regarding tuberculosis is an example of these difficulties. Does the discovery of tubercular-like lesions amongst precontact Amerindian skeleton indicate that the disease had a lower population threshold in the past, or that a similar unknown disease affected these individuals? (Ortner 1991) Obviously, in palaeopathology no diagnosis can be definite (Waldron 1994).

Biocultural models

Diagnoses, albeit tentative, can be incorporated in a model of environment/culture interaction such as that described by Goodman and co-workers (Goodman et al. 1988). Their model views the environment and culture as causing stressors which are mediated through the buffers of culture and host resistance. This physiological disruption is then manifested through various skeletal indicators such as disturbed growth, disease, and mortality. These individual physiological responses have broader consequences, not just for the individual, but also for the society. Stress is seen as "the biobehavioural response to environmental and cultural conditions" (Goodman et al. 1988:171).

This approach is useful in the ability to place skeletal markers in a frame which allows their analysis in terms of environmental or cultural factors. While the causes of stress may be defined as both environmental and psychosocial, in prehistory these factors are immeasurable and indefinable with the exception of external environmental effects which have potential archaeological indications. Similarly the initial impact of a stressor is immeasurable and imperceptible since it is the actual physiological mechanism visible solely in process. This leaves us with the physiological response to stress and the individual and social consequences of that response which tend to be difficult to distinguish. For example, the cessation of growth leading to enamel hypoplasia is a response to stress yet the presence of an

actual defect indicates recovery which is surely a later sequel to stress.

The difficulty in measuring cause and impact, and in distinguishing between response and consequence, is not the only problem with this model. The model assumes that the individual organism is in homoeostasis, and that on a populational basis the same thing occurs. This assumption is not necessarily justified, particularly at the level of society: while certain segments of society may be in a favourable balance, they may maintain this state at the expense of other groups.

Nevertheless, as a general framework, the model forms a useful way of incorporating the realisation that response to environmental and cultural conditions is not necessarily adaptive for the individual or for the society. Also the dual input of cultural as well as environmental conditions is essential in any model of biocultural interactions.

The major problem with the model is that the interpretative links are not defined or explained. There is no interpretative frame to allow us to extrapolate from the dim skeletal reflections of stress to the socio-economic conditions of the living society. We are limited to hypothesizing cause from skeletal consequence, which presumably is why so many palaeopathology studies concentrate on change. As the authors of this model point out:

> In order to become amenable to anthropological interpretation we will need to know how impairment or improvement influences productive behaviour at the individual, household, and population level... Unfortunately, many biological anthropologists stop short of providing data on these more socio-economically oriented questions. By doing so, we leave our data in a form which is inaccessible to most social anthropologists [and archaeologists] and deny ourselves a glimpse of the real consequences of biological dysfunction. (Goodman et al. 1988:195).

The lack of middle range theory, allowing the reconstruction of a living society from the skeletal remains, and the comparison of results with ethnographic and historic data, has been a major drawback of palaeopathology studies. What is needed in this instance is a very clear definition of what each skeletal indicator implies and a method of interpretation which allows as accurate as possible identification of disease processes, and, then, for an assessment of their socio-economic consequences. In the current model of analysis these are seen as step 3, differential diagnosis, and step 4, cause of death analysis.

The practicalities

While it is easy to espouse a middle-range theory, in practical terms the process is bound by limitations of the data and the need to assess carefully what each skeletal marker actually indicates. This includes the problems of variation in individual response and genetic difference (Wood et al. 1992), as well as statistical processes based on sample size (Waldron 1994). It is proposed that the analysis of pathology should proceed in several steps, with testing of the results at each level.

Firstly pathological lesions and the markers of stress need to be quantified independently. As advised by Cook (1979), recording pathology should not pre-empt diagnosis. Lesions are quantified by type of bony response, extent of the involvement, and nature of the lesions, active or healing. (More precise definition of these states is given in Chapter 5).

These lesions should then be quantified in simple statistical terms to test initially whether they demonstrate age or sex-specific variation. In addition this makes it possible to assess whether or not sample sizes are sufficient to justify any statistical manipulation at all.

At the same time, it should be possible to examine the association between the various pathological processes. It may be possible to discern the synergistic relation of variables and to attempt differential diagnosis of lesions.

Methods of recording pathology

As emphasised by Buikstra, methods of recording palaeopathology must describe not diagnose prematurely (Buikstra 1977). In addition recording needs to incorporate an unambiguous terminology, precise data on location, and a descriptive summary of the bone's morphology in order to enable the most precise diagnoses to be made (Ortner and Putschar 1981). In the current study a scheme similar to that used by Powell is employed (Powell 1988). Powell recorded for each bone three items: the nature of the lesions; the lesion's specific location; and the status of the lesion (whether active or healed). With the exception of osteoarthritis (discussed below), all pathological signs upon the bone may be systematically recorded in one of the following four categories:

1. Resorptive (porotic hyperostosis, osteoporosis).
2. Proliferative (infectious).
3. Traumatic.
4. Neoplasms (Powell 1988).

Naturally these categories are, to some extent, arbitary since they are not necessarily mutually exclusive. Nevertheless, given further subdivisions within each class of lesion, relatively precise definitions may result.

The combined results of demography and pathology can identify underlying disease processes and the age structure explained by examining the effect of pathology

There are two methods of anlaysis which are most useful for assessing the impact of disease. These are cause of death and

survivorship analyses. Cause of death analysis in skeletal groups is based upon a modification of the normal demographic method (Preston 1977; Palkovich 1978). The aim is to determine the extent to which variation in the age structure of death can be related to the impact of disease. Naturally in skeletal groups we have, except in unusual cases, no idea of the proximate cause of death. Assuming that skeletal markers are indicative of underlying diseases, Palkovich managed to demonstrate that it is possible to use this method in order to assess the role of various diseases on mortality (Palkovich 1978). Basically the method uses multivariate analysis to partition mortality at each age cohort according to the observed pathology. This enables the assessment of the pattern of disease and the relative impact of disease processes upon the pattern of mortality. Further analysis of data on disease impact combined with the techniques of differential diagnosis (as described earlier) allows the identification and isolation of different disease patterns operating in a group. Palkovich has been able to use this method of analysis to argue convincingly for the importance of nutritional deficiency as opposed to the impact of infectious disease in a Mexican Pueblo group, Arroyo Hondo (Palkovich 1987).

An analogous method which also tries to bridge the gap between skeletal markers and actual pathological processes is the study of survivorship. Using skeletal markers of stress such as enamel hypoplasia, Goodman and Armelagos (1989) have demonstrated that individuals who experience periodic episodes of stress and recovery during formative years suffered from decreased survivorship. Two hypotheses have been used to explain this (see Goodman and Armelagos 1989, Ortner 1991). One is that these episodes adversely affected the individual's immunological competence making them more susceptible to later physiological disruption. The other is that these individuals were always the weaker members of society and experienced differential access to care and nutrition, thus their life expectancy was less. It is obvious that the inability to discriminate between these two hypotheses is a major problem. In the first instance we have a situation of stress applied to all members of the society, the severity of which contributes to later dysfunction. In the second, the impact of the stress is felt only by a subgroup of the society and has limited impact on the health status of the population as a whole. Nevertheless the method demonstrates whether the conditions that the skeletal markers reflect are having an impact on mortality.

There is a paradox involved, as Cook points out:

> It is hardly a novel proposition that people who die are not healthy, but the consequences of this fact for the interpretation of stress indicators in prehistoric skeletons have not generally been appreciated... It is naive to assume that the frequency of developmental disturbances in a mortality sample directly reflects the experience of the living population...there may be thresholds in the level of stress... (Cook 1981: 134–40).

Ortner (1991) also is concerned with the possibility of misinterpretation; that the presence of high frequencies of periostitis in a skeletal sample is not necessarily indicative of poor health conditions but rather that an individual has survived long enough to develop skeletal lesions.

It is this complex relationship between a skeletal population, the living population and the pathological conditions faced by that society in that area which is inadequately addressed by many of the interpretative methods used in palaeopathology. These problems have most recently been summarised by Wood et al. (1992). They identify two major issues, apart from demographic nonstationarity: selective mortality (the sample consists of those who died) and hidden heterogeneity in the risks of mortality or morbidity.

The first issue is primarily one of interpretation. They suggest that the observed frequency of pathological conditions should overestimate the true prevalence of the lesions in the population. Yet the relationship is by no means this simple since it is determined by whether a pathological condition contributes to death or not (Waldron 1994). This difficulty in interpretation is to some extent offset by the use of multiple indicators such as stress markers (Goodman 1993). It also requires a closer examination of the relationship between morbidity and mortality, the status of the lesion, if possible the severity (Lukacs 1994) and the context.

The second issue they raise, hidden heterogeneity, means that there is inbuilt ambigiuity in any results. Any population consists of a series of subgroups varying in susceptibility and predisposition to disease due to both genetic and environmental conditions. This variation results in a population with individual levels of frailty. The sources of variation in susceptibility are difficult to infer (hence hidden) and over time as the more frail are removed from the population subgroups may diverge in their risks of death. This means that a decreasing prevalence of stress indicators for instance could indicate improving conditions, a lack of exposure of certain subgroups or gradual selective effects on a more frail population. Lower mortality may result in increased morbidity as the more frail survive. In cross-sectional studies of health, mortality, etc. heterogeneity has little effect. In models over time, however, it may create "systematic changes in the composition over time, change which may create the illusions of effects that are not really present (Lam and Smouse 1990:99)."

Wood et al. (1992) have suggested that mathematic methods modelling a distribution of heterogeneity may help to highlight situations where heterogeneity exists. However, the authors of one such method point our that this is problemmatic since there is no way to distinguish best fit between models incorporating hidden heterogeneity and one model without it (Trussel and Rodriguez 1990).

As Lam and Smouse (1990) point out, work on hidden heterogeneity has increased awareness of the potentials for

misrepresentation and the need to gather as much contextual data as possible.

Taking account of the nature of the lesion and what it represents, the impact of various diseases upon mortality can be discussed through the use of ANOVA and other forms of multivariate analysis (Palkovich 1978). This enables examination of the possibility of hidden heterogeneity and its incorporation into interpretation of results.

In Step 3 differential diagnosis allows for the identification of disease processes. By assessing the pattern of skeletal involvement in the population and its impact on mortality it should be possible to identify any specific pathological conditions as well as more general problems such as the relative impact of nutritional deficiency and infection, and begin to address issues such as the interaction of disease and social organisation.

The results of these analyses can be assessed in terms of the experience of each age class and the validity of ethnographic comparisons and archaeological inferences assessed

It is suggested here that the use of the two methods of analysis described above (cause of death and survivorship) are the first step in assessing the impact of disease, and in permitting the analysis of the living as opposed to the skeletal population. Yet survivorship analysis can be taken one step further. As in demography, the analysis of pathology by survival analysis makes the explicit assumption that all individuals in a skeletal sample experienced similar living conditions and that the concept of a synthetic cohort is a justifiable simplification. It is interesting that palaeopathology studies make use of this notion by the very lumping of skeletons into age cohorts. Our ability to separate skeletons diachronically may improve but at some level this notion will have to be implied; it is unlikely except in specific cases such as ossuaries or completely dated individuals, that changes by generation will ever be discernible.

Furthermore, the analysis of survivors encompasses the idea that a segment of society experiences sufficient physiological disruption at various times to cause a skeletal response. At the same time the rest of the population either does not experience this insult, or else experiences it without the same degree of physiological disruption possibly due to inherently lower frailty (Wood et al. 1992). In part this ignores the fact, accounted for in Palkovich's analysis (Palkovich 1978), that a further segment of the population experiences this insult and dies. For example, if we take enamel hypoplasia as a marker of stress:

at age 1:

- 30% of those born die
- of the 70% who survive: 40% develop enamel hypoplasia while 30% fail to show any signs of stress

at age 2 :

- of the 30% without hypoplasia a further 10% have died, 10% have developed hypoplasia, 10% are unaffected
- of the 40% with hypoplasia: 20% have further hypoplasia, 15% have died, 5% are not affected by further hypoplasic episodes.

This is the same view as used in survival analysis, but merely incorporates mortality into the analysis. Enamel hypoplasia is a specific case since it enables the ageing of episodes of stress and recovery and can be analysed in detail, but other indicators of pathology can be analysed in a similar fashion once account is taken of whether the lesion is active or healing, the common age of onset, and the relationship of lesions to mortality which is given by cause-of-death analysis. In part this form of subdivision addresses both the issues of selective mortality by looking at survivors as well as non-survivors, and potential heterogeneity by subdivding the population in regard to their morbidity experiences. It does, however, require a stable population so that all such analyses have to assume stability and assess the potential impact of fluctuations.

The aim is that, given differential diagnosis of disease, and using methods of percentage comparisons, examination of lesion nature and extent, and cause of death analysis it will be possible to examine the impact of this on each age class using the demographic technique of survivorship. It is then possible to view the population in a synchronic fashion, describing the age experience of each class, building up a reconstruction of living conditions which, unless there is an inconsistency in the demographic results, should correspond with the social composition implied by a straightforward palaeodemographic analysis.

In this fashion we come one step closer to a picture of the living society and its interaction with disease given the provisos on interpretation mentioned earlier. While this may bear little relationship to the experience of any single individual in the population it is a model of the collective experience of a cohort.

The first four steps of the analysis allow for the identification of disease patterns in the society (to the extent that skeletal indicators mirror pathology). This enables the comparison of results with the known archaeological background and with comparable historic and ethnographic data. The end result is a model of the living conditions of a past society described in terms which make it possible to further compare results from other sources, including further archaeology.

Conclusion

What is presented here is an analytical model designed to bridge the interpretative gap between skeletal remains and reconstruction of social structure. Basically, the model consists of melding together the results of palaeodemographic and palaeopathologic analysis more closely. The five steps are:

1. Skeletal recording.
2. Life table analysis.
3. Differential diagnosis.
4. Cause of death analysis.
5. Analysis of Survivorship (both pathologic and demographic).

The easiest way to demonstrate the usefulness of this approach is by means of a case study. The following chapters describe the application of this analytical model to two skeletal samples from Bahrain Island dated to c300 BC–AD 250.

Chapter 3

Bahrain Island

The skeletal population selected for analysis is from the state of Bahrain in the Arabian Gulf, and dates to c2300–1800 BP (300 BC–AD 250). This area was particularly suited to analysis because of the nature of the island settlements and cemeteries, and of the samples themselves.

Prior to any skeletal research, it is necessary to understand the physical and social context of the sample and its environment. Analysis can then provide additional data and hypotheses concerning the adaptation of past inhabitants. As one of the few studies concerning environment (Larsen 1983) clearly demonstrates, the geographical constraints of Bahrain have imposed limits on the extent of settlement and its nature.

Environment

Geography

The state of Bahrain is located approximately 20 kilometres off the east coast of Saudi Arabia (Plate 1). The nation consists of a group of 32 small islands. The largest, Bahrain Island (or Awal), is approximately 50 kilometres long and 20 kilometres wide and, as the largest of the islands (612 km^2), is the main centre of occupation.

This group of islands has been an important staging point for trade since the third millenium BC. Their location in the gulf and the supplies of fresh water upon the Island have meant that, for ships plying trade between India, Pakistan, Oman and Iraq, Bahrain was often a natural stopping point.

Bahrain Island has been through time the main focus of human activity so the following study will concentrate upon this island. The outer edge of the island is a narrow coastal plain, widest on the northern edge (c9 kms wide) and extending down the eastern and western sides (between 3–4km in width). These plains rise to barren limestone slopes forming a rimrock around a central depression. In the centre of this lies the island's sole 'mountain', Jebel Dukhan (c150 m a.s.l.).

This particular geomorphology means that cultivation has been restricted to the area of coastal plain. Elsewhere limestone bed-rock lies extremely close to the surface (less than 0.5 m), jutting up even in the coastal plains. Soil on these plains tends to be sand covered with a clayey loam which can be used for agricultural purposes, although the depth of the soil is variable and there are persistent problems with salinity and aeolian sand drifts (Vine 1986; Paskoff and Sanlaville 1984). Potentially arable soil, formed from weathered limestone, also occurs in the areas of wadi outfall on the lower edges of the limestone slopes where small, poorly developed wadis drain towards the coastal plain, and in limited areas of the central depression (Larsen 1983). Otherwise, the area between the coastal plain and rimrock is gypseous gravel unsuitable for agriculture.

Climate

The restricted extent of arable soil is not the main constraint on settlement. Climatically the island lies within the Saharo-Arabian zone (Rumney 1968). The average annual temperature is 26.4 degrees Celsius. At all times humidity is high, the average being 85%. The year may be divided into four periods. November till February is the coolest period with temperatures dropping to 17–19 degrees.

Rainfall is sparse, never more than seven cms per year, and falls in two well-defined periods: late autumn, around November, and in early spring, February to March. Apart from these clear short periods, about three to six days a year (Lorrimer 1908), no other rain falls. The Island is totally dependent upon ground water supplies for cultivation.

Water supplies

The reliance upon groundwater, more than the extent of arable soils, determines the extent of settlement and cultivation. The island is fed by fresh water springs rising along the northern and western coasts. Their source is an underground aquifer in the Saudi Arabian mainland which drains to the southeast (Larsen 1983).

In the days before mechanical drilling for water, the springs determined the amount of cultivatable land. In turn spring outflows are affected by a combination of sea level changes (higher sea water levels equals higher spring levels), recharge of the aquifer system in Saudi Arabia and climatic change. In recent years it has also been affected by the increased usage of water enabled by drilling (Beaumont 1977, Larsen 1983).

The pattern of settlement on the Island can be plotted against the potentiometric level of artesian springs (Larsen 1983). An irrigation system based on gravity flow from natural springs means that only areas below the spring may be watered. More complex irrigation such as the qanat systems involve digging down through the upper slopes to tap underground water. Tunnels or deep covered channels are then constructed to direct this water flow to larger areas of land. The canals are tapped by means of lifting devices. The qanat system therefore allows greater areas of land to be irrigated (Ebert 1965, Larsen 1983).

Agriculture

Traditionally cultivation on the Island has been based on date palm gardens. The date palm is extremely important, not just

as a source of fresh dates. Green dates were fed to cattle as fodder, the wood was used for timber in building, the leaves were used for matting and for building reed huts or barastis (Durand 1880). The dates themselves were eaten fresh, as well as dried or pressed to make honey (Rougelle 1982). High rates of caries in all skeletal samples from the Island demonstrate the importance of this was as a source of food (Littleton and Frohlich 1989).

The date palm is a traditional oasis crop. Amongst its advantages are an ability to tolerate high levels of salinity and towithstand periods of drought or even flooding. In addition it produces high yields, up to 100 kg per tree (Steven and Cresswell 1972).

In date palm gardens on Bahrain other fruit and vegetables are traditionally intercropped with dates. The palms provide useful shade to plants underneath, as well as maintaining a moist environment (Daggy 1959). Lorrimer, in 1908, listed agricultural products as 'chiefly fruits, lucerne and a few vegetables; the last including brinjals, cucumbers, carrots, leeks and onions' (Lorrimer 1908 II:241). The fruits included citrons, bananas, almonds, apricots, figs, grapes, limes, melons, peaches, pomegranates and some tamarinds. At the time of Lorrimer's report, plant growth and production were described as rank and mediocre. This may reflect lowered water levels or increasing problems with salinity. Yet at the same time Lorrimer observed that agricultural production was sufficient for local consumption (Lorrimer 1908).

By the early 1900's, very few sheep and goats were found on the Island. Lorimer estimated that there were about 500 sheep and 700 goats, mostly owned by the ruling family (Lorimer 1908). There was only one small herd of local cattle used for milk and some beef production. Slaughter animals were routinely imported (Lorimer 1908). Evidence of animal bones from Bronze Age cemeteries and settlements, however, indicates that this was not the case in earlier periods (Frohlich, M 1986; Nesbitt 1992).

The other source of protein was always fishing. Coastal populations on the Island are traditionally involved in fishing, largely by netting and in tidal weirs (Vine 1986). Even if not directly fishing themselves, trade probably ensured a supply of at least dried fish to most villages. Excavation of a Bronze Age village near Saar (the same area as one of the skeletal samples) revealed large deposits of fish bones and shell, indicative that non-coastal villages also benefited from fishing in the past (Nesbitt 1992).

Water and land-use

The ability of the Island's population to produce sufficient food for local consumption is obviously heavily reliant upon the supplies of spring water. Based on research by two companies into artesian water supplies a major thesis of land use on Bahrain has been constructed (Larsen 1983). This thesis suggests that throughout time settlement on the Island has been constrained by a shrinking water supply owing to depletion of the aquifers. Larsen has tied this to a survey of archaeological sites, suggesting that periods when settlement extended furthermost from the north coast correspond to times when water supplies were greatest. He hypothesizes that, during periods of high sea levels, after recharge of the aquifer system in Saudi Arabia, and during periods of moister climatic conditions, settlement would have extended over a greater area of the Island.

Surveyed settlement during the Bronze Age extended to an elevation of 12 metres a.s.l. It is suggested that this corresponds not only to naturally higher spring levels, but also rainfall patterns. The Bronze Age period is therefore interpreted as highly productive agriculturally (Larsen 1983). The nature of this agriculture is unknown. While Larsen suggests a similar pattern of cultivation to the Middle Islamic period, presumably post-qanat construction (Larsen 1983), skeletal evidence suggests that diets during these two periods were markedly different (Littleton and Frohlich 1989).

Following this high point, it has been suggested that there was a contraction of settlement towards the north of the Island. This, Larsen hypothesizes, is due to a decline in rainfall, a drop in sea level, and natural drying (Larsen 1983). It is suggested that this contraction lasted until c750 BC when settlement again apparently expanded due to a combination of environmental (increased winter rainfall) and economic (increased trade) factors. While settlement according to Larsen's survey, does not reach the extent of Bronze Age expansion, it does mark a period of relative agricultural prosperity (Larsen 1983).

Several problems exist with this thesis. Firstly it is based on the assumption that all sites of a given period would have been located at the same highest elevation possible (Potts 1985). Such an assumption fails to account for other factors affecting settlement siting, including population size. In addition, Larsen's corpus of sites, particularly those belonging to the period of the skeletal samples used here, is now incomplete due to discovery and excavation of several Tylos period cemeteries which serve to extend the limits of land-use during this period.

A further problem is that very little is known of the methods of irrigation used on the Island over time, and Larsen himself fails to account for changes in irrigation (Larsen 1983). Yet, as emphasised earlier, there are substantial differences in the extent of arable land dependent upon gravity flow as opposed to qanat irrigation. There is no direct physical evidence of what sort of irrigation was practised during the Tylos period. Two indirect pieces of evidence do exist.

Firstly sites from the Eastern Arabian mainland belonging to this period include stonebuilt wells indicating that deliberate tapping of aquifer water was known at this time (Bowen 1950). Given the close cultural ties between Bahrain and the mainland, it is reasonable to assume a similar technology on the Island.

Secondly, it is suspected that more extensive agriculture was introduced during the Iron Age with the introduction of cotton, which requires greater supplies of water than could be provided by gravity flow alone (Nesbitt 1992). This hypothesis is supported by the discovery of cotton cloth in an Iron Age context (c750–500 BC) on the Island (Frohlich pers. com.).

Whether this system was a fully fledged qanat system with its attendant complex of covered drains and tunnels, or a series of wells tapping the aquifer rather than relying solely on springs, is unknown. Irrigation completely reliant upon gravity flow alone is restricted to areas below springs and is constrained by the rate of discharge and the amount of evaporation. Wells tapping the aquifers extend the area that may be irrigated although it is still mainly areas below the wells. Yet the problem of evaporation and water loss through transport still needs to be managed. In contrast, the qanat system extends the area of potential irrigation by systematic tapping of the aquifer, possibly at higher elevations, and by decreasing the degree of evaporation. These caveats, applied to Larsen's thesis, suggest that during the Tylos period settlement on the Island was extensive and possibly prosperous especially if based upon a more economical irrigation system than gravity flow and open channels.

The skeletal samples analysed, one from a cemetery in the north of the Island, and one from a west coast cemetery, belong to this period (300 BC–AD 250) characterised by Larsen as relatively agriculturally prosperous. It is therefore important to determine from other sources of evidence than the purely environmental, i.e. historical and archaeological sources, what is known about settlement and agricultural practices at this time. Larsen's work provides a useful starting point and a framework for comparison.

Historical position

History

Before discussing in detail the historical evidence for Bahrain during the period 300 BC–AD 100, it is useful to examine the chronological scheme used by researchers on the Island (Table 3.1).

Early Dilmun is represented by levels 1–11 at QAB, Barbar Temple, and most of the burial mounds (Bibby 1970, Ibrahim 1982, Mughal 1983). During this period, the Island was actively involved in trade with Mesopotamia acting as an entrepot for trade between India, Oman and the Kingdoms of Mesopotamia. Towards the latter part of the second millenium this trade apparently declined in importance.

Less is known about the Late Dilmun period, although buildings on Qa'lat al-Bahrain (including a possible 'palace') and burials attest to continued occupation of the Island. There is some evidence of renewed contacts with the kingdoms to the north of the Gulf although it is probable that Bahrain operated as an independent kingdom (Lombard 1989).

Table 3.1
Chronological table for Bahrain

Name*	Dates	Alternative names**
Early Dilmun	?2300–1700BC	Middle Bronze Age
Middle Dilmun	1700–c1000BC	Late Bronze Age
Late Dilmun	1000–300BC	Iron Age
Tylos		
-Va	300–100BC	Seleucid
-Vbi	100BC–AD100	Early-Middle Parthian
-Vb2	AD100–250	Late Parthian
-Vc	AD250–630	Sassanian
Islamic	AD630–	Islamic

Based on *Lombard and Kervran 1989; ** Larsen 1983 and Salles 1987

The term 'Hellenistic' is loosely applied in the Gulf to the period 325 BC–AD 676 (the coming of Islam). The term is merely a convenience and, while it reflects the Gulf's cultural contact with Hellenistic settlements to the north, does not imply the presence of Greek colonists. In archaeological work this period has been further divided tentatively into Tylos Va, b and c (300 BC–100 BC, 100 BC–AD 250, and AD 250–676 respectively, Salles 1987).

Historical evidence on the Tylos period tends to be limited and subject to some dispute. It is suspected that at the time of Alexander's expeditions, eastern Saudi Arabia was a confederation of small states involved in trading. Gerrha, a city located somewhere in the region between Hofuf and Thaj, was the primary centre of trade, controlling movement of incense and other products between South Arabia, Mesopotamia and India (Salles 1987). A large number of coastal sites belong to this period and it has been proposed that Bahrain also lay on a trade route to India (Salles 1980).

The role of Bahrain during this period is obscure. Prior to Alexander's conquest of the Achaemenid empire, Bahrain appears to have been actively involved in trade. It is probable that this situation continued and that economic and trading concerns were the primary interests directing the policy of Alexander and his successors, the Seleucids, in the Gulf. This policy seems to have involved active commerce with the Gulf but not deliberate colonisation (Potts 1990; Salles 1987).

The Gerrhans maintained control of trade in the Gulf until 205 BC when Antiochus III, one of the Seleucid dynasty controlling Mesopotamia, mounted an expedition to Gerrha. His motives are subject to debate (Salles 1987) but the consequence of his visit appears to have been a tenuous partnership between the Gerrhans and Seleucids. This controlled trade until the disintegration of the Seleucid

empire after the death of Antiochus IV in 164 BC. It is suspected that part of Seleucid policy was maintenance of a naval presence within the Gulf (Potts 1990). Certainly such a fleet must have been in existence by the time of Antiochus III's visit to Gerrha since leaving Gerrha, he sailed to the island of Tylos (the Greek name for Bahrain) and left from there for Seleucia (Polybius 13:9:4–5). Maintenance of a fleet would probably have required the establishment and maintenance of entrepots for shelter and resupplying. Salles suggests that several such bases were maintained, including a military presence (Salles 1987). While it is possible that such bases existed, their location is unknown, and the interpretation of a Seleucid enclave on Bahrain during this period is tenuous.

After the death of Antiochus IV in 164 BC, the decline of centralist authority led to the resurgence of separatist states (Colledge 1967). In c130 BC the Parthian king Phraates finally defeated and killed the last of the Seleucids, Antiochus VII Sidetes. Thus the Parthians gained control of an empire extending from Babylonia to eastern Bactria with the Tigris and Euphrates rivers forming the western border (Colledge 1967). Attempts were again probably made to control Gulf trade.

In the first centuries BC and AD, Characene, a Parthian sub-kingdom at the head of the gulf (with Spasinou Charax as its capital), began to dominate trade (Nodelman 1960). It is suspected that by the end of the first century BC the Parthians, through Characene, had consolidated control over trade, clearly influencing the western Gulf as far as Qatar (Salles 1988).

Trade declined owing to increasing use of the Red Sea monsoonal route. The amount of caravan trade through Palmyra may also have lead to a gradual decline in trade handled through the Gulf in general, and Bahrain in particular. Parthian trade in any case appears to show a steady decline after the first century AD, ending at the end of the second century with the withdrawal of Characene influence in the Gulf (Potts 1990).

The extent to which these political changes influenced Bahrain is unknown. Salles has suggested that the period between 250–100 BC was reasonably prosperous for the Island (Salles 1987). Based on Aramaic inscriptions and the presence of Mesopotamian style pottery and other Mesopotamian products, he suggests that close relations existed with Saudi Arabia (Salles 1987). Evidence indicates that Bahrain was an important commercial market but more in the movement of foodstuffs than in luxury long-distance trade (Bouccharlat 1986; Salles 1988).

Later Parthian influence is clear in finds from the Island : 'Roman' glass, Parthian-style terracottas (Salles 1984). It is questionable whether this influence included the presence of a governor from Characene on the Island (Potts 1990, Salles 1987).

The effect of these political changes on Bahrain's trading and agricultural activities, particularly the possible presence of foreigners on the Island during the Seleucid and Parthian periods, is unknown. Colledge points out that in the context of Parthian trade 'economic interests usually outweigh political' (Colledge 1967:80) and that the effect of disturbances was probably short-lived. This could be particularly true for Bahrain, apparently mainly involved in local agricultural trade, with the exception of pearls which were an obvious luxury item.

Salles and Potts, who each attest to a foreign presence on the Island (one the Seleucids: Salles 1987; one, the Palmyrenes as Parthian representatives: Potts 1990) also emphasise the lack of effect such a presence would have had. It is assumed that, similar to Achaemenid colonies (Briant 1982) and Portuguese establishments (Kervran 1988), the foreign enclave would remain isolated from the local population dealing primarily with the local aristocracy.

Historical sources are as vague about Bahrain's economy as its politics during this period. Aristobolus described the Island, called 'Tylos' in this period, as a 'centre of intensive gardening' (Arrian 2:273–5). Theophrastus and Pliny, also writing about the Island, both mention cotton cultivation (Theophrastus Hist.Plant. 4.7.7–8; Pliny N.H. 12.21.38f). In addition pearl-fishing off the coast of Bahrain was described during this period (Athenaeus Deipn. 3:146).

The archaeology

Archaeological evidence is equally sparse. The most extensive settlement excavation has been at Qal'at al-Bahrain (Bibby 1970; Plate 3.2). Evidence from pottery indicates continuous settlement from the seventh century BC down through the Tylos period (Bibby 1970, Larsen 1983). Yet for the mid-first millenium there is no indication of the nature or size of settlement (Boucharlat 1986). Very few architectural remains are connected with the pottery from Qa'lat al-Bahrain: a floor, pit, some graves and possibly defensive walls. For the 'Parthian' period (100 BC–AD 250) soundings below the 'Islamic' fortress demonstrate habitation throughout this time (Bouccharlat 1986).

The most substantial building belonging to the Tylos period is the fortress, previously assumed to be an establishment of Islamic date. Excavation during 1982–3, however, demonstrated that this was an older construction. The exact date is subject to dispute. Salles (1987) suggests a Seleucid building while Potts (1990:II), similarly seeing the fort as the site of a foreign enclave, places its establishment within the Parthian period.

There are two possible surveyed settlement sites from this period: near Barbar village (Larsen 1983:site 203) and a settlement, possibly Tylos period, near al-Malikiyah (Anonymous n.d.). In addition settlement most probably

existed on Muharraq Island which is mentioned by ancient authors (Pliny N.H. 6.6.147).

In contrast to the scanty evidence of settlements, cemeteries are abundant and much more extensive than suspected by Larsen (1983). The extent of these cemeteries can be seen on Plate 2. These cover both the Seleucid and Parthian periods with no obvious chronological gaps. While cemeteries will be discussed in more detail in the next chapter, it should be noted here that several authors have suggested that cemeteries are, for this period, a better marker of the extent of habitation on the Island than the surveyed settlements (Larsen 1983; Salles 1984; Littleton 1990). The current known extent of Tylos period cemeteries indicates settlement reached 17 kms from the north coast.

This of course gives no indication of the density of occupation. As seen in Plate 2, cemeteries away from the north coast tend to be concentrated in a pattern following the distribution of Bronze Age burial mounds. Given the close tie between settlement and water this suggests that Tylos period land use may have been similar in extent to the Bronze Age period excluding the southernmost sites of the this earlier time. Certainly in the Tylos period there is also more evidence for habitation in the northeastern corner of the Island.

In terms of material finds the archaeological evidence can be seen to imply participation in a general Hellenistic cultural heritage (see also Herling 1994) but not of a profound Greek influence. The same can be said of the Parthian period where there is a uniformity of cultural material throughout the Gulf and into southern Mesopotamia. At the same time local Arab traditions persist, sharing common trends with the Arabian mainland.

Tylos period land-use

This gives a starting point for the reconstruction of settlement conditions during the Tylos period, to which skeletal analysis can then be compared. Climatically conditions may have been moister than currently. A slight increase in winter rainfall would mean less pressure to irrigate all year round, easing some of the demand for spring water. At the same time if we presume that the notion of steadily declining spring levels over time is correct then for the Tylos period only areas less than 6 metres above sea level would be utilised (Larsen 1983). This appears to be an underestimate. Settlement as indicated by cemeteries extends to circa 9 metres a.s.l. suggesting higher spring levels than estimated by Larsen. There is, however, no indication of how many springs were flowing at this time, nor of their rate of flow.

Larsen points out that there are two main areas of arable soil: the colluvial fans at wadi mouths, between 6–12 metres in elevation, and the coastal plains, less than 6 metres in elevation. He suggests that at all times the coastal plains could be irrigated by gravity flow from springs, while colluvial fans could only be irrigated by qanats or in times of higher spring levels (Larsen 1983). The evidence of Tylos period land use suggests that some areas of wadi outfall could have been utilised during this period.

The assumption that all cultivatable land would automatically be used cannot be proven for Tylos. Cultural and environmental factors in addition to the simple availability of arable soil and water play a role in determining land use; abandonment of gardens during the earlier twentieth century on Bahrain has been attributed to the land tenure system (Khuri 1980). Equally, in eastern Saudi Arabia examples abound of land abandonment due to the constant problem of malarial fever, as Philby so graphically describes:

> They cannot live in the midst of the palms, where the ague falls upon them that they die. It was ever thus here, but Hamad ibn Muradhdhat planted his colony at the edge of palms in the fever heat of the new religion. He is gone now, God have mercy upon him! and many of those who settled with him. The rest have fled, leaving their houses empty to live in the desert, and they come again only in the season to glean the dates which they tend not at all. They shun the fever as the devil himself. (Philby 1986:95)

With no idea of absolute population sizes during the Tylos period there is no way of estimating what amount of land may have needed to be cultivated. Using the assumption that arable land equals land use, Larsen estimated that the extent of cultivation was 65 sq kms during the Tylos period implying, with a population density of 125 people/sq km, a total population of 8,125 (Larsen 1983). While these figures are a beginning to discussion, this estimation of cultivated land for the Tylos period could well be an underestimate.

Of more importance for the present work are the implications of water and land supply for agricultural production. Historically, the evidence for the Tylos period indicates the importance of fruit and vegetable production, and cotton. There is no indication of cereal cultivation but the overall lack of environmental evidence makes it impossible to determine whether grain was grown or not. In addition sections of the Island community were involved in trade and in pearl-fishing. While the major part of agricultural production may have been subsistence level, there was obviously sufficient surplus to allow for trading, not just of cotton but of other agricultural products (Salles 1988).

The extent to which every agricultural village participated in this was probably variable. Larsen points out that in recent historic times the density of settlement was greater near market centres such as Manama (Larsen 1983a). Based on Von Thunen's work, he points out that more intensive production of perishable goods occurs close to markets, while the greater the distance, the less intensive production, and the greater the production of non-perishable goods (Larsen 1983a).

This pattern has also been demonstrated to work on a village level: gardens are closest to the village, with cereals or large

scale crops grown outside the garden belt (Steven & Cresswell 1972; Larsen 1983a). Such a pattern in Bahrain would not be determined merely by labour demands but also by irrigation patterns. If a system of gravity flow irrigation is used then crops requiring greater amounts of water need to be grown closer to the spring source while crops with lower water requirements can be cultivated at a farther distance.

The question of irrigation and its nature that becomes all important when trying to reconstruct agricultural land use on Tylos. Without a system of wells and constructed channels, cultivation is heavily dependent upon springs of sufficient flow and limited not only to downward flow but also by the amount of water wastage and evaporation. If a system such as qanats was in operation during this time then the possibility for cultivating large areas of land including colluvial fans is greatly increased. The physical evidence for this is non-existent so far.

This leaves us with the assumption of scattered villages both along the north coast and down about 17 kms of the west coast during the Tylos period. Settlement density may have been greater in the north and agricultural production was probably less intensive along the west coast.

The pattern of agriculture was probably based primarily on garden fruit and vegetable production with, at least in some areas, effort also being directed to cotton and possibly grain cultivation. The degree of labour required for this is equally intense. Work in gardens involves cultivation, repeated weeding, harvesting and fairly constant attention to irrigation. Grain or cotton cultivation is slightly less demanding, although it too involves periods of intense effort, both in ground preparation, planting and harvesting. The maintenance of irrigation canals is an additional concern which, dependent on the sophistication and extent of the system used, required a degree of cooperation and organisation on at least a village basis (Wilkinson 1974:10).

In Saudi Arabia, for instance, use of traditional irrigation methods (both extraction and distribution) has meant that the amount of water that could be applied to the land was severely limited. This was reflected in status hierarchies: the higher an individual's standing in society, the greater his access to water, and the greater his landholding. The situation is not so closely bound when artesian water is used, but again this depends on the rate and amount of discharge (Steven and Cresswell 1972).

Environmentally this system of agriculture may cause its own problems. Bahrain prior to eradication campaigns was a hyperendemic malarial area. The breeding of mosquitoes is encouraged by the presence of standing water particularly in irrigation systems and wells. In addition traditional housing of reed huts provides little protection from mosquitoes. Evidence from Saudi Arabia before eradication demonstrates continual malarial attack throughout the year (Daggy 1959).

On top of these environmental factors, settlement is constrained by the presence of springs which means there is a lack of opportunity to move away from polluted areas, and a pressure for settlement to be reasonably dense, both inter- and intra-village. This in turn enhances the opportunities for the transmission of malaria and other parasitic diseases.

Traditionally other health problems noted in association with this pattern of settlement are rheumatic disorders, particularly during the cold winter months when reed huts offer little protection from the elements (Lorrimer 1908). Lung disorders have also been blamed on the nature of housing (Hansen 1968).

Discussion

The above data indicate that during the Tylos period, settlement on Bahrain Island was probably centred in a number of larger townships on the north coast, surrounded with smaller agricultural settlements. The density of these settlements was probably less with increasing distance from the north coast. The degree of centralised or state control over the Island is unknown although evidence for cash crops such as cotton, plus non-agricultural activities, for example pearl-fishing, indicate that intra-island trade was well-established. There is no evidence to suggest that the island was not self-sufficient and there is the strong supposition that the inhabitants were involved in local trade of foodstuffs with at least nearby Saudi Arabia (Bouccharlat and Salles 1989, Salles 1988).

Agricultural settlement was heavily dependent upon irrigation systems. Evidence exists for production centred upon intensive gardening practices but quite probably more extensive crops were produced in some areas. The extent of land available for cultivation of these crops must, however, have been strongly constrained by the nature of irrigation. In addition these agricultural practices require constant labour input, plus a degree of village organisation and cooperation to maintain irrigation. One of the major health problems experienced in this environment would be malaria and probably other parasitic diseases.

It is upon this background of small agricultural settlement centred on springs and of local trade that the analysis of skeletal samples can proceed. The issues then become questions of the mortality and morbidity of intensive agriculturalists, and the impact of these two factors on social and economic organisation in a constrained environment.

Chapter 4

The samples and their origins

Introduction

The two skeletal samples analysed belong to the Tylos period, a time of supposed economic prosperity. Yet before they can be examined it needs to be established how representative they are, both of the local populations and of the Island's population as a whole.

The archaeological record cannot be accepted at face value. As emphasised in Chapter 2, one of the biggest difficulties in any skeletal study is dealing with the problems of bias. These originate not only from the methods used, but also simply from the choice of sample. Even the comparison of two populations is untenable unless differences in burial practices are recognised and accounted for.

There is no guarantee that every individual in a local group will be buried within a given cemetery. Noteworthy examples to the contrary are the secondary burial of some individuals (eg. Kenyon 1960), and exclusive burial of males (eg. Haglund 1976). Similarly decay and preservation will not necessarily affect all skeletons alike: the greater fragility of infant bones may lead to their under-representation (Walker et al. 1988). Even different locations within a cemetery may result in some groups of skeletons being preserved while others are totally destroyed (Gordon and Buikstra 1981). Robber activity is extremely important in this respect (Littleton 1995). The final stage of archaeological recovery can also be destructive. While the aim of excavators is to recover the maximum amount of material possible, methods used are a product of time available, personnel available, and excavation aims all of which vary substantially over time.

Given these factors, it is necessary to demonstrate that the two skeletal groups may be described as representative samples of local populations. Furthermore, these burials need to be placed in the context of Tylos cemeteries on the Island and any differences in practice discussed since these may offer important insights into local organisation.

The amount of archaeological data available on each cemetery (DS3 and Saar Mound 5) is variable. In addition the sheer extent of excavation means that there is significantly more information on burial practices at DS3 than at Saar. For this reason the following discussion addresses the sample from DS3 first. Finally, any significant differences in burial practices between the two sites will be analysed, and placed in the more general context of Tylos burials on the Island.

DS3, Hamad Town

Site description

The site is situated on the western edge of the al-Malikiyah Early Dilmun moundfield (Plate 2). This moundfield is one of the clusters of mounds comprising the larger Hamad Town tumulus field. These mounds border the eastern edge of arable land, extending up the sides of the wadis. Two hundred metres to the west is the line of an old qanat system and a settlement site of possible Tylos date (Anon. n.d.).

The Tylos mounds can be distinguished from the Early Dilmun mounds by their large size and sprawling, irregular shape. Aerial photographs of c1985 show that there were three main clusters of Tylos mounds along small wadis totalling approximately 18 mounds. There is a fourth cluster c500 metres to the south but the distance precludes it from being considered as part of the DS3 cemetery.

The site was excavated by the Directorate of Archaeology and Museums under the leadership of Mustafa Ibrahim and Mohammed Hassan, during 1984/5 and 1985/6 as part of extensive rescue excavations conducted during the building of Hamad Town. The Directorate excavated the entire area with the exception of the northernmost cluster of mounds which were bulldozed prior to the excavation. Thus it is estimated that c 80% of the original Tylos cemetery (15 mounds in all) was excavated. This included 292 burial chambers.

Preservation

Archaeological information on the cemetery has not yet been published. The author was present, however, for two months during the 1985/6 season and collected archaeological data on Mound 81, Mound 83, and part of Mound 73. In addition, it was possible to view all the site photographs and confirm that these mounds did not differ significantly from the others on site. The photographs also confirmed the presence or absence of bones within each chamber. Skeletal remains were not recovered from 22.5% of the chambers excavated.

The question of emtpy burial chambers has been one of the thorny issues of Bahrain archaeology (Frohlich, B 1986; Lamberg-Karlovsky 1986; Salles 1990; Littleton 1995) and demonstrates the importance of examining every aspect of a site for possible bias. In the case of DS3 the presence of 'empty' tombs is explicable in terms of preservation. The majority of the graves had silted in, in addition to having been robbed. When the chamber was left exposed after robbing, bone tended to be very badly preserved. Similarly if the grave was cut into bedrock there is a tendency for water to collect, again leading to poor preservation of skeletons.

Added to this, before a skeleton gets to the museum it needs to have survived, not only decay, but also archaeological recovery, itself a destructive process. It was possible at both DS3 and at Saar for a physical anthropologist to examine a selection of the bones in situ (myself at DS3, B. Frohlich at Saar). A comparison of the notes taken in the field with those

from my own laboratory examination demonstrates a loss of between 25–50% of information about each skeleton. This was most noticeably seen in the number of measurements which could be taken in the laboratory as opposed to the field; postcranial length and facial measurements often could not be reconstructed once the bones had been lifted from the grave. Age and sex indicators such as the pubic symphysis were also destroyed during recovery of the skeleton from chambers. The problem could be partly rectified by viewing all the photographs and confirming which tombs were actually empty at the time of excavation.

At DS3 preservation was surprisingly good as a whole. In addition chamber size, particularly length, was strongly correlated to personal age of the occupant (R^2=0.502). This made it possible to estimate the age cohort of the primary interment in tombs where bone was not preserved. Table 4.1 demonstrates the numbers estimated.

Table 4.1
Numbers estimated from empty tombs

DS3	N
Infant	19
Subadult	6
Adult	31
Total	**56**

Tomb construction

Tylos tombs at DS3 were built reusing Early Dilmun burial mounds. The original chambers were frequently destroyed or reused. The edges of these Early Dilmun mounds were then pitted, often down to, and sometimes into, bedrock. The sides of the pit were lined with rough stones held together with mortar and the inside walls covered with a thin layer of plaster which extended around the top to form a ledge for the capstones (Plate 3). Following interment the grave was covered with a small mound of earth. Since the chambers were built close together, these small mounds gradually coalesced and were in turn pitted by other graves, finally resulting in a large irregular mound. Orientation of the chambers was variable, often apparently following the external shape of the mound. This pattern of construction is the most common form found amongst Tylos period cemeteries on the Island (Salles 1984; Herling et al. 1993, Herling 1994).

Average adult tomb size was 200 cms long, 54 cms wide and 72 cms in depth. In all instances subadult chambers were significantly smaller than adult burials, suggesting that the child graves, at least, were built to fit the body and that there was no prior preparation of tombs to death. Yet there is no relationship between tomb size and either sex, adult age, or adult height, suggesting that something other than a basic size was responsible for the completed dimensions of adult tombs. Possibly this was a factor of wealth or status, although it could equally be the amount of raw material (stone, plaster, soil) available, time, or burial location.

Burial practices

The most common form of burial in the DS3 cemetery is similar to other Tylos burials on the Island (Herling and Salles 1989; Herling et al. 1993, Herling 1994). There was a single interment with the skeleton laid supine, hands either on hips or alongside the body (Plate 4). In this cemetery at least one instance of a board underneath the body, and two examples of complete coffins, were excavated. One of these tombs was so well preserved that the shroud was still wrapped around the body. While most undisturbed graves contained grave goods of pottery and glass, these two tombs with coffins did not contain any artefacts despite the excellent preservation.

Considering the large number of multiple burials, it is unlikely that all graves had coffins, especially where bodies were placed one on top of the other. It has been suggested (Herling 1990) that coffins represent a chronological variation.

Just over 50% of the chambers contained single burials (including the empty tombs). Approximately equal proportions of individuals from all age groups, both male and female, were buried in single graves, with the exception of the youngest age group. Figure 4.1 demonstrates that significantly fewer infants were buried in a single chamber compared to older individuals.

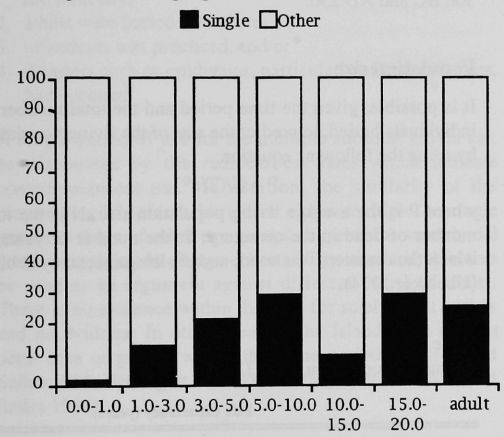
The remaining chambers all contained multiple primary interments, contrary to burial practices in other Tylos cemeteries on the Island (Herling 1990; Herling and Salles 1989; Herling et al. 1993, Herling 1994). In some cases the skeletons were clearly discrete: the second burial was found in position laid in the bottom of the chamber (often upon some silt) while the first burial had been pushed out of the way into one of the corners with the bones in varying degrees of articulation. There were cases, however, where very little time had elapsed between two burials since one body was laid out directly on top of the other with no disturbance of the original interment. This suggests that the lower body was only partly, if at all, decomposed at the time of the second burial. In other cases numerous infants and subadults were buried on top of the initial burial. As a result, the bones became entirely mixed.

Table 4.2
C14 dates for DS3 cemetery

Mound / chamber	ANU sample no.	Type	BP	SE (yrs)	Callibrated date	Error (yrs)
23 (Main)	8014	Bone	600	200		
30/1	8012	Bone	2195	164	267 BC	216
30/20	6903	Wood	2111	97	163 BC	124
69/5	8010	Bone	2600	210		
71/4	8011	Bone	3000	220		
73/18	6902A	Bone	2287	229	381 BC	281
73/18	6902B	Bone*	953	211	AD 1042	188
83/8	6905	Wood	2136	127	187 BC	157
83/29A	6906B	Bone	1431	138	AD 599	144
83/29A	6906	Bone*	156	202	AD 1709	155
100	8013	Bone	4440	210		

*Organic collagen (remainder apatite)

Figure 4.1
Percentage within single graves, DS3



In one tomb (Mound 83, Tomb 30) it was possible to clearly trace the phases of burial:

1.	Female adult supine	< 9 months
2.	Child 5–6 years	9 months +
3.	Infant c 6 months	3–6 months
4.	Child 12–18 months	<6 months
5.	Infant c 6 months	<6 months
6.	Child 18–24 months	6–9 months
7.	Infant 6–18 months	? time period
8?	Infant c 6 months	? time period
9?	Child 1.5–2 years	? time period
10?	Infant c 6 months	? time period
11.	Child c 18 months	6–9 months
12.	Child 12–18 months	

There is great uncertainty in the estimation of decomposition rates given varying climates. The time periods are a rough estimate (based on Ubelaker 1974) of the length of time before the next interment. This allows up to three months for a body to remain articulated, up to six months for partial disarticulation, and nine months for complete disarticulation. Given the humid and hot climate of the Island, these are likely to be overestimates of the time required for decay. In total, the tomb was possibly used over a period of less than five years. Unfortunately this is the only instance where we have sufficient data to make any estimate of the period of use.

In most of the multiple burials reburial occurred while the location of the grave was still obvious. Either these tombs remained open (improbable given the lack of disturbance), they were marked on the surface in some way, or they were intended for multiple use from the outset and therefore not covered with dirt after the first burial.

Each multiple tomb contained on average 6.5 individuals including approximately 2 adults and 4 subadults. The numbers in each tomb, however, varied widely from between two to 137 individuals. The mode was two and there is no evidence of a particular pattern of burial either in the order of interments or in the combinations of age and sex buried together. Within each mound multiple burials comprised nearly 50% of the graves and there is a remarkable consistency in their frequency (between 40 and 60%) with the exception of Mound 88 where all four burials were multiple. This lack of variation across the cemetery suggests that multiple burials were part of the normal burial ritual throughout the entire period of cemetery use.

The final class of burials found on site were jar burials, as well as two finds of loose bone in the fill of Mound 30. These burials tend to be more problematic. First, there is no

clear chronology (Salles 1986, Kervran et al. 1987); while some jars are clearly Tylos period in date others belong to the Late Dilmun period, including a bathtub coffin. The only jar burials discussed here are those within clearly Tylos mounds. Second, many of these jars were empty and their purpose is therefore unknown. It is not possible to assume that bones were always present, as in the case of empty chambers, since jars were also used as receptacles for food or small objects (Al Tarawneh 1970).

The frequency of jar burials varies between mounds. Primarily, those containing any skeletal material contain the body of a single infant. The oldest individual in a burial jar on the site was twelve years of age. This was an exceptional burial of two subadults within the one jar, each aged c10–12 years of age at death.

The disarticulated bones of three adults (two males, one female) and two infants were found in the fill of mound 30. The bones had probably been moved from a burial chamber which was reused since the bone was completely disordered. Removal of earlier interments, however, does not appear to have been a common practice.

Overall, there seem to have been three basic kinds of burial during the Tylos period at this site:

1. Single graves, including the coffin burials, with no distinction in either age or sex except for the under-representation of infants.
2. Multiple graves, including some with large numbers of infants, but no apparent age or sex bias overall.
3. Jar burials containing mainly infants.

If jar burials are considered to be the equivalent of a single burial, the frequency of this type of burial amongst all age groups is between 10 and 28%. These proportions do not suggest any major age related distinction. There is a lack of either sex or age related differences and the major difference in burial appears to be between those buried in either a single or multiple grave. These constitute 20% and 80% of the population respectively.

It seems that some graves were kept open simply for subadults as in the case of Mound 83, chambers 5 and 29, and Mound 73, chamber 18. On the other hand Mound 73, Chamber 1, contained 44 adults and only two subadults. Only a few chambers contained such large numbers and they are difficult to interpret in view of the overall stable proportion of single to multiple burials in each age group. The variation is possibly due to the greater frequency of subadult deaths: not only would the grave be needed more frequently, but also more subadults could be fitted in a chamber than adults.

Chronology

In terms of chronology, the lack of data on cultural finds is a serious drawback. Nine samples, seven of bone and two of wood, were submitted for carbon dating (Table 4.2). The earliest date of 4440 BP comes from the main chamber of an Early Dilmun tomb. Two samples (from Mound 69, chamber 5, and mound 71, chamber 4) were obtained from Late Dilmun chambers and the results of dating confirm this. Of the remaining dates three appear suspiciously late (23, main chamber; 73, chamber 18 (collagen), and 83, chamber 29a (collagen and apatite). It is suspected that these are the result of organic contamination in the case of the collagen dates and carbonnate contamination in the case of the apatite date. The disparity between dates obtained from the same sample confirms the presence of contamination.

The remaining dates indicate that the cemetery was used from the early Tylos period. Unfortunately the standard errors on callibrated dates are large but the uncallibrated dates are indicative of the general contemporaneity of the graves. One sample from a coffin burial in the south of the moundfield is dates to 187 BC while one from the north is 164 BC. These dates probably belong to the earlier part of the cemetery. Finds of blown glass demonstrate a continuation of use down into the Parthian period (c100 BC to AD 250). At present, based on the small amount of cultural data available, as well as the radiocarbon dates, the suggested time period covered in this cemetery is between 300 BC and AD 250.

Population size

It is possible, given the time period and the total number of individuals buried, to predict the size of the living population by using the following equation:

$$P = N/(T/e^{o})$$

where P is the average living population size, N is the total number of dead in the cemetery; T, the number of years in which the cemetery was used; and e^{o}, life expectancy at birth (Ubelaker 1974).

Table 4.3
Estimated population sizes, DS3

	Life expectancy (years)				
Time (years)	**20**	**25**	**30**	**35**	**40**
500	41	51	61	72	83
	(49)	(61)	(77)	(86)	(98)
400	51	64	77	90	103
300	68	85	103	120	138

* Figures in brackets represent adjustment for missing area.

Over a period of 500 years, a cemetery population of 1024 with variable life expectancy requires an average living population size of between 60 and 83 individuals (Table 4.3). Adjusting the cemetery population by 20% to account for the unexcavated portion only increases settlement size to some 75–100 persons. Shortening the time span covered by the cemetery has little effect on these estimates. At the same time, given mortality levels, the upper life expectancy is considered unlikely; 25 years was probably closer to reality.

While it would be possible to estimate life expectancy directly from the skeletal sample, arbitrary approximations have been used at this stage since the question is whether the sample shows signs of bias or not.

The estimated total population size is consistent with a small village. This is suggested by the surveyed settlement site adjacent to the present village. A population size of between 75 and 100 is consistent with the estimate of five years for the accumulation of bodies in Chamber 30, Mound 83. While these figures are tentative, they represent a hypothetical model that can be further tested, and elaborated upon, with a consideration of both the demographical and pathological data of the group.

Sample bias

It has been suggested, given the mass burials of infants and generally high subadult mortality (c 60%), that the frequency of infants is biased due to burial practices, with four possible explanations, that:

1. infant graves were used for a longer time than the rest of the cemetery;
2. adults were buried elsewhere;
3. infanticide was practiced; and/or
4. disasters such as epidemics, particularly affecting infants, had occurred.

A longer period of use for the multiple subadult graves can be discounted by the radiocarbon dates which indicate contemporaneous use. In addition, the similarity of the proportions of infants buried in either multiple or single graves to those of other age groups suggests the same burial practice applying to all age cohorts. This similarity can also be used as an argument against different burial of adults. There is no evidence within the area for solely adult burials and no evidence in other areas of the Island for a distinct adult area of graves within the same period (Herling and Salles 1989; Herling et al. 1993, Herling 1994; Lombard and Salles 1984).

It is difficult to argue for infanticide operating in this group. The ages of infants buried vary from neonates up to one year forming a very high proportion of total mortality. There is no suspicious peak within the first three months of life, nor is there evidence elsewhere on the Island for infanticide. Finally there is no sign of differential burial for any group of infants.

The final suggestion is a disaster in the form of an epidemic or some other circumstance. If an epidemic had taken place, however, one would expect abnormally high numbers of individuals; unequal representation of certain age groups and over representation of some age classes; evidence of a short time span between burials; and possibly evidence for extraordinary burial ritual because of the different circumstances of death (Brown 1981). An example of these conditions occuring on a wide scale can be seen in the accounts of burial during the Black Death (Aries 1980).

As demonstrated earlier, the total number of dead is not abnormal, but, rather, is consistent with a small settlement. While infant and generally subadult mortality is high, it will be demonstrated in the demographic analysis (Chapter 6) that all age groups are represented in the expected frequencies. Most importantly the evidence from Chamber 30, Mound 83, demonstrates accumulation over at least 12 months. This chamber had a similar age profile to other multiple tombs, and mirrors the composition of the cemetery population, making it possible to envisage accumulation over time in other multiple tombs. Certainly some tombs contain extraordinary numbers of subadults; it may be that some tombs were reserved for infants. Yet the remarkably stable proportion of multiple to single interments, regardless of age cohort, suggests that these multiple infant burials were part of the normal ritual.

Burial status

Reconstruction of social status from burial is notoriously difficult to evaluate (Chapman and Randsborg 1981, Morris 1987; Chapman 1995). In the present case, there are very few visible differences in burial practice beyond the multiple/single division and the small group buried in coffins without grave goods.

This group remains a mystery. Due to the vagaries of preservation, it is impossible to estimate the total number of individuals buried in coffins. Yet we can assume that multiple burials, especially where one burial has been laid directly on top of another, or where the lower bones are completely disarranged, did not contain coffins. The rarity of discoveries of wood in other Tylos cemeteries on the west coast of the Island, despite varying preservation conditions, suggests that the infrequency of these burials is not merely an artefact of preservation. In contrast, Herling (1990) reports the more common presence of backboards and wooden coffins in the Tylos cemetery of Karannah and at Saar (Herling et al. 1993) and suggests that this has chronological significance. Given the small number of tombs with coffins at DS3, as well as the radiocarbon dates which indicate these burials were contemporary with the main period of cemetery use, simple chronological variation cannot be the total explanation for the use of coffins at this cemetery.

The other major distinction of single versus multiple burial crosscuts any age and sex differences. At Saar, detailed analysis by Herling (1994) suggests a chronological basis for the distinction. This does not, however, apply at DS3 since it occurs in similar frequency in all mounds not just on the edges of mounds and also within a short space of time. The presence of the single/multiple division suggests something other than an entirely egalitarian society, while the persistence of the division throughout the entire period of cemetery use belies the possibility of either chronological variation or simple conservatism being the cause.

The meaning of individual mounds also remains obscure. Two possibilities suggested are that mounds were either used

successively by the entire population or by a family or other sub-groups. The age distributions represented within each mound demonstrate an equal distribution of males and females, as well as the older subadult groups, but the frequency of infants varies greatly. This suggests the use of more than one mound at the same time. If a mound was used by a smaller group, rather than the entire village, burials would be spaced further apart, yet many chambers demonstrate that little time had elapsed between burials (eg. Mound 83, Chamber 5).

Most probably a particular mound was used by a larger group than the immediate family during overlapping time periods (more than one mound in use at the same time), rather than used exclusively by a small group such as a family. The variation in the numbers of tombs per mound also supports this. In addition, the stable proportion of multiple to single graves in each mound strengthens the hypothesis that a mound represents use by more than one small group. If only one small group was using a single mound then we could expect mounds with solely single tombs or solely multiple tombs, yet there is only one case where all chambers within a mound contained multiple burials. The suggestion that more than one mound was used at one time has important implications for other cemeteries on the Island. There is no support for the idea that each mound is representative of the entire burying population in this cemetery, so analyses based on excavation of a single mound may be severely biased.

Burial practices at DS3 can be summarised as demonstrating:

1. no differences between the sexes;
2. no differential treatment of subadults except for the jar burials of infants rather than chamber burials;
3. a lack of obvious status graves, and generally a lack of differentiation of burial except for the single graves which affect 20% of the population and may reflect wealth or social group;
4. the cemetery probably serviced a small agricultural village of probably between 75 to 100 individuals.

Taking into account the low percentage of 'empty' tombs and the possibility of estimating the age cohort of missing skeletons, the skeletal sample from DS3 can be expected to show few signs of bias due to preservation. In addition the general uniformity of burial across the cemetery, lack of complex variation in burial practices (especially according to age or sex), and the completeness of the collection (80% of the entire cemetery) support the hypothesis that the skeletal collection from DS3 represents an unbiased sample of a village population.

Saar Mound 5

Site description

This mound was excavated during 1987/8 by the Directorate of Archaeology, Bahrain, as part of a rescue excavation. It was a large Tylos mound located to the northwest of Saar village (Plate 2). As such it forms part of the Tylos cemetery around which the village is situated. Sections of this have been excavated by the Danish (Glob 1959), Australians (Petocz and Hart 1981), numerous Bahrainni excavations, and a German team (Herling 1994).

On the aerial map from c 1980 it is possible to distinguish a minimum of 28 post Early Dilmun mounds. This number must represent a true minimum since there has been extensive building around Saar Village, and even in 1979 several of the mounds had been at least partly damaged by house construction (Petocz and Hart 1981).

The mound itself contained 106 tombs as well as 19 burial jars. Twenty-seven of the chambers were empty although overall skeletal preservation was extremely good. It is assumed that the empty chambers are due to disturbance of the tomb as at DS3 and/or recovery technique. This is further confirmed by the fact that the majority of empty tombs were found on the outer edges of the mound where they were most accessible to robbery.

Construction of the tombs is identical to those at DS3. Average chamber size is similar, with the same relationship between tomb length and age of the deceased. Since a greater percentage of tombs were found empty in this particular excavation it has been necessary to estimate the age of more of the primary interments. The age groups of the empty tombs can be seen in Table 4.4 and demonstrates that the pattern of preservation in tombs does not discriminate towards adults.

Table 4.4
Estimated missing individuals from Saar

Age group	Number estimated
0–2.9	4
3–5.9	3
6–9.9	1
Subadults	10
Adults	17

Burial practice

As in DS3, though more markedly, the most common form of burial (82%) is a single primary interment with the body supine. Frequently pottery was found, not only placed around the edges of the body, but also between the legs. An equal frequency of males and females were found in these graves. Similarly, there is no statistically significant difference between age cohorts in the percentage of individuals buried singly.

About 18% of all graves contained multiple burials. This is a significantly lower percentage than in any of the mounds from DS3. There is also much less variation in the numbers buried per tomb: the maximum number buried in a single chamber was only 5. As at DS3, however, burial in multiple

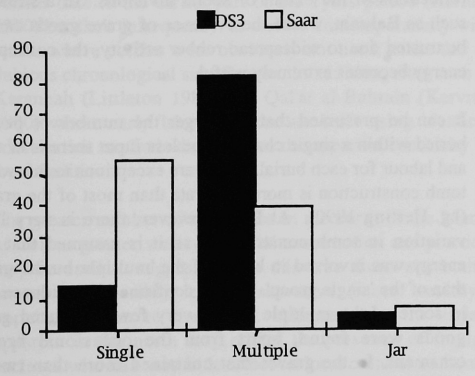
tombs crosscuts any age/sex categories. Approximately 60% of both adults and subadults were buried in chambers containing more than one interment. Again there is evidence of varying periods of time between the interments.

To some extent, these figures concerning multiple burials are misleading since on this site there were large numbers of burial jars containing not only infant remains, but also mixed adult remains. Jars form approximately 20% of the total number of burial containers (26/132). By their location, some appeared to be specifically related to a burial chamber. It is hypothesized that in some cases, the jar contained the original interment within the chamber which had been cleared out for another burial. Such secondary deposits of bone were apparently a variation of clearing the chamber floors of disarticulated bones as seen at DS3.

There were still the single infant burials within jars which was a common theme throughout the Tylos period (Salles 1986). As at DS3, these seem to be the equivalent of single subadult burials within small chambers. Yet the reclassification of these jars as either multiple or single burials makes little difference to the final proportions of single as opposed to multiple interments. In total, the percentage of single interments at Saar Mound 5 is still significantly higher than at DS3 (Figure 4.2).

Despite generally good preservation conditions there is no evidence for the use of full coffins within this mound. Overall there appears to be little evidence for social stratification in terms of energy expenditure.

Figure 4.2
Distribution of burial types, Saar & DS3



Biased or not?

The consistency of the proportions of single to multiple burials for each age group and the lack of visible sex-related differences in treatment, suggest that the mound represents an unbiased sample of a local population. Yet the higher frequency of single burials in Saar Mound 5 mean that a higher percentage of children were buried in jars. Preservation in jars is noticeably poorer and it is also impossible to estimate how many jars would have contained infants since some jars on this site contained adults. This means that, unlike DS3, the extent of shortfall in skeletons, particularly infants, cannot be estimated. Furthermore, the excavation at DS3 demonstrates that, while most age groups are evenly distributed across mounds, the youngest age cohort is not. Since only one mound was excavated at Saar during the 1988 excavation this adds a further degree of uncertainty concerning the frequency of the youngest age cohort.

This possibility of underestimation is further confirmed by a brief examination of the spatial distribution of the graves. As mentioned earlier, empty tombs are concentrated on the edges of the mound. At the same time, there is a slight tendency for multiple tombs to be concentrated near the outer perimeter of the mound, possibly because of easier accessibility or due to chronological variations (Herling 1994). This may mean that, by assuming the empty tombs represent a single burial, the total number of burials is underestimated. This is balanced, however, by the greater chance of skeletal preservation in a multiple tomb. Therefore, although it is not possible to estimate a number of individuals any higher than the bare minimum, the possibility of underestimation must be kept in mind.

There is a third area of possible underestimation, again connected with spatial distribution. The burial jars tend to be found concentrated near the centre of the mound, not near areas of empty tombs. Yet child burials are generally found distributed across the mound. It is suspected that robber activity on the perimeter of the mound may have destroyed some burial jars resulting in the present uneven distribution. Loose sherds were found in the mound fill but their attribution to burial jars is difficult. Again this would create a possible shortfall, primarily of infants, but the extent cannot be estimated.

The probability is that, although the older age groups found in the mound are representative of the living population, the infant age group in the skeletal sample from Mound 5 is under-represented by an unknown amount. Furthermore, the total number of burials excavated may be an underestimate of the original number of interments, at least by a small amount.

Chronology

The finds of pottery and glass in this mound are consistent with a date to within the middle Parthian period (c100 BC–AD 100). This dating is supported by a series of radiocarbon dates (Table 4.5) placing the mound between 150 BC and AD 100. From this data it is possible to hypothesize the size of the living population using the formula supplied by Ubelaker (1974). The results can be seen in Table 4. 6.

Table 4.5
C14 dates from Saar Mound 5

Mound / chamber	ANU sample no.	Type	BP	SE (yrs)	Callibrated date	Error (yrs)
4	8008	Bone	1924	215	AD 256	57
61	8007	Bone	2087	176	220 BC	135

Population size

Consistent with the small number of individuals, population sizes are quite low (Table 4.6). Rejecting a life expectancy of 40 years as too high (based on a comparison of contemporary life expectancies), only c 50 people were needed to produce this number of dead in 100 years, 100 in 50 years etc. As indicated above, since it appears likely that infants are under represented, this is unlikely to represent the total settlement size.

Table 4.6
Estimated population size for Saar Mound 5

	Life expectancy (years)				
Time (years)	20	25	30	35	40
50	60	75	90	106	120
100	30	38	45	53	60
150	20	25	30	35	40

An estimate of settlement size needs to take into account the entire cemetery. As of 1980, this consisted of 28 burial mounds. Yet we have no clear idea of the number of burials per mound which can can vary widely. Nevertheless, even using a low estimate of 50 burials per mound at Saar results in 1400 burials. A higher estimate of 100 burials per mound would mean 2,800 burials suggesting that the Saar cemetery was either used over a much longer time than DS3 (unsupported by the archaeological finds) or that it served a larger settlement. This hypothesis is further supported by the fact that by the time the 28 mounds were counted destruction of the cemetery had already commenced; the total number of mounds may have been significantly higher (c35–40?).

The suggestion of a larger settlement population at Saar than al-Malikiyah corresponds to Larsen's work on village size ranking (Larsen 1983a; 1986). It also indicates the probability of more than one mound being used at once. This agrees with the data from DS3, but means that demographic or social reconstructions based upon the analysis of a single Tylos mound must be treated with caution.

DS3 and Saar Mound 5 compared

Burial practices at both these sites can be classified into the same three groups: single burials, multiple burials and jar burials. The major difference between the two sites is in the proportion of individuals buried singly. At Saar this comprises 50% of the sample, at DS3 only 20% of the bodies were buried alone. Even so, on both sites the only sign of ordering according to age or sex is the placement of infants in burial jars.

In general on Bahrain, the extensive disturbance of tombs by robbers means that it is extremely difficult to evaluate burial practices in terms of the material artefacts found in the grave. Status, however, is not solely demonstrated by the number or quality of objects buried with the dead. Tainter, in a survey of ethnographic literature, found that status of the living person could be inferred from the amount of energy expended on their burial (Tainter 1978). The notion of energy encompasses not only the effort involved in burial construction, but also the value of material used.

It must be noted that in anthropological studies, the actual burial is only last in a series of activities which may reflect status and involve members of the community (Metcalfe and Huntington 1991). The archaeological material recovered only dimly reflects this range of activities. Yet differences both within and between cemeteries in the degree of energy involved in burial and in the range of expenditure are reflections of the extent of social divisions. In a situation such as Bahrain, where the evidence of grave goods cannot be trusted due to widespread robber activity, the concept of energy becomes extremely useful.

It can be presumed that the larger the number of people buried within a single chamber the less input there is of time and labour for each burial. There are exceptions to this where tomb construction is more elaborate than most of the graves (eg. Herling 1990). At DS3, however, there is very little variation in tomb construction, so it is assumed that less energy was involved in burial of the 'multiple burial' group than of the 'single group'. This is confirmed by evidence that in some of the multiple graves very few associated grave goods were found, apart from the occasional bronze ornament. In the graves that contained more than twenty individuals the reason for this may simple have been lack of space, but even in this case the decision to reuse an old tomb rather than build a new one is indicative of the 'least effort' principle.

At DS3, 80% of the population was buried in this fashion. At Saar only 50% were buried in a communal grave, and on top of that, the numbers in each communal grave were lower corresponding to the notion that more energy was expended

on burial at Saar than at DS3. Additionally, at Saar, disturbed remains were placed in jars rather than simply thrown out or cleared away. Again, this process involves more time, energy, and material goods.

At neither site, however, is this expenditure ordered with age or sex. The remarkable consistency of the proportion of single to multiple burials across age and sex categories suggests a fairly rigid difference in burial practices right across society in these two areas. Yet this distinction is not visible in all Tylos cemeteries on the Island suggesting differences in social organisation between settlements on the Island.

Tylos cemeteries on Bahrain

There have been frequent excavations of Tylos cemeteries on Bahrain. While very few excavation reports have been published, it is possible to discern several common features from unpublished reports, museum displays, and conversation with those present during excavation.

First, the cist form of burial is the most common throughout the period. Construction varies from some elaborate stone slab construction (Salles 1984); to slab tombs reusing slabs and headstones (Littleton 1983); mortar and rock construction (Lombard and Salles 1984, Herling 1990, Herling et al. 1993, Herling 1994); and tombs cut into bedrock (Herling 1990, Al Wohaibi 1980). There are scattered instances of simple pit graves (including some with a headstone, eg. A'ali). Some chronological trends are discernible in these construction methods within sites (Herling 1994). Yet is is unclear whether these chronological divisions apply across the whole island. It is possible that full coffins date to the earlier part of the Tylos period (Herling 1990) but their infrequency, as well as contemporaneity with tombs where coffins were obviously not used, make this of dubious chronological significance. In addition, sites such as Karannah (Littleton 1983) and Qal'at al-Bahrain (Kervran 1990) where early Islamic tombs are built using identical construction methods demonstrate the apparent continuity of local tradition.

The greater number of cemeteries in the north of the Island, as well as greater variety in burial construction and practice, may be used to suggest that in this area there was both a larger and possibly less conservative (more wealthy?) population than to the south. Alternatively, the variety apparent in the north may reflect the presence of foreign enclaves who had very little effect on local cultural traditions (Salles 1986, 1987). In any case, the increased variety and greater range of variability in energy expenditure implies a more complex social organisation than is visible at DS3.

Cemetery and settlement

It is argued that the pattern of Tylos cemeteries reflects settlement distribution throughout this period. The argument rests upon the following points:

1. the close relationship between Early Dilmun burial patterns and the Tylos period;
2. the presence of Tylos cemeteries bordering arable land, and, when in arable land, their proximity to water sources;
3. the continuity of this pattern into the Islamic period, and the relationship between some current villages and Tylos cemeteries; and
4. analysis from other ethnographic, historical, and archaeological studies which demonstrates a close link between formal, bounded cemeteries and control of resources.

Early Dilmun burial mounds, particularly of the late-type, are clustered into definite regions of the Island. These areas clearly border arable land and density throughout the moundfields tends to vary, being most dense along wadis and on the edge closest to arable land (B. Frohlich, pers.com.). There is one possible exception to this. The al-Hajjar burial field (including Karannah) which is situated in the north of the Island, may have encroached onto arable land though even within irrigated lands there are areas of waste caused by necessary earth removal from the gardens (Paskoff and Sanlaville 1984).

Both Larsen and Frohlich have noted that the burials mounds cluster into discrete and clearly localized areas. They see this as indicative of a nearby settlement or cluster of settlements (Frohlich 1986, Larsen 1986). Survey data would appear to confirm this in some instances.

The clustered distribution of mounds is even more obvious in the Tylos period. The majority of cemeteries, particularly those along the west coast, are focussed upon a small section of the earlier moundfields. Invariably this is situated at the very edge of arable land. Cemeteries along the northern coast are often, though not invariably, located within areas of non-productive land, or, as at Janussan South, on the remnants of soil dumps (Paskoff and Sanlaville 1984). Despite the availability of waste land, these burials are located in dense groupings. A relationship can be postulated between the siting of these cemeteries and the existence of nearby springs.

Even today this pattern holds, with some earlier burials forming the basis for Islamic cemeteries. This is most obvious along the north coast, for example Karannah North, Shakoura, sections of Al-Hajjar, and Jidhafs. Obviously the pressure on land means that cemetery location in the north is constrained. At the same time, despite widespread population redistribution on the Island, frequently individuals are still buried according to the location of the original family village (A. Sowaileh, pers. com.).

At the same time many of the ancient cemeteries are associated with modern villages. This is most clearly evinced by examining the list of villages extant in 1908 (Lorimer 1908) and the location of Tylos cemeteries. For instance, in the area of the Saar cemetery lies Saar village, consisting of

(in 1908) 30 huts of farmers, 17 donkeys and 7 cattle. The gardens comprised about 10000 date palms. DS3 cemetery is located behind al-Malikiyah village which, in 1908, consisted of '100 huts of Bahrainah, engaged in cultivation. The dwellings stand amidst dates ...Among the livestock are 18 donkeys and 10 cattle. Dates are about 6000' (Lorimer 1908 II:224). The historical link between settlement, cemetery, agricultural land and water is clearly apparent. Cemetery location was not a haphazard event: the clustering, the reuse of some areas, and ignoring other suitable areas makes this obvious.

This link between burial location, settlement, and agricultural land has been studied in a variety of anthropological, historical and archaeological cases. Saxe, based on a survey of ethnographic literature, explicitly stated the nature of the relationship (Sax-e 1971) which was later recast to:

> If a permanent, specialised bounded area for the exclusive disposal of the group's dead exists, then it is likely that this represents a corporate group that has rights over the use and/or control of crucial but restricted resources. This corporate control is most likely to be attained and/or legitimised by means of lineal descent from the dead, either in terms of an actual lineage or in the form of a strong, established tradition of the critical resource passing from parent to offspring (Goldstein 1981:690).

As Goldstein emphasises, a formal cemetery is only one way of legitimising descent. Similarly territorial pressures are not the only symbolic function of cemeteries.

Ancestors, however, are frequently used to legitimate claim to land, and the most direct way of indicating their presence is by a clearly defined burial. The use of burials as indicators of ownership is clearly seen amongst the Mbeere of Kenya (Glazier 1984). Traditionally, these people practised shifting cultivation and small-scale herding therefore they were mobile and did not experience any shortage of land. Their pattern of burial was simply corpse abandonment. Under a government attempt to confer land rights and the prohibition of corpse exposure, this practice has now changed and "a grave site now establishes a visible connection between a particular territory and forbears buried within it, thereby forging new and socially valued links between the land and its claimants" (Glazier 1984:44).

Historically, the same link between cemetery and land division can be seen. For instance, in Ireland early seventh century property laws were based upon location of burial mounds (Chapman and Randsborg 1981). Throughout Britain and Europe, the association of cemetery and land division is apparent (Chapman and Randsborg 1981). In Britain, early Anglo-Saxon burials were found on existing land boundaries between different communities and apparently served as boundary markers (Bonney 1976). Within Australia the pattern of Aboriginal burial changes regionally from dispersed burials in areas of known low population density, prior to European contact, to clearly localized and bounded cemeteries in locations sustaining high populations such as the Murray River (Pardoe 1988). In Bahrain, today, the insistence on burial within the ancestral cemetery can be seen as part of the same trend.

There is no rule to state that there need be either only one cemetery to one settlement or only one settlement to one cemetery. There is, however, clear evidence to suggest that the existence of a formal and bounded cemetery reflects the presence of nearby resources, either land or water, controlled by descendents of those buried within the cemetery.

In the case of DS3 and Saar cemeteries, as well as other Tylos cemeteries on the Island, the location of graves was obviously marked by the large burial mound covering the graves. The mounds were highly visible objects within the landscape. At the same time, the multiple graves at DS3 were probably marked in same way, which suggests that temporary markers were used on the mounds' surface, thereby accentuating their visibility.

The number of burials within each of these cemeteries is consistent with a small village. Given that, while arable land is in short supply, non-arable is not, the presence of a Tylos cemetery at al-Malikiyah and one further south implies that those burying their dead in these locations had control over some nearby resource. The cemeteries in the Tylos period, as in the Early Dilmun and the later Islamic periods, can be seen as indicative of the spread of settlement and agriculture on the Island during this period. The parallel between the size of villages at al-Malikiyah and Saar in the historical period and the hypothesised population sizes for the Tylos period is apparent.

Settlement during the Tylos period was probably most dense in the north and, based on the variability of burial practices in this same northern area, these settlements probably also represented a wealthier, possibly less conservative, and more stratified section of the Island's population.

Conclusion

At DS3 the location and size of the cemetery suggests that it was connected to at least one agricultural village located in the arable lands nearby. The lack of elaboration in mortuary practice supports this model and makes it possible to demonstrate that the skeletal sample deriving from this cemetery represents an unbiased sample of the living population. Nevertheless, there is evidence of social differentiation independent of age and sex (i.e. single vs multiple burials) and of co-operative organisation above the immediate family (unequal distribution of infant burials).

At Saar, there was slightly more elaboration, certainly more expenditure on mortuary practices, while the burying population was probably larger. This may suggest that social organisation in this area was more comparable to the northern settlements than to small agricultural villages along the west coast. The larger population size and greater use of

single graves, however, means that Saar Mound 5 does not represent an unbiased sample of the living population. It appears probable that the youngest age cohort is under-represented due to the combined problems of poor preservation (in burial jars) and inadequate site sampling.

The present data also point to the possible existence of significant differences between areas of the Island in terms of population size and social organisation. In the following chapters the demographic and pathological evidence from both sites will be examined with the aim of reconstructing social organisation while taking into account the reservations mentioned above.

Chapter 5

Step 1: Recording of age, sex and pathology

Introduction

Methods of recording, while adhering to general principles (outlined in Chapter 2), vary according to the nature of the samples. The range of age groups represented determines what methods of age estimation need to be used. Age and sex estimation methods also depend upon the degree of preservation, completeness of the collection, availability of time and equipment, and whether or not bones may have been destroyed. Similarly, the estimation of sample size depends upon the nature of burial, whether in individual graves, mixed multiple graves, or after secondary interment. This factor (whether one is dealing with individuals or comingled remains) affects the recording of all skeletal characteristics, including pathological lesions.

In Chapter 2 general principles of conservatism in estimation, use of multiple methods, and assessment of representatives were described. The issue of representativeness was dealt with in the preceding chapter. In this chapter the specific methods that were used in estimating age and sex of individuals and in recording the specimens will be discussed and explainned.

Age estimation

The greatest problem in recording these samples was the comingling of remains from multiple graves. This placed restraints upon the number of methods that could be applied to individuals, and upon the nature of these methods of estimation.

Subadult age

Perinatal and neonatal deaths

While dental development is the most accurate indicator of the age of subadults, including foetuses, the lack of preservation of the very youngest infant remains means that it is difficult to determine which deaths have been those of neonates, and which were of older infants. This problem has been encountered in several studies (eg. Stewart 1979), and becomes especially acute in the present case where numbers and ages of infants have to be calculated on the basis of individual bones and the resulting age distribution curves compared (as explained in the calculation of minimum numbers).

The distinguishing of foetal and neonatal skeletons from post-natal deaths has been approached in several ways. Several modern studies, commonly used in forensic cases, present standards for each long bone using either linear regression (Schuer et al. 1980) or ranges for each week (Kosa 1989). In contrast, Stewart (1979), when dealing with the Arikara skeletons plotted the frequency of long bone lengths and isolated the mean of this distribution. He claimed, based on the assumption that most deaths occur directly at birth, that this length (c77.5 mm for the femur) was the mean femoral length for full-term babies. He further isolated the range from 73.5–81.5 mm as the amount of variability present in newborn femoral lengths. Infants with long bone measurements less than that were either pre-term or small for age.

In a study of infant skeletal material from Teohituacan, Storey (1986) could plot dental development of foetal skeletons against their length. Her work demonstrated that small for age infants were common and that growth had slowed during the last month in utero.

In the present sample, there is clear evidence to suggest that the population was smaller at all ages compared to modern whites as well as the Arikara Indians (see following section). This indicates that it may be inappropriate to use standards based on any of these groups, although there is a general convergence of all modern growth curves, based primarily on European populations, around birth: Oliver and Pineau (1958) found an average femur lengths of 73 mm at 10 lunar months; Kosas (1989) 74.3 mm; Schuer et al. (1980) 80 mm. A further confounding factor is that the current sample is a mortality sample rather than a living population.

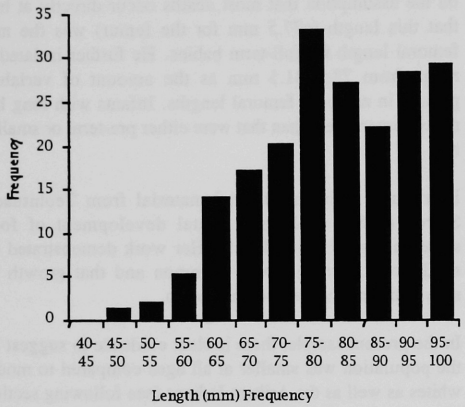
Stewart's method (looking for a peak frequency of femur length to indicate birth) was attempted on the infant bones from Bahrain (Figure 5.1). A plot of long bone lengths of infants less than six months failed to indicate a peak incidence of measurements; rather, there is an even distribution of femur lengths from c 65 mm onwards. The average femur length for the few neonates that could be aged from teeth (n= 3) was 67.5 mm.

It appears, therefore, that a femur length of between 65–70 mm was probably the average length of infant femora at birth. This figure is at the lower end of the range in other studies (Stewart 1979, Storey 1986). Accepting 65–70mm as average length, all smaller infants may then represent either small-for-age babies or preterm deaths. Considering the large number of infants involved and the lack of accompanying female skeletons, it is improbable that all smaller infants represent foetal deaths. It seems more probable that a sizeable percentage are small-for-age infants, a phenomenon that will be discussed more fully in Chapter 7 (childhood growth).

Given the rate of infant growth seen in other studies (Oliver & Pineau 1958; Schuer et al. 1980), it is probable that femur lengths of less than 75mm represent deaths from birth (or prior to) to the end of the first month. Femoral lengths of less

than 70 mm may be categorised as neonatal deaths but this probably includes some foetal deaths.

Figure 5.1
Frequency of femur lengths <100mm



The inclusion of some foetal skeletons in the demographic analysis, while unavoidable, may result in a slight overestimation of infant mortality. By accepting the lower end of the range of femoral lengths at birth (i.e. 70mm rather than 75 mm which is the average of modern studies), deaths at birth may be underestimated in favour of the broader category of neonatal/perinatal deaths (i.e. deaths within the first month). Considering, however, the small size of all the subadult remains, and the apparent slow growth of the subadults, it appears better to be conservative in deciding a 'neonatal length' rather than to overestimate the number of neonatal and foetal deaths which may have occurred.

Subadult ageing (post-perinatal)

Ubelaker's chart of dental formation and eruption was used for the estimation of subadult age (Ubelaker 1978). In cases where dental age could not be estimated the degree of epiphyseal closure was used as an alternative method (Brothwell 1981). This method was primarily used when dental material was missing.

Since so many of the subadults were buried within multiple graves, it was frequently necessary to age dental remains independently of the rest of the skeleton. In other cases only long bones were preserved. To age these remains a growth chart was constructed plotting the long bone length against dental age of each complete skeleton. This could then be used as a basis for the estimation of dental age from long bone length. A brief comparison of the resulting curve demonstrated that using either the growth charts of North American Indian groups or Modern White standards (the two most commonly used charts in skeletal research) would lead to serious underestimation of dental age. Even though the number of complete skeletons from the two Bahrain samples was relatively small, it was decided that the 'Bahraini' growth curve was preferable. In order to avoid spurious accuracy in estimation, age assessments were within one year for children up to three, then broader age categories of 3–5.9, 6–9.9, 10–14.9, were assigned (Table 5.1). In many cases the age assessments of older subadults could be cross-checked with the degree of epiphyseal closure, allowing further confirmation of accuracy.

Adult ageing

Ageing of adults was most difficult. Variable preservation plus comingling of skeletons meant that a rigid multifactorial approach could not be adopted. The methods of age estimation used included: pubic symphysis metamorphosis, changes in the sacroauricular surface, and cranial suture closure, using both the vault and antero-lateral sutures. Three useful indicators, bone trabeculation, osteon ageing, and dental wear, had to be ignored: the former two due to the lack of equipment, while the rate of dental attrition could not be used on this sample since inherent assumptions of the method could not be met.

Several methods of pubic symphyseal ageing have been devised. The two basic are those by Todd (1920, 1921) and by Stewart and McKern (see Stewart 1979). The second method has been found to be strongly susceptible to interobserver error, as well as complicated to use (Suchey 1979). Todd's method, on the other hand, has undergone extensive revision and testing (eg. Angel et al. 1986). A recent revision by Meindl et al. (Meindl 1985) was used for the assessment of age since this method most closely agreed with the amendments suggested both by Suchey and Angel (see Angel et al. 1986), as well as introducing a more clearly assignable series of stages.
At the same time the sacroauricular method was used since, as Lovejoy and coworkers point out, this area of the pelvis tends to be better preserved than the pubic symphysis (Lovejoy et al. 1985a). The obvious advantage of this outweighs the disadvantages of difficulty in recognition of various phases, the lack of race and sex specific standards, and lack of extensive testing (Iscan and Loth 1989).

The two final estimates were based on the closure of the ectocranial sutures. While suture closure is notoriously unreliable (Perizonius 1984), the method devised by Meindl and Lovejoy (1985) seemed to provide a technique of at least narrowing down the ages of adults represented by cranial remains only.

Incomplete closure of the iliac crest and clavicular epiphysis was also used as an indicator that an individual was less than 30 years of age (Brothwell 1981).

Records were made of each age estimate, and no correlation or comparison was made between these estimates at the time of collection. Since bones were sorted on a weekly basis, the pubic symphysis and sacroauricular stages were checked by

Table 5.1
Comparative skeletal growth

	FEMUR			HUMERUS			TIBIA		
Age (years)	av.	s	n	av.	s	n	av.	s	n
0	67.5		3	69	12.7	2	54	5.6	1
0.25	84.1	6.8	10	77.7	11.7	19	71.1	6.8	9
0.75	100.5	9.8	26	80.5	8.3	13	82.9	10.4	12
1.5	122.2	13.3	12	95.6	8.7	14	93.8		8
2.5	136	2.8	2	107	7.1	2			
3.5	158.6	16.6	8	122.4	10	8	147.5	3.5	2
4.5	173	7.5	5	125.8	8	5	138	10.6	3
5.5	196.4	3.6	5	140.5	8.3	4	153	8.5	25
6.5			0	125		1	139		1
7.5	212		1	155		1			
8.5	230.5	17.7	2	169		1	184	19.8	2
9.5									
10.5	280		1	203.5	13.4	2	143		1
11.5	260		1						
13.5	319		1	209.8	29.2	4	231		1

	RADIUS			ULNA			FIBULA		
Age (years)	av.	s	n	av.	s	n	av.	s	n
0	45		1	51		1	51		1
0.25	55.6	5	9	63.6	6.1	7	66.8	2.6	4
0.75	62.7	5.9	15	72.8	2.7	5	82.8	10.9	6
1.5	75.9	7.8	8	81.8	8.8	13	96.3	10.1	3
2.5									
3.5	93	11.3	2	103	11.8	3			
4.5	109			108.5	12	2			
5.5	111	2	3	123	6.1	3			
6.5							152		1
7.5	136.3	6.4	3						
8.5	128		1	135		1			
9.5									
10.5									
11.5	150.5	9.2	2	178		1			
13.5	173.5	33.2	2	190.5	27.4	3	215		1

progressive seriation of results, and the earliest estimates (the first 20 adults) were rechecked. In addition photographs were taken of sample sacroauricular and pubic symphysis surfaces for further checking after the collection of all the data. This resulted in a significant decrease in observer error.

While it was judged inappropriate to use a multifactorial method of ageing, age estimates based on different methods were compared to check for systematic bias and inaccuracy. Comparison of ages based on the pubic symphysis and sacroauricular area demonstrated a lack of consistency in results in the 20–24, 25–29, and 30–34 year age groups (Table 5.2). This was not sex-specific and there was no evidence of the overaging of females by the pubic symphysis method (Meindl and Lovejoy 1985). Rather, sacroauricular ageing compared to pubic symphyseal estimates resulted in older age estimates for both males and females in these age groups. Between 35–50 years of age there is a close correlation between the estimates, while after 50 years there was a tendency for estimates based on the sacroauricular surface to be lower. This is to be expected in an open ended age group. The lack of matching in scores between the ages of 20 and 34 years, however, indicates that the inaccuracy of the methods, when used on samples without independent ageing, is too great to justify the use of 5 year age cohorts. When 10 year cohorts are used the match becomes better, particularly in the 30–40 year age group (Table 5.2). There is still a tendency to overage those estimated between 20–30 years by the sacroauricular method but this can be controlled by incorporating the data on incomplete epiphyseal closure since incomplete fusion of the iliac crest indicates a probable age of less than 30 years (Brothwell 1981, Jackes 1992).

Table 5.2
Pubic symphysis vs sacroauricular ageing

Symphysis	Sacroauricular estimate						
Age (yrs)	20–	25–	30–	35–	40	45–	50+
20 – 25	3	4	4				
25 – 30	1	2	7	2			
30 – 35			1	2	1		
35 – 40			1	12	1	1	
40 – 45					3	–	1
45 – 50				1	–	7	5
50+					2	1	8

Epiphyseal closure agreed well with pubic symphyseal stages and acted as a control on ageing in this group. Also, development stages were most clearly defined between 20–30 years, so that in this age group observer error was minimized. Overall, however, the tendency was for the youngest to be overaged and the oldest to possibly be underaged.

These comparisons suggest that assessing palaeodemographic results is not so simple as declaring that

all estimates are too young. It also supports observations by Koningsberg and Frankenberg (1994) and Bocquet-Appel and Masset (1996) of the mimicing of the original reference samples. In this case, because of the multiple age estimation methods available, the 20–30 year age group is the most accurately aged adult cohort.

A comparison of the two cranial suture age estimates against both pubic symphysis and sacroauricular methods demonstrates the variability inherent in these methods (eg. Table 5.3). Unfortunately, at least in this sample, the variability in age estimates suggests that ageing based on cranial sutures is too inaccurate to be used as an independent indicator. Even the use of 10 year age groups fails to resolve this problem. The only correspondence between cranial and pelvic age estimates is when the complete range for each phase of suture closure is used (Table 5.3). Since these ranges are not only very wide, but also markedly overlap, this appears to be of limited value.

Table 5.3
Pubic symphyseal vs A-L suture estimates

Symphysis	Antero-Lateral Suture			
Age (yrs)	20–29	30–39	40–50	50+
20 – 29	3	1	2	
30 – 39		4	5	
40 – 49			9	2
50+				3

Rather than classifying all adults aged cranially as simply unaged, it was decided to use the widest ranges as a basic indicator of age and assign individuals to 10 year age cohorts by curve fitting. For example, if three individuals are aged by the degree of vault closure to between 32–49 years, then they are apportioned to the two age cohorts, 30–39, 40–49, on the basis of the normal distribution of individuals in this sample.

When using this method, however, it is necessary to be cautious that the age distribution of crania is not the result of preservation factors rather than truly representative of the age distribution. In cranial age estimates substantially more old adults were represented. This bias to old adults was not due to the method since regression of cranial age against pelvic indicators demonstrated wide but random variability. The problem is that a completely fused skull has greater probability both of surviving relatively intact and of being recorded than an unfused skull, particularly in multiple tombs. Thus samples aged solely by cranial suture closure may be consistently biased towards older age categories compared to those aged on the pelvis, simply because of preservation.

In the present instance it was decided that, in order to avoid this dilemma and yet still use the data based on cranial suture closure, all totally unaged adults would first be apportioned to age cohorts based on the age distribution determined by pelvic indicators (as described above). The individuals aged solely by crania would then be apportioned to their respective age cohorts by the method outlined above. This helps to standardise the normal distribution of age estimates to those based on the pelvis and avoids possibly biasing the sample to older ages by apportionally the individuals aged by cranial sutures first.

In this way all ageing has been standardised to estimates based upon pelvic indicators, while avoiding the systematic biases apparently inherent in the ageing of younger specimens by these methods. Unfortunately, while the age distribution subsequently obtained is useful on a population basis and can be used for demographic analysis, it cannot be used for individual ages, which are the basis for palaeopathological comparison. Therefore, rather than increase inaccuracy, it was decided that analyses of pathology would operate on two levels–based on the total number of adults (divided by sex where necessary) and on the basis of narrower age groups counting only those individuals aged by pelvic indicators.

The result of the preceeding method is a conservative age estimate. Yet the variability in the methods of age estimation fails to justify in this sample dependency upon five year adult age cohorts. It also calls into question analyses based solely upon the degree of suture closure, particularly where preservation is not complete. Even multifactorial methods are of doubtful value when, due to incompleteness, a large proportion of cases are still aged by only one technique. At this stage further work really needs to be done on a wide range of samples, identified by race and sex, to determine the relationship between each method and absolute age, and between the methods themselves, so that age estimates can take into account the full range of variability for each age phase (Koningsberg and Frankenberg 1994). In individual cases it will probably always be necessary to use the widest range of ages available. Until these ageing methods are improved, five year age cohorts, except in the most completely preserved samples, are unjustified and, as will be demonstrated in the following sections, unnecessary for the majority of palaeodemographic analyses.

Life tables are based upon using the middle value within any cohort (eg. 25 years for 20–30 yrs). Given that age estimates within ten year cohorts actually mean an individual could lie at either end of the age range, it was decided that additional life tables should be constructed using both the lower age estimate and the upper age estimate. While giving a 'fuzzy' result, this also indicates outside limits to demographic parameters. This is preferable to arbitarily manipulating age categories up or down since methods of age estimation do not consistently either overage or underage all age groups.

Sexing

Sex was determined for adults only. Determination was based upon standard morphological characters rather than discriminant functions since there was not a sufficiently large core sample; many individuals were only represented by a single diagnostic bone, eg. the pelvis. Initial determination of sex of crania was based upon a core sample sexed by pelvic indicators.

Four measurements of demonstrated value in identifying sex were recorded (Bass 1981). These were femur head diameter, vertical and transverse diameters of the humeral head, and antero-posterior midshaft diameter of the femur. Measurements for males and females are significantly different, with very little overlap (Table 5.4). These figures were subsequently used to sex individuals unsexed by other means and as a double check on those sexed by crania alone. The most clearly discriminating measurement was femur head diameter which was preferentially used in all cases where the pelvis was not preserved.

Table 5.4
Sexual dimorphism according to humerus and femur head diameters

	Females	Males
Average femur head diameter	40.3	47.0
No.	64	61
Range	36–45	43–53
Average minimum humerus head diameter	36.9	40.8
No.	49	51
Range	33–43	38–48

Bias in sexing methods was tested for by comparing the sex distribution of the core group (sexed by pelvic indicators) and the peripheral (sexed by other means). No significant differences were found.

Recording methods and minimum numbers

After excavation the skeletal materials from both these sites were transferred to the Bahrain National Museum storage facilites where they were recorded. Categories of data collected were:

1. Archaeological location and data.
2. Age and sex.
3. Metrics.
4. Dental pathology.
5. Skeletal pathology.
6. Osteoarthritis.

Given the variation in preservation from complete skeletons to a few fragments of bone, it was not possible to obtain a complete record on all skeletons.

A major problem was the number of graves that contained more than one skeleton. At times these were easily identified: either the skeletons had been collected and bagged separately; or the grave contained an adult and subadult the bones of which could be easily distinguished. In other cases the bones were completely mixed and could not be separated.

In these cases all bones were recorded individually then the age and sex distributions based on crania compared with those based on innominates, femora and mandibles. The bones that could be associated were placed on the same record sheet. This results in the most complete demographic data for adults possible. In terms of palaeopathology, however, the bones which cannot be associated with particular individuals had to be treated separately.

The calculation of minimum numbers was more difficult when recording multiple infant graves since preservation of infant dental remains is different from that of infant long bones. In these samples there is a tendency for the long bones to be much better preserved than the dental remains. In order to cope with this problem, the minimum number of individuals was calculated for each bone. Results based on the right and left sides were then matched to obtain a single figure for each bone type for example the mandible or femur. Using growth standards based on the Bahrain material, an age distribution from the grave contents was constructed on the basis of the most numerous long bone. This was then compared to the age distribution based on dental data. If the two distributions corresponded then the most complete, generally either the femur or the mandible, was used. In the majority of cases the data fit was good. Those cases where it was not adequate were examined individually to determine reasons for the poor fit. The most common was that the youngest infants were represented by long bones only, in these cases the long bone length was accepted as the most accurate indicator of age.

As for adults, the resulting minimum numbers were recorded only in terms of archaeological location and age. Palaeopathological data from these graves is recorded in terms of individual bones only since it was not possible to identify complete individuals. Thus the analysis of pathology will be conducted on two levels:–aged and sexed individuals, and the number of bones (Appendix 2).

Palaeopathology recording

Palaeopathology was recorded for each skeleton possible using standardized categories. Not all of the data gathered has been used in the current study (dental disease, cortical bone area etc). Rather the concentration has been on those markers of disease and stress which occur amongst a significant proportion of the population, and which most clearly reflect conditions of life and death for the majority of the sample.

With the exception of childhood stress indicators (enamel hypoplasia and long bone growth) all pathological signs were

systematically recorded in one of the following four categories:

1. resorptive (porotic hyperostosis, osteoporosis);
2. proliferative (infectious);
3. traumatic; and
4. neoplasms (Powell 1988).

Naturally these categories are, to some extent, arbitrary since they are not necessarily mutually exclusive. Nevertheless, given further subdivisions within each class of lesion, relatively precise definitions resulted. These basic categories of lesions were also described in regards to location (Table 5.5).

Finally, where appropriate the status of the lesion at death was recorded as:

1. active;
2. partially remodelled–signs of some areas of healing and bone remodelling visible but still with active areas; and
3. totally healed with entire lesion remodelled.

Table 5.5
Recording of lesion location

Category	Long Bones	Vertebrae	Other
1	Proximal	Superior	<10%
2	Distal	Inferior	10 – 25%
3	Epiphyseal	Articular	25 – 50%
4	Shaft	Body	50 – 75%
5	All	All	75 – 100%

Analysis

The coded information was analysed by using SPSS-x (Nie 1986). Given the categorical nature of most of the data, the statistics have been kept as simple as possible.

The recording of palaeopathology rests upon visual examination although in a few cases x-rays were used as an aid in the diagnostic process. In addition, in the analysis of porotic hyperostosis and the hypertrophic condition, neutron activation analysis was conducted.

Step 1 concluded

The resultant records were used as the basis for all further analysis. The basic age and sex distribution forms the basis of life table analysis (Step 2), while the pathological and metrical data is used within the analysis of actual disease states.

The skeletal samples, like every other sample ever used, are not perfect. Certainly Step 1 served to identify several shortcomings. These are:

1. The possible under-enumeration of infants from Saar Mound 5.
2. The small size of the Mound 5 sample.
3. A need for caution regarding age estimation, particularly of the oldest age group.
4. Smaller sample sizes for palaeopathological analysis because of co-mingling of skeletal material.

As the following analysis demonstrates, each of these problems can be avoided by using a basically conservative approach as warranted by the data.

Chapter 6

Step 2: Palaeodemography

The analysis of demography follows several necessary steps, first of which is the necessary testing for bias due to under- or over-enumeration, then to determine the suitability of life table construction both with and without a growth rate. Further analysis includes separate calculation of male and female patterns of mortality, as well as subadult mortality. The additional parameters of a life table such as dependency and fertility rates, widowhood and orphanhood rates can then be estimated. Finally an estimate of fertility can be attempted.

Since DS3 is the larger sample it will be discussed first. As it is suspected that the Saar sample is biased, this group will be analysed using DS3 as a baseline comparison. Finally the results of the two samples will be collated in order to establish which sections of the age structure need to be explained by the analysis of pathology.

D.S.3

Age at Death

The total sample size for DS3 cemetery was 1,051 individuals. The majority could be aged and sexed. Of 631 subadults (less than 15 years) only 10 had to be partitioned across relevant age categories. Adult ageing, unfortunately, was less complete.

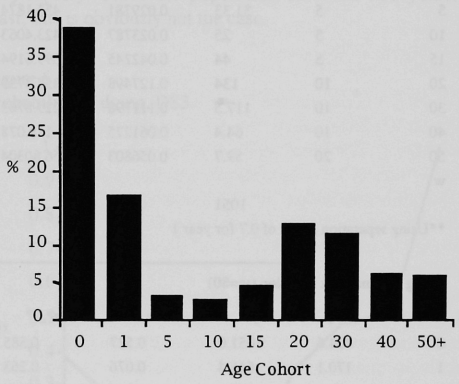
In order to assign individual adults to age cohorts the methodology outlined in Chapter 5 was applied. The adult sample was divided into several categories (total adults = 420):

1. Aged and sexed (95 males, 110 females)	48.8%
2. Aged but unsexed (11 individuals)	2.6%
3. Unaged but sexed (85 individuals)	20.2%
4. Unaged and unsexed (69 individuals)	16.4%
5. Aged by cranium and sexed (48 individuals)	11.4%
6. Aged by cranium and unsexed (2 individuals)	0.5%

These groups were apportioned, in turn, to a sex category where necessary (19.5% unsexed), and then to an age category based on the distribution of ages at death for that sex (36.7% had no evidence of age at all).

The resulting age at death distribution can be seen in Figure 6.1. Infant deaths were the most common followed by the 1–4, 20–29 and 30–39 age cohorts. The sex ratio of adults is close to one but this varies between different age groups indicating that it is necessary to consider the sexes separately at some stages of analysis.

Figure 6.1
Distribution of age at death, DS3



The distribution of ages at death follows the expected patterns of mortality: more deaths occur in the first year of life than at 5 years; between 30–70% of deaths occur before 15 years; and more deaths occur between 15–19 years then between 10–14 years (Buikstra and Mielke 1985). In order to check for a normal distribution of the adult ages, however, it is necessary to construct mortality curves from life table statistics.

Construction of a life table

As discussed in Chapter 2, the three assumptions of life table use are:

1. No demographic disruptions in the previous 50 years.
2. No net migration.
3. Constant birth and death rates over time (Weiss 1973).

Since the cemetery was used for a period between 300–500 years, it is expected that the possibility of demographic disruption (assumption 1) is nullified. This follows Weiss' work which demonstrates that long periods of accumulation negate disruptions (Weiss 1975).

Assumption 2 is more difficult to test and at this stage of the analysis remains an assumption. The probability is that the most common form of migration would be between villages for marriage. Trade at this time is more confined to local products and there is no evidence for mass immigration in the archaeological record. Whether in- and out-marriage would in fact balance each other will be examined later.

Table 6.1
Life table for DS3

Life Table (w=70)

x	N	nD_x	d_x	l_x	nq_x**	nL_x	T_x	e_x	C_x
0	1	404.6	0.384967	1000	0.384967	749.7716	15146.2	15.1462	4.950228
1	4	170.1	0.161846	615.0333	0.26315	2136.441	14396.43	23.40756	14.10546
5	5	31.33	0.029781	453.1874	0.065715	2191.484	12259.99	27.0528	14.46887
10	5	25	0.023787	423.4063	0.05618	2057.564	10068.51	23.77978	13.58469
15	5	44	0.042245	399.6194	0.105714	1892.483	8010.942	20.04643	12.49477
20	10	134	0.127498	357.3739	0.356763	2936.251	6118.459	17.12061	19.38605
30	10	117.5	0.111798	229.8763	0.486341	1739.772	3182.207	13.84313	11.48652
40	10	64.4	0.061275	118.078	0.518936	874.4053	1442.436	12.21595	5.773099
50	20	59.7	0.056803	56.80304	1	568.0304	568.0304	10	3.750316
w								{15}	
		1051	1			15146.2			100

**Using separation factor of 0.7 for year 1

Using a Gompertz function (w=80)

x	nD_x	l_x	nm_x	nq_x**	nL_x	T_x	e_x	log (nm_x)	C_x
0	404.6	1051.0	0.527	0.385	767.8	15903.7	15.1		5.084768
1	170.1	646.4	0.076	0.263	2245.4	15135.9	23.4		14.8702
5	31.3	476.3	0.014	0.066	2303.2	12890.5	27.1		9.842735
10	25.0	445.0	0.012	0.056	2162.5	10587.2	23.8		7.979705
15	44.4	420.0	0.022	0.106	1989.0	8424.7	20.1		8.357143
20	134.0	375.6	0.043	0.357	3086.0	6435.7	17.1	-3.13679	15.35323
30	117.5	241.6	0.064	0.486	1828.5	3349.7	13.9	-2.74481	10.69298
40	64.4	124.1	0.070	0.519	919.0	1521.2	12.3	-2.65817	6.611511
50	36.8	59.7	0.089	0.616	413.1	602.2	10.1		3.358537
60	16.6	22.9	0.113	0.722	146.4	189.1	8.3		1.449505
70	5.3	6.4	0.144	0.836	37.0	42.7	6.7		0.445783
80	1.0	1.0	0.183	1	5.5	5.7	5.4		0.08209
w							-0.2393		84.12819

Life Table (w=80)

x	N	nDx	d_x	l_x	nq_x**	nL_x	T_x	e_x	C_x
0	1	404.6	0.384967	1000	0.384967	749.7716	15430.22	15.43022	4.859112
1	4	170.1	0.161846	615.0333	0.26315	2136.441	14680.45	23.86935	13.84583
5	5	31.3	0.029781	453.1874	0.065715	2191.484	12544.01	27.67951	14.20255
10	5	25.0	0.023787	423.4063	0.05618	2057.564	10352.52	24.45056	13.33464
15	5	44.4	0.042245	399.6194	0.105714	1892.483	8294.957	20.75714	12.26479
20	10	134.0	0.127498	357.3739	0.356763	2936.251	6402.474	17.91534	19.02923
30	10	117.5	0.111798	229.8763	0.486341	1739.772	3466.223	15.07864	11.27509
40	10	64.4	0.061275	118.078	0.518936	874.4053	1726.451	14.62127	5.666837
50	30	59.7	0.056803	56.80304	1	852.0457	852.0457	15	5.521929
w								{15}	
		1051	1			15430.22			100

Birth and death rates were probably not constant over time but, rather, would have fluctuated cyclically thereby following a consistent, although underlying, trend. It is expected that the length of time of accumulation and completeness of the sample would negate any fluctuations and allow accurate estimation of the underlying rates. The possibility of unbalanced migration, and systematic disruptions such as epidemics, will be discussed in relation to the findings, initially based on a stationary growth rate and then an estimated growth rate.

The resultant life table can be seen in Table 6.1. Since the age classes are truncated to 50 years, two possible maximum life spans were estimated: 70 and 80 years. This affects life

expectancy figures and the proportion living in the population. The differences are only slight.

An alternative method is to extrapolate life expectancies for older ages using the Gompertz model whereby the average decrement in the mortality rate for adult age cohorts under 50 years is calculated and used for higher ages by loglinear extrapolation. This has no effect on earlier ages but possibly mimics the actual demographic characteristics for old adults.

Firstly, an examination of the mortality curve (Figure 6.2) shows the typical 'U' shaped pattern characteristic of pre-industrial populations: mortality steadily declining from infancy to reach a low between 10–15 years, increasing slightly in the 20s then steadily increasing through later adult years. This suggests that the data represents a normal population distribution (Waldron 1994).

The expectation of life at birth is only 15.1 years which is extremely low, although not exceptional for skeletal samples (Weiss 1973). It is the result of two factors: high infant mortality and high mortality among the young adult age groups. Expectation of life increases dramatically after the first year. People who reached the age of 5 could expect to live until 32.1 years, those who reached 15 could expect to live until 35.1 years of age.

Based on the assumption of a stationary rate of growth, the Crude Birth Rate of this population was 0.066 births/person/year, equivalent to the death rate. Thus the population would seem to fall into the category proposed by Howell of high fertility/high mortality populations (Howell 1976). To some extent this is misleading since the value of fertility and mortality has to be balanced under the current assumption of a stationary growth rate. If, however, we compare these statistics with the stationary model populations tabled by Weiss (1973) the meaning is clear. Given the high proportion of deaths at young age groups, unless fertility rates were high the population would decline very rapidly. Since the population survived 300 years, at least, this is obviously not the case.

Figure 6.2
Probability of dying, DS3

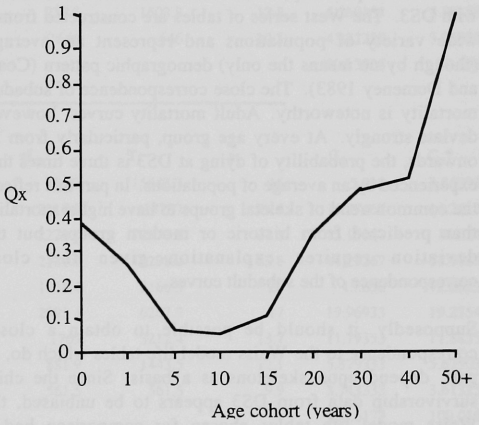
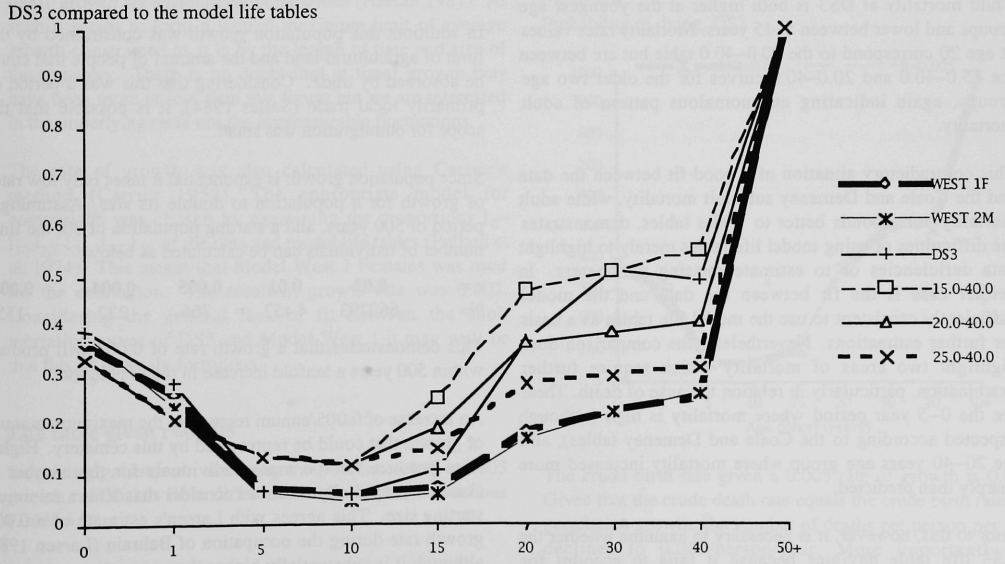


Figure 6.3
DS3 compared to the model life tables



Model life table comparison

Comparison with both the Weiss (1973) and Coale and Demeney Life Tables (Coale and Demeney 1983) highlights two unusual points. The mortality curves were used as the initial basis for comparison after adjusting the model life tables for wider age intervals. It must be emphasised though that the aim of this comparison is not to suggest that the curve of mortality should be identical to that of the model life tables but rather to highlight areas where the DS3 population deviates from the 'norm'.

Figure 6.3 compares the Coale and Demeney tables with the lowest life expectancy, West 1 Females and West 2 males, with DS3. The West series of tables are constructed from a wide variety of populations and represent an 'average' (though by no means the only) demographic pattern (Coale and Demeney 1983). The close correspondence of subadult mortality is noteworthy. Adult mortality curves, however, deviate strongly. At every age group, particularly from 20 onwards, the probability of dying at DS3 is three times that experienced by an average of populations. In part this reflects the common trend of skeletal groups to have higher mortality than predicted from historic or modern groups, but the deviation requires explanation given the close correspondence of the subadult curves.

Supposedly, it should be possible to obtain a closer correspondence to the Weiss model life tables which do, in part, depend upon skeletons as a basis. Since the child survivorship data from DS3 appears to be unbiased, the Weiss model life tables chosen for comparison had a survivorship at 15 years of 40 and varying life expenctancy at birth of 15, 20 and 25 years. There is relatively little comparison between these curves and DS3 (Figure 6.3). Child mortality at DS3 is both higher at the youngest age groups and lower between 5–15 years. Mortality rates values at age 20 correspond to the 20.0–40.0 table but are between the 15.0–40.0 and 20.0–40.0 curves for the older two age groups, again indicating an anomalous pattern of adult mortality.

This contradictory situation of a good fit between the data and the Coale and Demeney subadult mortality, while adult mortality corresponds better to Weiss tables, demonstrates the difficulties of using model life tables merely to highlight data deficiencies or to estimate missing parameters. In neither case is the fit between the data and the model sufficiently consistent to use the model life tables as a basis for further estimations. Nevertheless, the comparison does highlight two areas of mortality which require further examination, particularly in relation to cause of death. These are the 0–5 year period where mortality is high (although expected according to the Coale and Demeney tables), and the 20–40 years age group where mortality increased more sharply than predicted.

Prior to this, however, it is necessary to examine whether the DS3 life table deviates because it fails to account for population growth or because it incorporates systematic bias in adult ages.

Estimation of population growth

By examining the archaeological evidence it is possible to estimate limits on the rate of population growth. In relation to DS3 a series of limits emerge. The associated burying population used the cemetery for between 300–500 years during which some 1280 people were buried in the cemetery. It is possible that later use of the cemetery also occurred although this is probably minor. There appears to be an associated small settlement nearby according to survey data which may be the source of the bodies. There is no evidence to suggest declining use of the cemetery over time, rather the peak period of usage appears to have been between c 100 BC–100 AD though this is dependent on the accuracy of chronological estimates from pottery. Based on the above, average settlement size was about 100.

From this it can be suggested that steady population decline from the beginning of cemetery use is unlikely. It is possible, however, population growth occurred during the period of use, though this may have been more marked in the earlier part of the period covered by the cemetery, and matched by a corresponding decline towards the end.

The minimum starting size of the population, if the population was growing, can be estimated at c30 individuals. This is for two reasons. Firstly, cemetery use was already established thereby requiring a minimum population. Secondly, c100 persons is the estimated average size of the contributing population therefore both the minimum and maximum population sizes are limited by this figure.

In addition, this population growth was constrained by the limit of agricultural land and the amount of people that could be absorbed by trade. Considering that this was a period of primarily local trade (Salles 1984), it is possible that the scope for outmigration was small.

Since population growth is exponential it takes only low rates of growth for a population to double its size. Assuming a period of 500 years, and a starting population of 30, the final number of individuals can be calculated as below:

r =	0.02	0.01	0.005	0.004	0.003
N=	660793	4 452	366	222	135

This demonstrates that a growth rate of 0.005 will produce within 500 years a tenfold increase in the population.

An increase of 0.005/annum represents the maximum amount of growth that could be represented by this cemetery. Higher rates produce far too many individuals for the number of skeletons, especially when we consider that 30 is a minimum starting size. This agrees with Larsen's estimate of a 0.005 growth rate during the occupation of Bahrain (Larsen 1983) although it is substantially higher than an earlier estimate of

Table 6.2
DS3 life table with population growth

Life Tabe (r=0.005); w=80)

X	N	nD_x	l_x*	nq_x**	nL_x	T_x	e_x	C_x	C_x*
0	1	404.6	1066	0.38	780.2	16277.7	15.3	5.099346	4.792619
1	4	170.1	660.4	0.26	2275.3	15497.5	23.5	14.87124	13.97673
5	5	31.3	488.6	0.065	2334.5	1322.3	27.1	15.25817	14.34039
10	5	25	456.9	0.055	2193.8	10887.7	23.8	14.33856	13.47609
15	5	44.4	431.6	0.104	2020.3	8694	20.1	13.20458	12.41032
20	10	134	386.7	0.355	3179.6	6673.7	17.3	20.7817	19.53167
30	10	117.5	249.3	0.483	1890.3	3494.1	14	12.3549	11.61175
40	10	64.4	128.8	0.513	957.8	1603.8	12.5	6.260131	5.883582
50	30	59.7	62.8	1	646	646	10.3	4.222222	3.968254
		1051						106.3908	99.9914
		d=0.065		b=0.070					

Life Table (r=-0.005)

X	N	nD_x	l_x*	nq_x**	nL_x	T_x	e_x	C_x	C_x*
0	1	404.6	1036.3	0.389	755.7	15542.7	15	5.038	4.862934
1	4	170.1	632.7	0.266	2216.2	14787	23.4	14.77467	14.26126
5	5	31.3	464.3	0.067	2272.6	12570.8	27.1	15.15067	14.6242
10	5	25	433.4	0.057	2131.9	10298.2	23.8	14.21267	13.71879
15	5	44.4	408.7	0.107	1958.4	8166.3	20	13.056	12.60232
20	10	134	364.9	0.358	2995.4	6207.9	17	19.96933	19.27542
30	10	117.5	234.2	0.489	1769	3212.4	13.7	11.79333	11.38353
40	10	64.4	119.6	0.525	881.9	1443.5	12.1	5.879333	5.675032
50	30	59.7	56.8	1	561.5	561.5	9.9	3.743333	3.613256
		1051						103.6173	100.0167
	d=0.068	b=0.063							

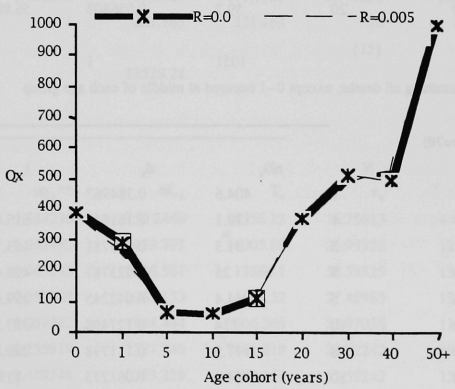
0.001 growth for agricultural populations (Hassan 1981). As such it seems to form a natural maximum limit of average growth constrained as it is by the length of time and size of the cemetery. There is no doubt that at times growth may have been more or less than this figure but we are interested in the underlying rates not the immeasurable fluctuations.

The rate of growth was also calculated using Carrier's method (Carrier 1958). The appropriate model for comparison was chosen by examining the proportions l_0–l_{10},l_{20}–l_{30} ,l_{10}–l_{20} of the data and model life tables (Pollard et al. 1974). This meant that Model West 1 Females was used for the estimation. The resultant growth rate was 0.002. Considering the general lack of fit between the adult mortality curves of DS3 and Model West 1 it may well be that this fit is purely fortuitous.

Life table given r=0.005

Surprisingly, however, assuming the population had a 0.005 growth rate makes little difference in the life table functions (Table 6.2).

Figure 6.4
Probability of dying, DS3



The crude birth rate given a 0.005 rate of growth is 0.070. Given that the crude death rate equals the crude birth rate less population growth, the number of deaths per person per year declines to 0.065/person/year. More importantly the probability of dying is slightly less at all ages. The

difference, however, is never more than 0.020. Figure 6.4 shows how little real change the growth rate makes.

The two most important functions with respect to social composition are life expectancy and population distribution. If the growth rate was 0.005, life expectancy at birth increases by 0.3 years, and not at all at age 15. Similarly the age distribution of the living population shows little change. Basically there are more individuals in older age classes but the difference is never more than one percent or one

Table 6.3
DS3 life tables using age limits

(w=70)

x	N	nD_x	d_x	l_x	nq_x**	nL_x	T_x	e_x	C_x
0	1	404.6	0.384967	1000	0.384967	749.7716	13075.32	13.07532	5.734251
1	4	170.1	0.161846	615.0333	0.26315	2136.441	12325.55	20.04045	16.3395
5	5	31.3	0.029781	453.1874	0.065715	2191.484	10189.11	22.4832	16.76047
10	5	25	0.023787	423.4063	0.05618	2057.564	7997.621	18.88876	15.73625
15	5	44.4	0.042245	399.6194	0.105714	1892.483	5940.057	14.86429	14.47371
20	10	134	0.127498	357.3739	0.356763	2298.763	4047.574	11.32588	17.58093
30	10	117.5	0.111798	229.8763	0.486341	1180.78	1748.811	7.607616	9.030604
40	10	64.4	0.061275	118.078	0.518936	568.0304	568.0304	4.810637	4.344295
50	20	59.7	0.056803	56.80304	1	0	0	0	0
w									
		1051	1			13075.32			100

Assuming all deaths above 20 years occur at lower limit of each age group

(w=70)

x	N	nD_x	d_x	l_x	nq_x**	nL_x	T_x	e_x	C_x
0	1	404.6	0.384967	1000	0.384967	749.7716	15146.2	15.1462	4.950228
1	4	170.1	0.161846	615.0333	0.26315	2136.441	14396.43	23.40756	14.10546
5	5	31.3	0.029781	453.1874	0.065715	2191.484	12259.99	27.0528	14.46887
10	5	25	0.023787	423.4063	0.05618	2057.564	10068.51	23.77978	13.58469
15	5	44.4	0.042245	399.6194	0.105714	1892.483	8010.942	20.04643	12.49477
20	10	134	0.127498	357.3739	0.356763	2936.251	6118.459	17.12061	19.38605
30	10	117.5	0.111798	229.8763	0.486341	1739.772	3182.207	13.84313	11.48652
40	10	64.4	0.061275	118.078	0.518936	874.4053	1442.436	12.21595	5.773099
50	20	59.7	0.056803	56.80304	1	568.0304	568.0304	10	3.750316
w								{15}	
		1051	1			15146.2			100

Assuming all deaths, except 0–1 occured at middle of each age group

(w=70)

x	N	nD_x	d_x	l_x	nq_x**	nL_x	T_x	e_x	C_x
0	1	404.6	0.384967	1000	0.384967	749.7716	17217.09	17.21709	4.354811
1	4	170.1	0.161846	615.0333	0.26315	2136.441	16467.32	26.77468	12.40884
5	5	31.3	0.029781	453.1874	0.065715	2191.484	14330.88	31.6224	12.72854
10	5	25	0.023787	423.4063	0.05618	2057.564	12139.39	28.67079	11.95071
15	5	44.4	0.042245	399.6194	0.105714	1892.483	10081.83	25.22857	10.99189
20	10	134	0.127498	357.3739	0.356763	3573.739	8189.343	22.91534	20.75693
30	10	117.5	0.111798	229.8763	0.486341	2298.763	4615.604	20.07864	13.35164
40	10	64.4	0.061275	118.078	0.518936	1180.78	2316.841	19.62127	6.858187
50	20	59.7	0.056803	56.80304	1	1136.061	1136.061	20	6.59845
w								{15}	
		1051	1			17217.09			100

Assuming all deaths occured at upper limit of each age group above 20

individual in each age group.

Therefore it makes little difference to any subsequent analysis of the DS3 population structure whether the population was growing, on average, to its maximum possible or stationary. It must be emphasized though that this is an average. It is expected that in fact population growth fluctuated around zero to 0.004 falling outside these limits for short periods of time. Given that the cemetery appears to indicate a slight increase in use over time and then a slight decline, it may be seen that a stationary growth rate provides a reasonable estimation. The effect of altering age estimates is of more significance to the demographic parameters.

Life tables using age limits

In order to examine the effect of differences in ageing, the life table was recalculated using the lower and upper end of each age cohort. This means that, rather than calculating life table functions as if all deaths occurred across the age cohort, the same functions were calculated as if all deaths occurred at the lowest age possible (i.e. 20 yrs for the 20–30 cohort) and again at the highest possible age (i.e. 29.9 yrs for 20–30 cohort). Since the age estimates are broad, this helps to establish demographic limits, as well as mimicking the effect of a systematic bias in the methods of age estimation (G. Santow pers. com.). Not all life table parameters are affected: only those that are cumulative such as L_x (the number of years to live within a cohort), T_x (total number of years lived), and, most importantly, life expectancy and C_x (the proportion in a living population). In order to mimic these effects for the oldest age group, the three tables were calculated using 70 as the maximum age. The results are presented in Table 6.3.

The difference in life expectancy at birth is approximately two years using a lower estimation, so that the lower limit becomes 13.1 yrs. At 15 years, however, the difference has accumulated; an individual reaching this age could only be expected to live until 29.9 years of age, compared to 35.5 years. The difference age estimation makes is more substantial when an upper limit is used. At birth there is little difference, life expectancy increased only from 15.2 yrs to 17.2 yrs. By 15 years of age, it has increased from 20.5 years

Table 6.4
Sex-specific life tables, DS3

FEMALE LIFE TABLE (w=70)

x	N	nD_x	d_x	l_x	nq_x**	nL_x	T_x	e_x	C_x
0	1	202.3	0.385701	1000	0.385701	749.2946	13529.28	13.52928	5.53832
1	4	85.1	0.16225	614.2993	0.264122	2132.698	12779.98	20.80416	15.76358
5	5	15.6	0.029743	452.0496	0.065795	2185.891	10647.28	23.55335	16.15675
10	5	12.5	0.023832	422.307	0.056433	2051.954	8461.392	20.03612	15.16677
15	5	31.9	0.06082	398.4747	0.152632	1840.324	6409.438	16.08493	13.60253
20	10	83.2	0.158627	337.6549	0.469791	2583.413	4569.113	13.5319	19.09498
30	10	54.1	0.103146	179.0276	0.576145	1274.547	1985.701	11.09159	9.420661
40	10	28.2	0.053765	75.88179	0.708543	489.9905	711.1535	9.371859	3.621705
50	20	11.6	0.022116	22.1163	1	221.163	221.163	10	1.6347
w								{15}	
		524.5	1			13529.28			100

Assuming all deaths, except 0–1 occured at middle of each age group

MALE LIFE TABLE (w=70)

x	N	nD_x	d_x	l_x	nq_x**	nL_x	T_x	e_x	C_x
0	1	202.3	0.384236	1000	0.384236	750.2469	16756.13	16.75613	4.477446
1	4	85.1	0.161633	615.7645	0.262492	2139.791	16005.89	25.99352	12.77019
5	5	15.6	0.02963	454.1311	0.065245	2196.581	13866.1	30.53325	13.10912
10	5	12.5	0.023742	424.5014	0.055928	2063.153	11669.52	27.48993	12.31282
15	5	12.5	0.023742	400.7597	0.059242	1944.444	9606.363	23.97038	11.60437
20	10	50.8	0.096486	377.018	0.255919	3287.749	7661.918	20.32242	19.62117
30	10	63.4	0.120418	280.5318	0.429248	2203.229	4374.169	15.59242	13.14879
40	10	36.2	0.068756	160.114	0.429419	1257.36	2170.94	13.55872	7.503878
50	20	48.1	0.091358	91.35802	1	913.5802	913.5802	10	5.452214
w								{15}	
		526.5	1			16756.13			100

Assuming all deaths, except 0–1 occured at middle of each age group

to 25.2 years (i.e. an expected life span from 35.5 years to 40.2 years), and those who survived until 30 years of age (the point of maximum divergence) could be expected to live until 50.1 years of age rather than 44.5 years. Altering age estimates has little effect overall on the demographic parameters, primarily because the shape of the population curve is dominated by the high levels of subadult mortality.

Differences do occur in the composition of the living population implied by these sets of figures. Using the middle estimate, 47.2% of the population was less than 15 years of age. This changes to 54.3% using a lower estimate and to 41.5% using an upper limit. In a population of 100, this means that if the upper limit is closer to reality there were 58 adults (15+ yrs) looking after 42 subadults, as opposed to 53 looking after 47. The lower estimate implies 46 adults caring for 54 children. This is a very high ratio of dependents, and considering that most criticisms of ageing methods suggest that under-ageing not over-ageing occurs, the lower estimates will be ignored. The middle and upper estimates will be used where feasible in the following discussion since they set possible limits to the demographic parameters.

The resultant Crude Birth Rate which is a useful figure to compare between models is 0.77 for the lowest estimate and 0.58 for the highest. Again the difference is significant, although not sizeable. This will become clearer as the social composition implied by the life table is discussed.

Males vs females

Significant differences existed between the mortality experience of males and females. In order to explore this a life table was constructed for each sex (see Table 6.4). For this it has been necessary to assume equal mortality between sexes during childhood ages. This is an unreasonable assumption but a necessary starting point given the limitations of skeletal sexing of subadults. To some extent the proposition may be tested at a later stage through comparison of the frequency of stress indicators.

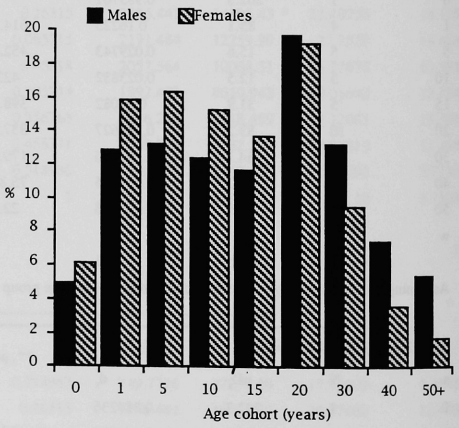
The differences between male and female adult mortality are immediately obvious. The crude indicator, life expectancy, indicates that at birth females could expect to live 13.5 years (range 11.7–15.3 years), males 16.8 years (14.4–19.1 years). Comparing the mortality rates female probabilities of dying were greater at every age from 15 years on. The disparity is most marked both at 20–29 years and at 40–49 years.

The resulting population structures are also quite different for males and females (Figure 6.5). As a percentage of the total population, there are substantially fewer females in the older age groups than males. Males predominate from age 30 onwards. It should be noted here that if this sex-specific mortality originated during childhood then the disparity would be even more marked.

For females the probability of dying increases during the 15–20 year age group, climbs most sharply between 20–30 years, and continues to increase during 30–50 years. Male probability of dying on the other hand does not increase until the 20's when it begins to increase steadily to 30–40 years. At this point it levels off. The difference suggests two completely different cause of death patterns.

One possible reason for apparent low male mortality could be that out-migration of males was occurring. While not discounting this possibility, there are two reasons to doubt that such migration had a significant demographic impact. Firstly male mortality, whilst lower than female, is lower at all ages (not merely those traditionally affected by emigration). The two curves map each other at younger adult ages–real disparity in trends does not occur until 40–50 years, beyond the years generally affected by unbalanced migration. Secondly, the structure of the living population implied by these life tables indicates an equal number of males and females between 20 and 30 years rather than an obvious shortfall. This, however, does not disprove the possibility that young males may have left the village temporarily, merely that they (or replacements if migration was balanced) were probably buried within the DS3 cemetery.

Figure 6.5
Sex-specific population structure, DS3



Comparing the male mortality curves to Coale and Demeney tables demonstrates the same excess of young adult mortality seen in the combined sexes comparison (Figure 6.6). The difference, however, is less marked, and evident only from 20 years onwards. The shape of male adult mortality when compared to the Weiss tables (Figure 6.6) deviates from the average mortality curves. Expected mortality is substantially less through the subadult years. There is also none of the levelling off of mortality between 20–40 years seen in the Weiss tables. Rather between 20–30 years male probability of dying, given the life expectancy at age 15, is less than

Figure 6.6
Comparison of male probability of dying

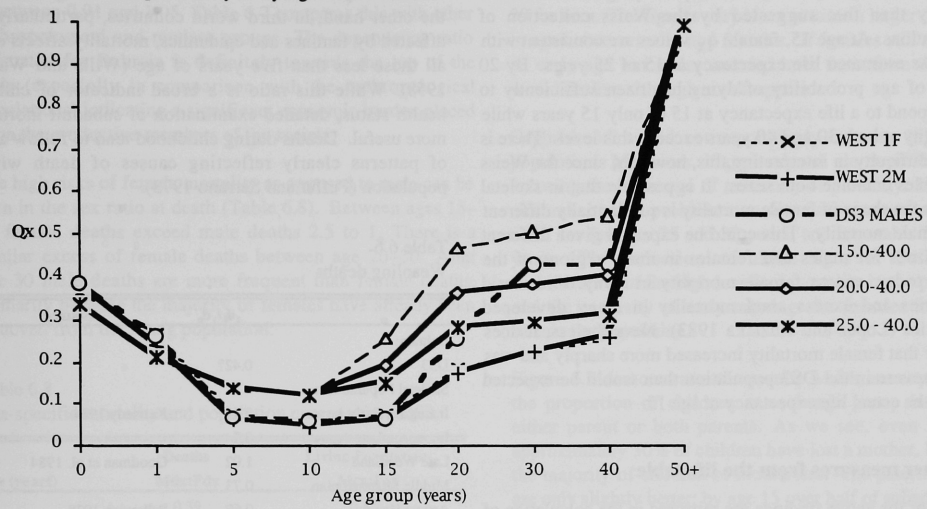
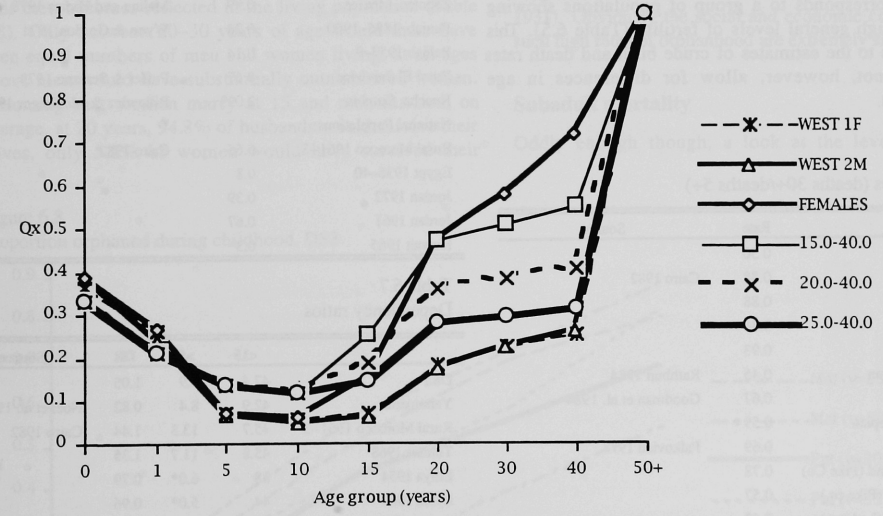


Figure 6.7
Comparison of female probability of dying



expected. It does increase to expected levels between 30–40 years. In terms of male mortality there are two deviations which need to be explored: the later onset of increased mortality, and the low male mortality relative to other skeletal groups between 20–30 years.

Predictably female adult mortality is differs from the model life tables (Figure 6.7). At every age from 15 years on, the female probability of death is higher than that established by the corresponding Coale and Demeney tables. This difference is least obvious at age 15, but at all ages above the probability of death among DS3 females was over two times greater than the Coale and Demeney West tables. The difference, however, is more one of scale than actual difference in the shape of mortality.

The differences from the Weiss tables are more unexpected (Figure 6.7). The female probability of dying increases more steeply than that suggested by the Weiss collection of populations. At age 15, female q_x values are consistent with a Weiss estimated life expectancy at 15 of 25 years. By 20 years of age probability of dying has risen sufficiently to correspond to a life expectancy at 15 of only 15 years while mortality at both 30 and 40 years exceeds this level. There is some difficulty in interpreting this, however, since the Weiss life tables combine both sexes. It is possible that in skeletal groups the shape of female mortality is substantially different from male mortality. This could be expected given different mortalities for males and females in many regions of the world today: excess female mortality in many third world countries and excess amel mortality in many developed countries (Lopez and Ruzicka 1983). Nevertheless, it does appear that female mortality increased more sharply and was more severe in the DS3 population than would be expected given the actual life expectancy at age 15.

Further measures from the life table

Many of the above findings are mirrored in the calculation of ratios. The ratio of deaths age 30+ to deaths age 5+ is indicative of relative fertility. A low ratio tends to reflect high fertility rates, a high ratio correspondingly low fertility (Buikstra et al. 1986). The ratio of deaths in the DS3 sample is 0.5. A list of this ratio in other populations demonstrates that DS3 corresponds to a group of populations showing moderate–high general levels of fertility (Table 6.5). This corresponds to the estimates of crude birth and death rates which do not, however, allow for differences in age distributions.

Table 6.5
Fertility rates (deaths 30+/deaths 5+)

Population	Rate	Source
DS3	0.50	
Egypt 1936–40	0.88	Cairo 1982
Jordan 1961	0.88	
Jordan 1972	0.86	
Syria 1965–70	0.93	
Dinkha Tepe, Iran	0.45	Rathbun 1984
Late Woodland	0.67	Goodman et al. 1984
Middle Missisippian	0.59	
Arroyo Hondo	0.69	Palkovich 1978
Middle Woodland (Pike Co)	0.78	
Late Woodland (Pike co.)	0.57	
Late Woodland (Ledders)	0.48	
Schild Miss.	0.41	

High mortality is particularly obvious amongst young age groups. Examining weaning deaths in comparison to infant deaths, the ratio is 0.42. Compared to both modern and prehistoric groups this ratio is in the middle of the range (Table 6.6). In modern western populations most deaths under five years occur in the neonatal period reflecting the intractability of deaths due to perinatal causes and the massive improvements in health care for all ages above. On the other hand, in third world countries, particularly those affected by famines and epidemics, mortality affects equally all those less than five years of age (Wills and Waterlow 1958). While this ratio is a broad indicator of childhood health status, detailed examination of subadult mortality is more useful. Deaths during childhood tend to follow a series of patterns clearly reflecting causes of death within a population (Puffer and Serrano 1973).

Table 6.6
Weanling deaths

	${}_4d_1/{}_1d_0$	Source
Bahrain:		
DS3	0.42?	
Skeletal Populations:		
Inamgaon Early Jarwe	0.41	Kennedy 1984
Inamgaon Late Jarwe	0.2	
Late Woodland	1.02	Goodman et al. 1984
Middle Misisssippian	0.73	
Arroyo Hondo	0.69	Palkovich 1978
Small Populations:		
Yanomana Males est	0.37	Neel et al. 1977
Yanomana Females est	0.16	
Matlab, Bangladesh	0.58	Chen et al. 1980
Guyana 1937–46	0.38	Giglioli 1972
Zapotec, Mexico	0.99	Malina and Himes 1978
Punjab, 1886–1900	0.28	Wyon & Gordon 1971
Punjab 1957–9	0.14	
Rural El Salvador	0.67	Puffer & Serrano 1973
Keneba, Gambia	2.09?	Billewicz & McGregor 1981
National Populations:		
Rural Morocco 1961–3	0.65	Cairo 1982
Egypt 1936–40	0.8	
Jordan 1972	0.39	
Jordan 1961	0.67	
Kuwait 1965	0.3	

Table 6.7
Dependency ratios

	<15	>50	DR	Source
DS3	47.4	3.9	1.05	
Yanomama	42.9	8.4	0.82	Neel et al. 1977
Rural Morocco 1961–3	45.7	13.3	1.44	Cairo 1982
Tunisia 1968	45.8	11.7	1.35	
Libya 1954	38	6.0*	0.79	
Lybia 1964	44	5.0*	0.96	
Lybia 1973	49	4.0*	1.13	
Jordan 1961	45.6	3.8*	0.98	
Kuwait 1965	49	5.0**	1.17	

* Using 65 yrs; ** Using 60 yrs

High fertility and high mortality is also reflected in the dependency ratio which measures the proportion of the population that is economically dependent. In the present

group, since the age categories are truncated to 50 year of age, this age was used for the cutoff for old adults. The ratio is between 0.91 and 1.05. Table 6.7 compares this with other anthropological and modern groups. The dependency ratio estimated for Bahrain is definitely towards the top of the table (especially in comparison with the anthropological populations) indicating a significant economic burden placed upon the productive members of the society.

The high risks of female mortality as opposed to male can be seen in the sex ratio at death (Table 6.8). Between ages 15–20 female deaths exceed male deaths 2.5 to 1. There is a similar excess of female deaths between age 20–30. After age 30 male deaths are more frequent than female deaths, primarily because the majority of females have already been removed from the living population.

Table 6.8
Sex-specific mortality and population composition, DS3

	Deaths	Living Population
Age (years)	Mdx:Fdx	Mcx:Fcx
15	0.39	0.85
20	0.61	1.03
30	1.17	1.39
40	1.28	2.06
50+	4.14	3.38

This then becomes reflected in the living population (Table 6.8). Only between 20–30 years of age would there have been equal numbers of men and women living, at all ages above men would have substantially outnumbered women. Assuming that women marry at 15 and men married, on average, at 20 years, 94.8% of husbands would survive their wives, only 5.2% of women would have survived their husbands. The mean age of widowerhood for these men would be 36 years of age if the average age at marriage was 20 for men. The figures vary if we assume a larger difference in age between males and females. If females still married at an early age but males married later then these disparities would decline although not disappear.

Orphanhood would correspondingly be very high in the population. Measures of orphanhood are dependent upon knowing the mean age of maternity or paternity. In order to circumvent this problem two alternative mean ages were used: for females 25 and 30 years; and for males 30 and 35 years. The selection of these ages is based upon consideration of the high levels of mortality in the population and the necessity of early marriage for females in order to sustain the population.

Figure 6.8 demonstrates the results which are expressed as the proportion of children at each age group having lost either parent or both parents. As we see, even by age 5 approximately 30% of children have lost a mother, by age 15 the majority of children of motherless. The paternal figures are only slightly better: by age 15 over half of subadults were fatherless. This translates into approximately one-third of the population being absolute orphans by age 15. In fact these figures are minima since they fail to take account of puerperal deaths and posthumous orphans (percentages of orphans could never start at 0 from birth), as well as the correlation between the deaths of husbands and wives (Lotka 1931). Obviously the social and economic consequences of such high rates of orphanhood are extremely significant.

Subadult mortality

Oddly enough though, a look at the level of subadult

Figure 6.8
Proportion orphaned during childhood, DS3

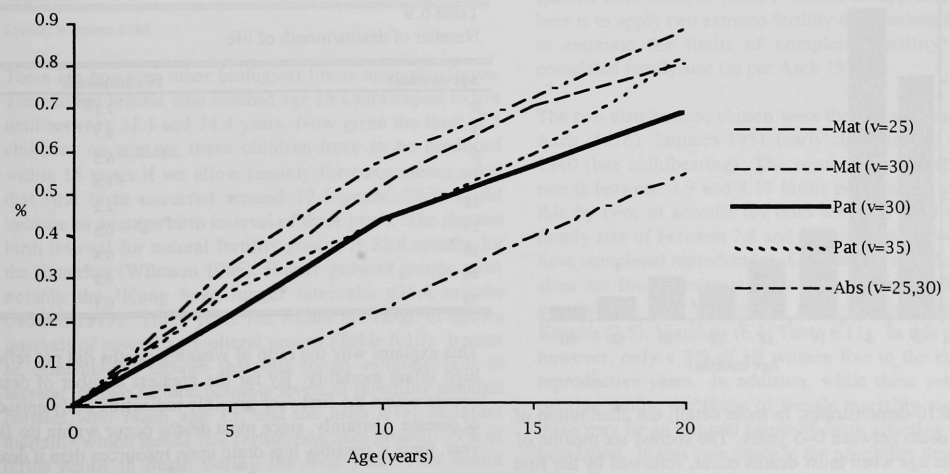
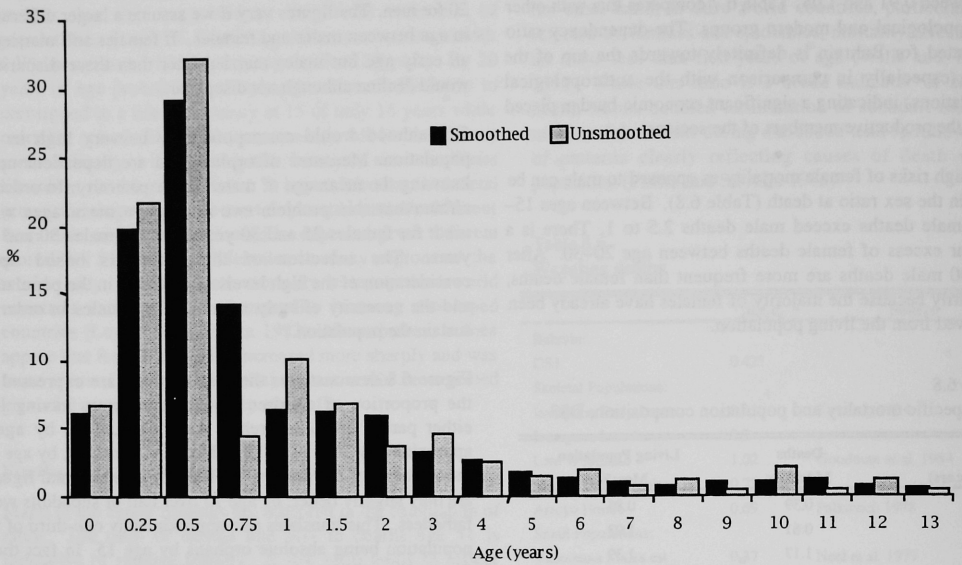


Figure 6.9
Smoothed vs unsmoothed age at death, DS3



mortality indicates that the situation may not have been as bad as it first appears. Figure 6.9 shows the distribution of ages at death from 0 to 13 years. The two columns demonstrate the effect of smoothing which was carried out to minimise the effects of age lumping at 0.5, 1, 3, 6 and 10 years. Clearly the majority of deaths occur within the first two years of life, especially the first. Substantially fewer deaths occurred between 2–3 years, while once a child reached 3 years, death became increasingly unlikely with age.

Figure 6.10
Subadult age at death, 0–5 years

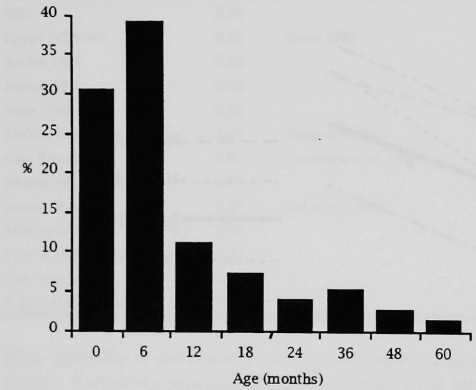


Figure 6.10 demonstrates, in more detail, the distribution of ages at death between 0–5 years. The second six months of life is the age when most deaths occur, followed by the first six months. After one year there is a steady decline in deaths to 5 years.

A clearer way to examine this is to calculate the number of deaths/month of life in each cohort (using 100 deaths between 0–5 yrs as the baseline (Table 6.9). This clearly demonstrates that the majority of deaths occurred in the first month of life. Deaths then declined until between 6–12 months when they again increase to nearly the same levels as perinatal deaths. By age 1 there is a marked drop in the number of deaths which becomes even lower by 2 years of age.

Table 6.9
Number of deaths/month of life

Age (months)	Deaths/month
0	7.1
3	4.7
6	6.5
12	1.8
18	1.2
24	0.3
36	0.4
48	0.2
30	0.1

This explains why the ratio of weaning deaths did not reflect high infant mortality. By far the greatest number of deaths occurs within the first year possibly indicating early episodes of disease. Certainly, since most deaths occur within the first year, there is possibly less drain upon resources than if deaths

had remained at a slightly lower but more constant level throughout childhood. The question which is then raised is what level of morbidity did the population sustain after this first year of life and what pattern of disease and other causes of death created this particular pattern?

Fertility

The number of children actually born is the final parameter to be estimated for this population. Fertility is measured by two general measures: gross reproduction rate (GRR: based on age-specific fertility rates) and net reproduction (NRR: based on both age specific fertility and mortality). The child/woman ratio plus the ratio of deaths 30+/deaths 5+ indicates a high general fertility in this population. This, however, is the only specific information we have on fertility.

In the stationary situation NRR must equal one since deaths equal births. Using this supposition we can calculate the number of live births a female must have in order to replace her entire birth cohort. For the DS3 population this means 5 children for each woman. Note that this is merely the level of fertility specified by a zero growth rate and the levels of mortality in the population. It was basically necessary for each woman on average to have 5 children so that two could survive to age 15 and begin reproduction. This represents a biological minimum for a viable population.

Table 6.10
Comparative birth intervals

Population	Birth interval (months)
DS3	36
Semoi	42
17th C French:	
Crulai	25.9
Hirschland	33.6
Argenteuil	20.8

Source: Wilmsen 1986.

There are however other biological limits upon this figure. The average female who reached age 15 could expect to live until between 31.1 and 34.4 years. Now given the limit of 5 children, on average these children have to be produced within 15 years if we allow (mainly for convenience sake) that first birth occurred around 17.5 years. This would indicate an average birth interval of three years. The shortest birth interval for natural fertility groups is 22.4 months for the Hutterites (Wilmsen 1986). Hunter-gatherer groups, most notably the !Kung have longer intervals, c35.4 months (Howell 1979). Three years fits within the range of known intervals of several agricultural groups (Table 6.10). It must be remembered though that this rough estimate fails to account for two factors: possible levels of infertility amongst females who reach age 15; and the high level of infant mortality which means that (estimating zero growth) 39% of births result in death during the first year. This would substantially reduce the length of birth intervals thereby easing some of the pressure of reproducing every three years. Infant death is often followed by a shorter birth interval (eg. Billewicz and McGregor 1981; Wyon and Gordon 1971), which means that the three year birth interval is actually easier to sustain.

The above estimates are based on a zero growth rate. A growth rate of 0.005 makes little difference to this figure. Calculation of NRR, allowing for growth, requires an estimate of the generation length. Average generation lengths given by the Weiss tables for life expectancies at 15 years of age of 15 and 20 are 25.5 and 27.5 years respectively (Weiss 1973). Female mortality is in fact higher in the DS3 population than in either of these two groups so mean family size was calculated for three alternative generation lengths: 20, 25 and 30 years (* Using a l_{15} of 0.4 based on the female life table at r=0.004):

r=0.004	20	25	30
NRR*	1.08	1.1	1.12
MFS	5.4	5.5	5.6

This would have the effect of shortening the possible birth interval but not to unreasonable levels. On the other hand it would be difficult, given the levels of female mortality, for the population to sustain any higher rate of population growth unless a corresponding drop in mortality occurred.

The figures given above are determined purely by the life table conditions and rate of growth. Any other measures of fertility depend upon external information about age-specific fertility. The shape of the fertility curve against female age is fairly standardised. Rising sharply from 15–20, peaking between 20–25, declining slightly to 30, more steeply to 35, then dropping to near zero around 45 (Coale 1972). The scale of this curve of age specific rates is, however, highly variable. This makes it difficult to judge to accuracy of fertility estimates based on applying an average set of age-specific rates (such as Weiss'). Rather the approach followed here is to apply two extreme fertility distributions to the data to estimate the limits of completed fertility and thus completed family size (as per Asch 1976).

The two distributions chosen were the two extremes used by Asch (1976): Jamaica 1951 (early childbearing) and Spain 1940 (late childbearing). The resulting gross reproductive rate is between 3.9 and 4.17 births per female. Multiplying this by two, to account for both sexes, gives a completed family size of between 7.8 and 8.3 children for women who have completed reproduction. Comparable completed family sizes are found amongst other high fertility/high mortality populations: the Yanomama (8.2), Khanna, India (7.5); Keneba (7.5); Manduar (6.4; Table 6.11). In this population, however, only c 3% of all women live to the end of their reproductive years. In addition, while these estimates do provide limits, the shape of female mortality suggests that there may be an unusual cause of death affecting the female population. In this case, there is the possibility that fertility is

severely perturbed by disease factors. This suggestion will be explored more thoroughly within the analysis of pathology.

Table 6.11
Comparative total fertility rates

Population	Completed family Ssze	Source
Anthropological:		
Yanomama	8.2	Neel et al. 1977
Khanna	7.5	Wyon & Gordon 1971
Keneba	7.5	Billewicz & McGregor 1981
Manduar	6.4	
!Kung	4.7	Howell 1979
Kel Tamasheq	6.6	Fulton & Randel 1988
Agricultural Bambara	8.1	
National:		
Bangladesh	6.34	Wilmsen 1986
Guatemala	7.01	
Jordan	6.41	
Turkey	5.60	
South Lebanon	4.68	
Denmark	1.78	
USA	2.34	
Finland	1.61	
Historical:		
Hutterites	9.50	
Quebec	8.00	
Crulai	5.60	

Results: the social composition of DS3

The DS3 skeletal sample differs in several respects from other skeletal samples. Most obviously, it appears to be extremely complete, especially in the youngest age-groups. Until further analysis is conducted it is difficult to say whether the same can be said for the adult age groups. It could be suggested that adult mortality appears to be artificially high during early adult years. Yet the ages 20–30 are the most secure in terms of adult skeletal ageing, since not only are the pelvic changes reasonably clear, but also epiphyseal closure is still occurring (Mensforth 1990). A second rebuttal is to consider that the majority of skeletal populations show similar tendencies. It is proposed that these figures should not be rejected out of hand but analysed further in order to clearly see what they imply.

Recalculation of life table functions using both the upper and lower limits of age estimation demonstrates the degree of variability inherent within the data. These calculations, especially using the upper limit, indicate the age estimations can make changes to the results. The differences, however, result in little appreciable change in the social composition implied by palaeodemography, and the relative ranking of this population against others does not change.

Howell, in 1976, characterised prehistoric populations as belonging to one of four types: (1) high fertility/low mortality (growing population); (2) low fertility / low mortality (stationary); (3) high fertility / high mortality (stationary); and (4) low fertility / high mortality (declining) (Howell 1976). Assumption of a stationary growth rate assumes a population belongs to either type 2 or type 3. All current indicators suggest that DS3 was a type 3 population where, at least for some of the time, fertility may have exceeded mortality causing a low rate of population growth. Whether this growth was pushed by increased fertility or pulled by declining mortality is hard to determine in absolute terms.

The high mortality affected two age groups: infants and adults, especially young adults. The pattern of infant deaths implies two periods of greatest risk: during the first month of life and during the second six months. The marked decline in mortality after age 1, and even greater decline after age 2, indicate a cause of death structure different to that implied by other skeletal samples (eg. those used for Weiss' tables). It appears that a child who survived the first year of life had a greatly increased chance of survival at all further ages. The obvious speculation is whether the level of morbidity in the society was high because of the stressful conditions of childhood, or whether there was a strong selection operating which meant that weaker children were quickly removed from the population leaving only the strongest to survive.

The high adult mortality suggests, however, that morbidity may have been a significant factor. Very few adults lived beyond 50 years. Most males died in middle adulthood (c30–40 years): the period of peak productivity. Male deaths did not, however, suddenly increase with adulthood, frequently a sign of a high number of accidental deaths (Preston 1977). Rather there is a steady increase until 40, and certainly mortality between 15–30 is lower than that of comparable groups. This suggests the absence of accident caused mortality which affects the most active sector of the population, and the possible presence of disease factors which began to extract their toll increasingly during adulthood.

Females experienced entirely different mortality. Levels of female deaths were higher than expected at all ages above 15 years. The excess of female deaths is most obvious at 20–29 and 40–49. In particular, the probability of dying rose sharply between 20–29 years. It is tempting to relate this to the risks and costs of childbearing and lactating. Certainly there was probably some relationship between high infant mortality and high mortality of reproductive females. Presumably an analysis of pathology will offer more clues as to the nature of this relationship.

The most profound effects of both the high infant and female mortality are seen in fertility. Fertility is constantly constrained by biological limits as well as those imposed by population structure. In order for the population to maintain itself each woman needed to have 5 children. Considering that most women died before completing their reproductive careers, these children had to be born from an early age. This in turn can result in high infant and female mortality because

of the increased risks of childbearing at young ages. Consequently a cycle can be formed where an infant death leads to a shorter birth interval, leading to both an increased need to reproduce plus an increased risk as repeated pregnancies take their toll on the mother.

Yet those women who completed their reproductive years must also have had large families though only about half would have survived to reproductive age. The implication is that the majority of women were either childbearing and lactating, and frequently with several older children.

This population, experiencing high mortality and fertility, has therefore a particular age composition as well as specific economic requirements. In a population of 100 (the estimated village size for DS3) this would mean between 4–5 infants, around 12–13 toddlers (between 1–4.9 years), 13–15 children between 5–10 years, and c13 children between 10–15. Looking after these 42–46 children in total would be c54–58 adults, c12 aged between 15 – 20; 19–20 between 20–30, 12–13 aged 30–40; 6–7 aged 40–50; and around 4–5 aged over 50 years. Assuming that adults between 15–50 represent the economically productive members of the society, each adult would have to produce sufficient food to feed one subadult as well as themselves.

Comparison shows this is a high proportion of dependents in a population although, in many agricultural societies, the value of children as labour is considerable (Caldwell 1983). From the age of five onwards, children may increasingly be entrusted with tasks such as collecting firewood, shepherding, collecting water, childminding younger sisters and brothers. This leaves adults and adolescents more free for tasks such as cultivating, harvesting, possibly fishing and maintenance. The youthfulness of the population would mean however that there was little time left for leisure amongst the population. Even so, the pattern of work itself would not necessarily have been all that different from traditional agricultural populations today.

What is obvious is the noticeable lack of old people: only 4–5 adults would have been over 50 years of age. In part their productivity depends on patterns of disease and morbidity and how these change with age: in other words: how capable these individuals were of caring for themselves or others? Analysis of pathology may be able to answer this. The social implications are marked. Few children would have known their own grandparents and it may be presumed that these old adults played an important role in the village in guiding experience-based decisions. Whether old age is reflected by greater status could be tested in an analysis of burial practices.

Socially, the village is faced with several problems. Owing to the high female mortality in particular, few marriages would have survived the full reproductive period. Since women died most frequently during their reproductive career, this meant that a large number of families were motherless. This would result in either a great burden of care, both on the older subadults in each family, as well as the father, or else a push for remarriage or family amalgamation. Remarriage, however, could be a problem in this village since high female mortality also means a shortage of adult females for adult males even if the age of marriage for both sexes was very similar. This may have necessitated male exogamy with corresponding female endogamy.

A greater gap in the age of marriage (older males to younger females) would not solve the problem. The only result would be more absolute orphans in the population. As it is one-third of those who reached 15 years had already lost both parents. Thus, of 14 adolescents only nine had at least one parent. Since there are only 4 'elders' in the society, their role as substitute parents could not have been great. Presumably the responsibility fell upon the community and especially the extended family. The reliance upon the community for provision of the large number of orphans may also explain the lack of status differences visible in the cemetery. In terms of inheritance, the picture would also be affected, since if vertical descent applied there must have been land tied to children too young to work it. Presumably social organisation was adapted to this occurence.

The situation can almost be described as a vicious circle. Females have to bear large numbers of children in order to maintain the population. In turn they suffer high rates of female mortality. The consequent lack of adult labour results in a high demand for more children to provide labour, and so the cycle continues.

The question is how vulnerable is such a population to economic or environmental crisis. The survival of the DS3 population over time implies that it was either in a stable environment or reasonably invulnerable. An economic crisis, however, precipitated by climatic change or changes in water level could presumably cause disaster to a population carrying such a heavy load of dependents. On the other hand, the fact that the population is fairly young in age means that it could be quickly replaced.

A change in mortality levels could also lead to problems. Presumably since disease most frequently affects the youngest and oldest members of a community, the effects of an epidemic could be recovered from fairly quickly. A decline in mortality, though, particularly either of older infants or young females, could have precipitated rapid population growth. This could make environmental and economic fluctuations even more devastating. An agricultural population such as this would be very reliant upon the stability of its adult members and is finely balanced between either rapid decline or rapid growth, which may go some of the way to explaining why there is an apparently close tie between population and water levels on the Island (see Larsen 1983).

It is possible, however, that the extreme conditions which existed at DS3 did not occur in the larger villages towards the

north of the Island. Saar village is an example of one of these.

Saar Mound 5

The age at death distribution

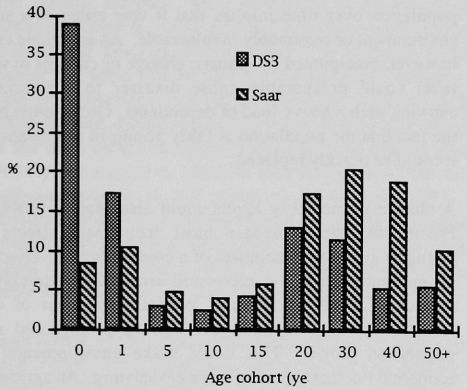
The sample size for Saar Mound 5 is substantially smaller than DS3. It consists of 152 individuals the age distribution of which can be seen in Figure 6.11 Despite the fact that some of these skeletons were extremely well-preserved, there is a similar percentage of aged and sexed skeletons as in the DS3 sample.

Twenty-eight percent of the sample is less than 15 years of age. The majority of these individuals could be aged to specific cohorts, if not specific years. Only two could not be classified with any certainty.

The situation is less secure for adults. Of 110 adults, 76 (69%) were sexed. This resulted in an unbalanced sex ratio of .8 males: 1 female. Due to the small size of the sample, however, this divergence from equality is not statistically significant. The remaining 34 adults were assigned sex on the basis of the 0.8 sex ratio in order not to falsify results.

51 adults could not be assigned age on the basis of pubic indicators. 10 (9.1%) were aged purely by cranial indicators while 41 (37.3%) had to be apportioned across age classes.

Figure 6.11
Age at death (Saar and DS3)



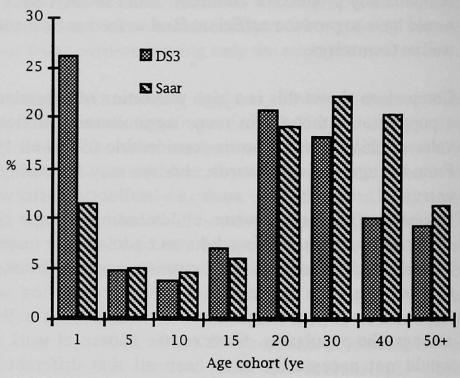
The final distribution of ages at death shows some significant deviations from DS3 (Figure 6.11). Most obvious is the under-enumeration of subadults age 0–1 year. Only 8.3% of the Saar sample belonged to this group compared with 38.9% in DS3. The same trend appears to also hold for the 1–4 age group although the difference in proportions is less marked. It is obvious that owing to these marked differences in distributions all other percentages are not comparable. The need is to identify if these sections of the sample are biased.

The search for bias

Again the three ways of testing for bias may be used. The ratio of deaths 0–1:1–20 is 0.25 in this sample. The Saar skeletons then would just manage to fit into the 'normal' distribution of ages at death (Brothwell 1971). The sample though is obviously on the limits of acceptability.

The problem is more obvious when the shape of the mortality curves is examined. The first criterion of a normal distribution is violated since mortality between 0–1 is less than for successive age groups. The only criterion held is that there are fewer deaths between 10–14 than between 15–19. Otherwise a subadult mortality (before 15) of 27.7% falls outside the acceptable limits defined by Buikstra and Miekle (1985). The probability of bias affecting the youngest age group matches the analysis of burial practices; it has already been suggested that due to differential burial practices infants could be under-represented.

Figure 6.12
Age at death (1+ years), Saar and DS3



There are two results. Firstly life table construction and comparisons between the samples need to exclude the youngest age group. Secondly, the distribution of subadult deaths, particularly at younger ages, must be examined to identify exactly at which age the distribution of ages at death becomes representative of the contributing population. Figure 6.17 demonstrates the problem: one result of excluding the 0–1 age group is that it accentuates a possible under-enumeration of subadults between age 1–4 years. This in turn affects comparisons in the older age cohorts. Elimination of the 0–4 age group entirely would increase the compatibility of the two samples but this may be obscuring significant differences between the two samples. The problem highlights the difficulties of relying solely upon age at death distributions. In order to clearly identify differences in mortality between the two samples it is necessary to compare a measure such as Qx that does not incorporate cumulative enumeration errors. This necessitates the construction of a life table.

Life table for Saar

Unfortunately one of the assumptions of the life table is already violated. As for DS3, it is possible to assume no net migration, and constant birth and death rates over time. The primary assumption, however, of no demographic disruptions in the previous 50 years cannot be made. This is for two reasons: the sample is small (152 individuals); and it is suspected that it may only cover a period of 50 years. This is probably insufficient time to balance out the effects of random fluctuations. Consequently all analysis using the Saar samples needs to take account of the fact that it may not be entirely representative of the living population over one year of age. Obviously this necessitates careful evaluation of the demographic results as well as independent verification by analysis of the pathology.

The resulting life tables can be seen in Table 6.12, including and excluding the 0–1 age group. The q_X values are unaffected by omission of the youngest age groups which means that a direct comparison with the DS3 population is possible, although one considering the upper and lower limits is not (Figure 6.13). Apart from the two youngest age groups, the curves are similar: low mortality during the mid-subadult years, climbing slightly at 15 years, then more sharply between age 20–30. Up to this point there is marked similarity between the two populations although the probability of dying at Saar was slightly lower in younger adult years. In contrast, while the probability of dying declines slightly at age 40–49 in the DS3 group, it continues to increase at Saar. These figures are independent of differences in neighbouring age cohorts, so this difference represents a true divergence not merely an error of accumulation. Nevertheless in general, the correspondence of the curves is good, making the divergence in the two younger age groups even more obvious.

Similar trends can be seen in the comparison of life expectancy (Figure 6.14). Life expectancies at all ages tend to be higher for the Saar population than DS3 with the exception of the 40–49 year cohort. The difference is a consistent 2 years so that at age 15 an individual from Saar is expected to live to 36.6 years, one from DS3 till 35 years. In both groups the life expectancy is greatest at age 5 indicating

Table 6.12
Life table, Saar
(w=70)

x	N	nD_x	d_x	l_x	nq_x**	nL_x	T_x	e_x	C_x
0	1	12.6	0.082895	1000	0.082895	946.1184	28267.5	28.2675	3.347018
1	4	16	0.105263	917.1053	0.114778	3457.895	27321.38	29.79089	12.23276
5	5	7.1	0.046711	811.8421	0.057536	3942.434	23863.49	29.39425	13.94688
10	5	6.3	0.041447	765.1316	0.05417	3722.039	19921.05	26.03611	13.1672
15	5	8.5	0.055921	723.6842	0.077273	3478.618	16199.01	22.38409	12.30607
20	10	26.5	0.174342	667.7632	0.261084	5805.921	12720.39	19.04926	20.53921
30	10	30.8	0.202632	493.4211	0.410667	3921.053	6914.474	14.01333	13.87124
40	10	28.6	0.188158	290.7895	0.647059	1967.105	2993.421	10.29412	6.958894
50	20	15.6	0.102632	102.6316	1	1026.316	1026.316	10	3.630727
w							{15}		
		152	1			28267.5			100

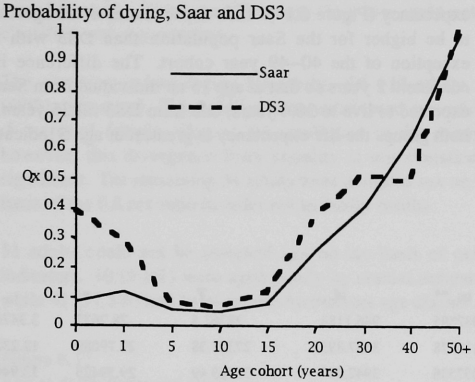
Assuming all deaths, except 0–1 occured at middle of each age group

x	N	nD_x	d_x	l_x	nq_x**	nL_x	T_x	e_x	C_x
1	4	16	0.114778	1000	0.114778	3770.445	29790.89	29.79089	12.65637
5	5	7.1	0.050933	885.2224	0.057536	4298.78	26020.44	29.39425	14.42985
10	5	6.3	0.045194	834.2898	0.05417	4058.465	21721.66	26.03611	13.62317
15	5	8.5	0.060976	789.0961	0.077273	3793.042	17663.2	22.38409	12.73222
20	10	26.5	0.1901	728.1205	0.261084	6330.703	13870.16	19.04926	21.25047
30	10	30.8	0.220947	538.0201	0.410667	4275.466	7539.455	14.01333	14.35159
40	10	28.6	0.205165	317.0732	0.647059	2144.907	3263.989	10.29412	7.199875
50	20	15.6	0.111908	111.9082	1	1119.082	1119.082	10	3.756456
w							{15}		
		139.4	1			29790.89			100

Assuming all deaths, except 0–1 occured at middle of each age group

that even in Saar, where the youngest age groups are under-enumerated, infant mortality has a significant effect on the average life span. Figure 6.14 also demonstrates the lack of effect infant misreporting has on the curves of life expectancy. Truncation of the older age groups is much more significant, but in this analysis both samples are truncated to an arbitrary age of 70 years to avoid biases for misreporting of older age groups.The constant difference between the two groups means that, despite basic similarities in the shape of mortality, it cannot at this stage be assumed that the level of infant mortality at Saar was identical to DS3. Instead it appears to have been slightly lower though the amount cannot be estimated since mortality of infants and of weanlings is highly susceptible to fluctuations in conditions.

Figure 6.13
Probability of dying, Saar and DS3



Comparison of the probability of dying in Saar with the model life tables demonstrates convergence at the same points as DS3. Figure 6.15 shows the plot of q_x values, demonstrating the effect of infant under-reporting. From age 5 to 19 the Saar mortality curve shows a close correspondence to Coale and Demeney West 1 Females. As for DS3, differences appear at age 20 where mortality outstrips that suggested by the model life table. Coale and Demeney Model West 9F was also plotted since it corresponded with q_1 value of the Saar life table. This is the only value at which the two tables converged however; the fit with the West 1 table is obviously much better.

Figure 6.14
Life expectancy, Saar and DS3

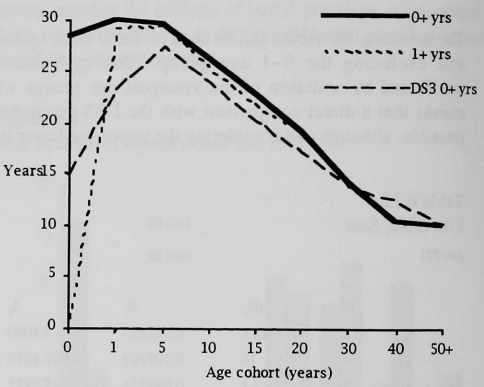
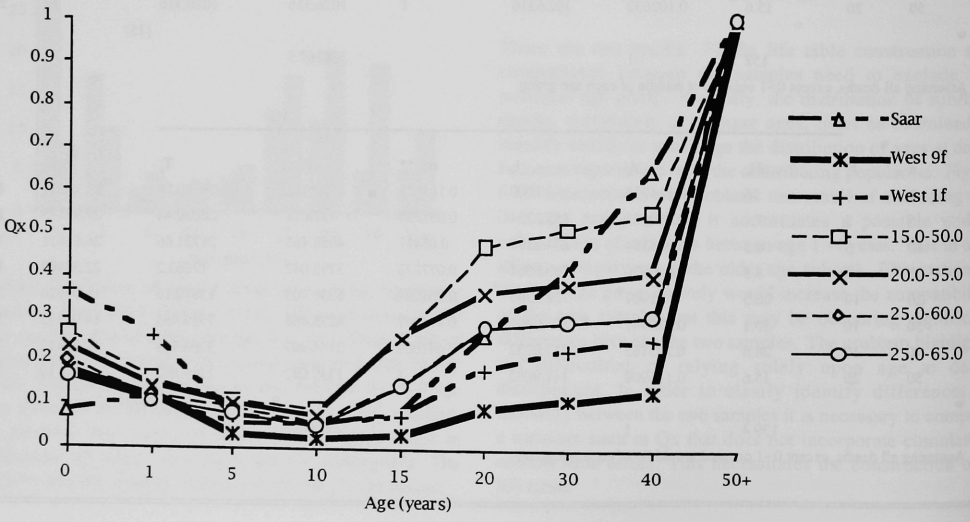


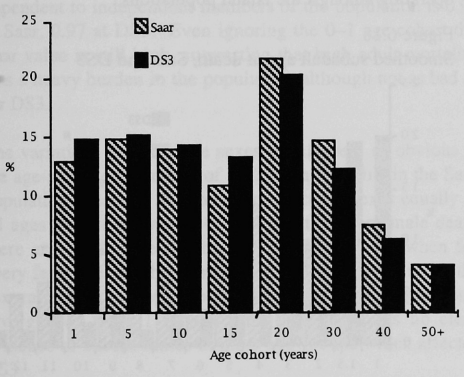
Figure6.15
Comparison with model life tables



The Weiss model life tables, in general, provide a better fit to the Saar sample (Figure 6.15). This may be suggestive of a trend towards under-reporting of the youngest age groups in the populations Weiss used for his models. The comparison is an interesting contrast to the Coale and Demeney tables. Ignoring the 0–1 age cohort, subadult mortality at Saar follows a similar curve to Weiss table 25.0–65.0 until age 15 when the probability of dying at Saar was less than the modelled value, though the values again correspond at age 20. Above this age the mortality experience at Saar outstrips predictions.

This demonstrates some significant differences with DS3. Subadult mortality in the DS3 sample closely matched the West 1 tables, divergence only becoming obvious after age 15. There was very little correspondence between the sample and the Weiss tables in terms of subadult mortality. The probability of dying at DS3 was much higher in early years and lower in the later subadult years. Saar on the other hand, corresponds reasonably well to a model life table suggesting at life expectancy at age 15 of 25, and survivorship to 15 of 65%. Divergence does not occur until age 30.

Figure6.16
Living population, Saar and DS3



This suggests that there may be differences in the causes of death in these two populations. In DS3 the shape of subadult mortality is very steep (the youngest age groups severely affected, adolescents much less affected) while in Saar it is possible that subadult mortality was less steep, tending to decline more gradually. DS3 adult mortality outstrips comparative models at all ages, while Saar mortality corresponds to a greater life expectancy which only significantly deteriorates after age 30. The difference suggests that while direct comparison of life table measures (life expectancy, q_x) implies little contrast between the two groups, in fact there may be some real underlying dissimilarities in mortality experience.

These fundamental differences may be reflected in the age structure of the living populations derived from the life table (the C_x series). Figure 6.16 shows the comparison between the Saar and DS3 populations calculating the figures from age 1 rather than age 0. This actually makes little difference in the DS3 population since the 0–1 age group incorporates only one year compared to the longer age intervals of the remaining cohorts. It is clear from the comparison that while there are no extreme differences in composition especially in the older age groups, the Saar population tends to have fewer young individuals as a proportion of the total and correspondingly more adults, particularly during their productive years. This will also be reflected in the dependency ratio to be calculated later. It suggests that economically the Saar population may have been better placed since it comprised a higher proportion of productive members (both economically and biologically).

Given the problem of incomplete representation, it is impossible without estimates of infant mortality to calculate other life table measures for example Crude Birth Rate. Additionally, the lack of both time depth to the sample and archaeological background make predictions of population growth difficult. Some of the same constraints on DS3 apply to Saar. We know that the cemetery was used both before and after this period. It is suspected that a settlement is located nearby and population decline appears unlikely considering the size of the Tylos settlement at Saar, which leaves population stationarity or growth as alternatives The high mortality (while slightly less than DS3) still makes massive population growth unlikely since it would be very difficult to maintain. It is therefore assumed that 0.004 is an upper maximum of sustainable population growth experienced by this population. Since there is little difference in the two life tables ($r = 0$, $r = 0.004$) the second will not be calculated for Saar.

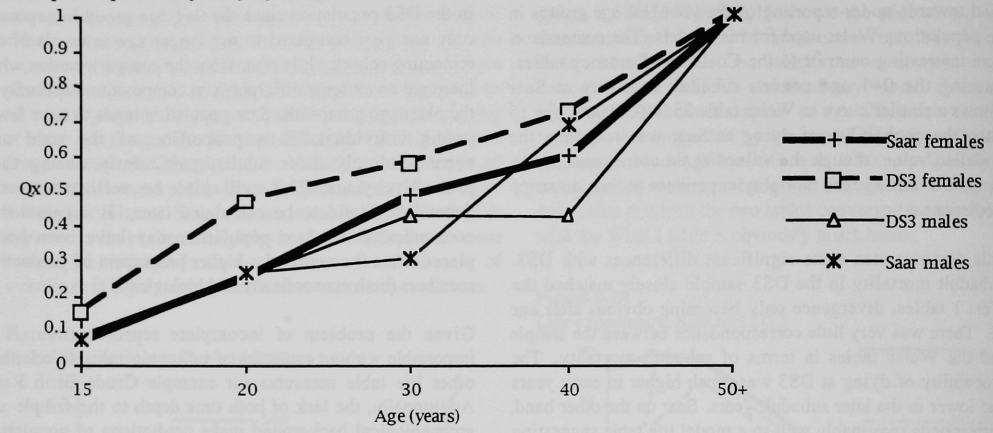
Males vs females

Similarly since the sample size is small and the sex ratio deviates from what is expected, it is not possible to calculate male and female life tables from 0 years. Rather they are constructed from age 15. Unlike the DS3 population, mortality experience amongst adults does not differ greatly with sex (Figure 6.17). The most significant divergence is at age 30 where female mortality exceeds male mortality. In this population there is little evidence of the clear sex specific differences prevalent in DS3.

This can be seen in comparison of sex specific mortality in the two samples. DS3 female values exceed those of Saar at every age group and most markedly at 20 years. It suggests that the probability of deaths for females in Saar rose steadily but more slowly than at DS3, reaching a point at age 30 equivalent to age 20 at DS3, following which the probability of death declines. The shape of the curves is basically similar but steeper earlier in DS3. This supports the suggestion of slightly higher mortality in the DS3 population.

There is no similarly clear trend in male mortality. The probabilities of dying are similar at ages 15 and 20 in both

Figure 6.17
Sex-specific probability of dying, Saar



populations. Probability of death for DS3 males however continues to rise at the same rate to age 30 then level out to 40 years. For Saar males the reverse happens: the probability of mortality levels at age 30 then rises sharply again to greatly exceed DS3 at age 40. Its difficult to determine, however, how significant these differences are since slight under-reporting of males at age 30 and slight over-reporting at age 40 could easily account for observed differences; there is not the consistency seen in the same comparison of female mortality. It is also noteworthy that Saar males and females, as well as DS3 males, experienced the same probabilities of dying between ages 15 and 20. The obvious test to determine whether this is a meaningful similarity is to examine the pathology of these skeletons and check if they experienced similar cause of death structures.

Subadult mortality

Analysis of pathology should also help in distinguishing significant differences in subadult mortality between the two samples. The smoothed age at death distribution from age 1 to age 15 can be seen in Figure 6.18. There is, unsurprisingly, an obvious decline in subadult mortality with age after 3 years. The frequencies before this appear to be clearly anomalous especially when the corresponding DS3 data are examined. After age 3, the shape of the age at death distributions closely correspond, before this Saar is clearly under-represented. It suggests that the Saar sample lacks skeletons from the 0–2 age group, not just the 0–1 year.

Knowing this, it is possible to compare in detail the probabilities of dying for those subadult age groups which are unbiased. The two curves can be seen in Figure 6.19. The comparison demonstrates the lack of differences between the two populations. Mortality from age 3 onwards is nearly identical. The problem of course, as mentioned earlier, is that this tends to be, at least in skeletal populations, a fairly invariant period, especially compared with the initial age cohort, so it is difficult to know what else could be expected.

The earlier comparisons with the Weiss tables do demonstrate, however, that in terms of subadult mortality from age 3+, Saar and DS3 are more similar to each other than to those populations forming the basis of Weiss' tables. It emphasises the possibility of specific cause of death factors operating amongst the Island populations.

Figure 6.18
Smoothed subadult age at death, Saar and DS3

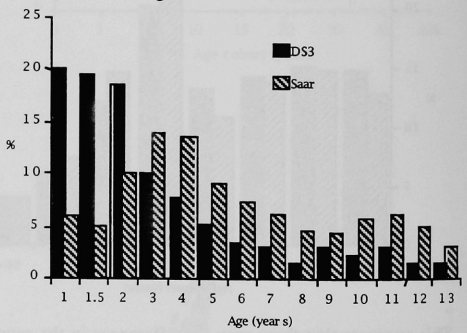
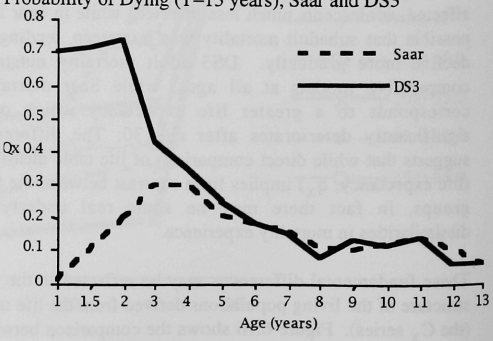


Figure 6.19
Probability of Dying (1–13 years), Saar and DS3



Further demographic measures

The extent to which ratios can be used as indicators of demographic parameters is limited with the Saar material by the avoidance of the 0–2 age group. Some ratios, however, such as that of relative fertility (D30+:D5+) are extremely useful in allowing comparisons of factors otherwise unmeasurable. This ratio in the Saar population is 0.62. Comparison demonstrates that it is possible that the Saar population experienced lower fertility levels than the DS3 population. The list of comparative ratios demonstrates that the Saar value falls around the middle which is probably indicative of moderate fertility rather than the high levels experienced to the south of the Island (Table 6.5). Other cannot be calculated owing to their reliance on the infant data.

Dependency ratios also cannot be computed, though by ignoring the youngest age group, comparison can be made with DS3. 41.5% of the Saar populations (1+ yrs) was less than 15 years of age, compared with 44.3% of the DS3 population. This mirrors the suggestion of both slightly lower fertility and better mortality conditions for Saar. Interestingly the percentage of the population over 50 years is nearly identical in the two populations: 3.8% of Saar, 3.9% of DS3. These figures indicate that the ratio of economically dependent to independent members of the population is 0.91 at Saar, 0.97 at DS3. Even ignoring the 0–1 age cohort the Saar value is still high, suggesting that high adult mortality was a heavy burden in the population, although not as bad as for DS3.

The variations between the sexes which were so obvious in the age-specific sex ratios of DS3 are not visible in the Saar population. Death at Saar tends to affect the sexes equally at all ages except between 30–39 when for every male death there are 1.6 female deaths and between 40–49 when for every female death there are 1.6 male deaths. As a result the sex ratio of the living population shows no significant divergences from 1 at any adult age. There is no clear evidence of excess males at older age groups which affected DS3.

This is reflected in the percentages of widow- and widowerhood. If the age of marriage for females was 15 years and males 20 year, 51.7% of wives would survive their husbands and 49.3% of husbands their wives. The mean age of widowerhood for men given these conditions would be approximately 38 years compared with 36 years for DS3 males. The average woman reaching age 15 could expect to live to 36.7 which is a marked improvement over the DS3 population, allowing greater time for women to successfully replace themselves.

Unfortunately the corresponding orphanhood rates cannot be calculated since they are reliant on the l_x series which is strongly affected by misreporting of the youngest cohorts. Similarly fertility measures are dependent on the youngest cohorts and cannot be estimated.

Results: the social composition of Saar

The effects of bias in the Saar population clearly have a significant impact on the amount of reliable analysis which can be done. Unlike the DS3 sample where analysis can be conducted solely within terms of the one population, Saar can only be analysed fully by comparative measures. The problem is not simply due to the under-representation of the 0–2 year age group, but also to the smaller sample size which makes it more difficult to assess whether or not dissimilarities are truly significant.

In many respects Saar is startlingly similar to DS3. It appears that the population represented by the skeletons would be classed as a type 3 population (high fertility/high mortality) though without suffering the extremes of DS3. The results suggest that the Saar group was more stable population which could maintain itself longer. Certainly the amount of archaeological remains around the Saar area attest to the time depth of occupation around this particular site.

The lack of an accurate estimate of the 0–2 age group means that it is difficult to assess subadult mortality even in comparison to DS3. The problem is not because estimates for older age groups are affected but because infant mortality and the 2 year old death rate are sensitive indicators of living conditions and therefore highly variable. Subadult mortality on the whole, however, appears to be very similar to DS3 but at a slightly lower level, possibly suggesting better living conditions in this village An analysis of disease patterns, especially stress indicators, should make it clear whether this is the case or not.

Adult mortality, while generally lower at Saar (life expectancy at 15 years is 1.6 years greater than DS3), is still greater than expected by a comparison with the model life tables. Fewer younger individuals died, but more died during their reproductive years than predicted. The onset of this increased adult mortality is later than at DS3; it is not until age 30 that the Saar figures begin to exceed those predicted by comparable models, while by age 20 DS3 mortality was already significantly greater than the model mortalities.

Also, unlike DS3, there is no clear evidence of sex-specific differences. This can be checked by comparing male and female levels of pathology. The mortality curves suggest that both sexes experienced a similar cause of death structure which in turn is comparable to that experienced by DS3 males. Nevertheless, the elevated mortality levels after 30 years of age do need to be explained.

It is noteworthy, though, that the shape of the Saar female mortality curve is similar to the corresponding DS3 curve. The primary difference is that increased female mortality at Saar does not occur until 30 years compared with 20 years at DS3. This is immediately visible in the difference between the female life expectancies at 15 years. On average Saar females lived until 36.7, almost 4 years longer than DS3 females.

In terms of fertility this is an obvious benefit. While actual levels of fertility could not be estimated, females would not have had to produce so many surviving children in order to maintain the population. It is probable, however, that fewer children died thereby increasing the average birth interval and decreasing the average fertility / woman. Its impossible to determine the interplay between these two factors though levels of morbidity and indications of the time of weaning may indirectly demonstrate whether birth intervals were longer in the Saar population.

Economic conditions for the population may have been slightly better at Saar than at DS3. This is not only because mortality is lower but also because fewer of the living population were less than 15. For instance, in a population of 100 at Saar, 41 people would have been aged between 1 and 15 years, compared with c44 at DS3. It is probable also that there were more infants in the living population at DS3 than a population of comparable size at Saar.

In addition, adults tended to live longer at Saar so that in the living group there were slightly more adults at old ages and slightly fewer at younger ages than at DS3. This means that there is a more experienced group of adults to care for the group economically. Yet there are still very few individuals in the oldest age group. While this means that these few adults would be less of an economic drain, it also means that there is little depth to the knowledge gained by experience within the population.

Socially the population would have experienced fewer of the problems suffered by DS3. While few marriages would survive the full reproductive span, the older age of women at death (four more years) implies that not only did they have better chance of reproducing themselves, but that by the time of death the average age of the oldest surviving child would be greater than at DS3. These survivors would be better placed to help with the tasks of caring and maintaining the family although rates of orphanhood must still have been quite high. The similarity between male and female mortality means that there is no excess of widowers over widows, so that remarriage would be a more viable option than at DS3 where the shortage of females would have caused real problems.

In conclusion, the analysis suggests that the Saar population may have been better placed both in terms of living conditions and the chances of surviving economic or environmental crises. Nevertheless, there are major similarities between the two populations since the underlying population structures are quite similar. The greatest differences are more those of scale than of structure.

Comparative studies

Lest it be thought that the conditions described for DS3 and Saar are totally novel, the following is a brief review of several small-scale demographic studies on modern traditional groups. The closest comparisons are clearly with high mortality populations, generally agricultural, and located within tropical and subtropical areas. Two studies, in particular, have a close relationship to the Bahrain study. These are the Khanna study, a survey in rural Punjab, India (Wyon and Gordon 1971), and the Keneba study in Gambia (Billewicz and McGregor 1981).

Before examining these two surveys in detail, a brief comparison of absolute rates with a range of populations is informative. The figures most commonly compared between populations are the crude rates, survivorship and life expectancy. The estimated crude birth and death rates for DS3 are extremely high: 60–70/1000. Historically crude birth rates in excess of 50 per 1000 have been reported from the Cocos-Keeling Islands between 1888–1947 (55.8/1000) and from Candalaria, Colombia (>60/1000; Asch 1976). Modern populations with high rates include the Yanomama (between 56–59/1000; Neel et al. 1977); the Fulani, West Africa (40–60/1000; Hill and Thiam 1988); Matlab, Bangladesh (up to 46.4/1000; Billewicz and McGregor 1981); and Khanna, India (37.7/1000; Wyon and Gordon 1971). All of these groups are currently experiencing population growth. In no modern instance has a comparably high crude death rate been reported. For instance the crude death rate for Matlab is only between 13.6–16.5 per 1000; for Keneba, West Africa, the reported rate is 36.7 per 1000 (Billewicz and McGregor 1981). Closest to the Bahrain rate are the estimated rates for the Yanomama (between 48 and 50 per 1000; Neel et al. 1977). Howell has characterised this group as the archetypal high mortality/high fertility population, a combination which also appears to match the Bahrain populations (Howell 1976) and historically this may well have been a common pattern. Wyon and Gordon describe the Khanna population prior to 1921 as having death rates roughly approximate to the birth rates, between 40–50 per 1000 (Wyon and Gordon 1971).

The main difference between past populations, such as DS3 and Saar, and modern groups is the high adult mortality. Comparable infant and child mortality rates are easily found. In the DS3 population c60% of all those born died by 15 years of age, 39% within the first year. Closest comparisons again are with those agricultural and pastoral groups without health care in tropical and subtropical areas. The high rate of mortality prior to five years is matched by rates from Keneba, Gambia (50% die before 5 years; Billewicz and McGregor 1981); the nomadic Kel Tamasheq, Mali (deaths to age 5 are between 30–40%; Randel 1991); the Yanomama (between 40–50% dead before 15 years; Neel et al. 1977); and Khanna (just over 60% of live births survive to adulthood; Wyon and Gordon 1971).

On the other hand, comparable life expectancies at 15 years are more difficult to find, reflecting the substantial difference in adult mortality between the Bahrain populations and modern populations. Closest comparisons occur again with the populations showing the highest levels of fertility and mortality, especially mortality. These are the Gourma Kel Tamasheq where life expectancy at 15 is between 29–33 for

males, and 30–39 for females (Randel 1991); and the Yanomama where life expectancy at 15 is approximately 25 for males and 27.5 for females (Neel et al. 1977). Life expectancies at 15 for DS3 were between 16.1–19.4 years for females and 24.5 and 27.9 years for males. Obviously the rate of death amongst young adults, particulalry females, in the Bahrain populations is extremely high and requires explanations.

Nevertheless many of these populations carry a similar high load of dependents. Some also have a high proportion of children who are either partial or complete orphans. Figures of orphanhood are hard to obtain. Rates have been reported, however, for the Fula Bande, an agricultural village population in West Africa (Hill and Thiam 1988). In this group c30% of those who reach 15 years are fatherless, and c15% motherless. This is indicative of a group where male mortality exceeds female, yet it demonstrates that the conditions implied by the skeletal samples from Bahrain, whilst harsh, are not unique.

Even more important than the comparisons of exact rates, however, are the structural similarities evident between the DS3 and Saar populations and those of small agricultural villages such as Khanna and Keneba.

The village of Khanna was studied by Wyon and Gordon from 1957–59 (Wyon and Gordon 1971). The village is largely dependent upon agriculture based on a mixture of rainfall, wells and increasing amounts of irrigation. Deaths in the population during the period of study were high during early childhood. While children less than 5 form 14.5% of the population, they constitute 55% of deaths. These primarily occur in the first month, and again between 6–11 months of age, declining by three years of age. This primarily reflects the impact of infectious disease and diarrhoea upon the population, including specific diseases such as tuberculosis, tetanus, and pneumonia.

The causes of death change with age. Between 5–14 years tuberculosis, typhoid and accidents are the primary causes. From 15–44 most deaths are due to tuberculosis with maternal deaths occurring in 30 of 10 000 live births. From 45–64 cancer replaces tuberculosis as a major cause of death, and after 65, while cancer is still important, heart disease, senility, and accidents are also major causes of death.

At each age, except the youngest, female deaths outnumber males. This means that there is an excess of males in the adult population; approximately one-fifth of men over 25 years are unmarried. The imbalance is partially offset by emigration of young adult males. Amongst the farming caste, however, the numbers involved are quite small: 11/1000. The impact of migration is felt in the village in terms of ameliorating population pressure. Female exogamy does occur but this form of migration tends to be balanced.

The average of marriage for women is 15.4 years, with age of first cohabitation between 16.1–18 years. First birth then occurs early resulting in a high birth rate. On average, a woman who survives until 45 years experiences 7.5 live births. Only 34–44% of these, however, would survive until adulthood. Amongst farmers, for instance, the average completed family was 3–4 children. Only 15% of couples had never lost a child and nearly 50% of women had lost three or more live born children.

The frequent loss of children promotes childbearing. Two factors occur here. Firstly the period of postpartum amenorrhea is shortened resulting in a decreased birth interval, and secondly there is a desire for replacement of the child. Yet more frequent childbearing is not mirrored by increased numbers of children. The cycle of increasing child mortality with decreasing birth intervals, hypothesized for DS3, occurs within the Khanna population. Conception is also limited by the frequent sickness of women.

While levels of adult mortality in this population are not as high as those estimated for either DS3 or Saar, the structural similarities are obvious. The population experiences many similar problems including high infant mortality, a cycle of increasing births and deaths, a need for a large number of births in order to replace the population, and an excess number of adult males. There are some differences, however. The Khanna population is a useful analogue in terms of demonstrating how these demographic rates look in a living population and how the rates estimated for Bahrain are possible in a biological sense. Yet the village of Khanna experiences a significant number of deaths from epidemic diseases such as measles while eradication programs and health campaigns have significantly reduced the impact of chronic diseases such as malaria and tuberculosis. In addition the environment differs, since agriculture in Bahrain is primarily dependent upon irrigation in contrast to Khanna where agriculture is more extensive and dependent to a much greater extent on rainfall. These differences limit the power of the Khanna study to be a satisfactory analogue for either DS3 or Saar. Yet in examining the cause of death structure, the data from Khanna should prove to be a useful comparison providing a model of disease and mortality against which the finds from DS3 and Saar can be examined.

The second comparison of relevance to the DS3 and Saar populations is with two Gambian villages: Keneba and Manduar (Billewicz and McGregor 1981). These small agricultural villages, barely five miles apart, were studied intensively from 1951 to the 1975. Isolated from medical care at the time of survey, their mortality levels are similar although slightly worse than those reported for Khanna. Subadult mortality is comparable to the rate estimated for DS3 and Saar although the pattern differs. High rates of mortality peak in the first six months but persist until the third year of life. The importance of this comparison to Bahrain is not structural, as for Khanna, even though the cause of death structure does provide an alternative model applicable to malarial groups.

The important feature of the Keneba study is the nature of demographic differences between the two neighbouring villages. Despite similar living conditions and a similar age structure at death, mortality and fertility at Manduar is consistently less than at Keneba. The crude death rate is 36.7 per 1000 for Keneba, 24.7 for Manduar, with neonatal mortality 85.2 per 1000 live births at Keneba and only 49.6 at Manduar. The differences in vital rates persist throughout all aspects of the demography: maternal mortality (10.5/1000 births in Keneba, 9.5 in Manduar); crude birth rate (58.4 in Keneba, 49 in Manduar), and total fertility rate (7.5 per female in Keneba, 6.4 in Manduar). The resultant growth rates demonstrate that, despite higher fertility levels, the greater levels of mortality create less population growth for Keneba: 1.1% per annum compared to 2.2% per annum in Manduar. (Billewicz and McGregor 1981)

These differences are related to malaria parasitaemia, particularly amongst young children (Billewicz and McGregor 1981). The interpretation has been questioned (Costello 1986), but no alternative explanation is currently available.

The different demographic rates result in different age structures and in different socio-economic conditions with the two villages despite an outward similarity. Living conditions are slightly better in Manduar in terms of mortality, particularly for young children and women of reproductive years. Better mortality rates, however, are not necessarily indicative of better morbidity; at times improvement in mortality is accompanied by worsening morbidity. This is partially true in the Gambia situation where the pattern of disease is very similar in the two villages. Nevertheless, if malaria parasitaemia is less at Manduar, one can assume that levels of morbidity are also slightly lower in this population. The resultant lower mortality is not accompanied by higher fertility. The cycle of less survivorship and more births versus greater survivorship and fewer births is in place here, so that Manduar, while growing at a faster rate than Keneba, has both lower mortality and lower fertility.

The situation is possibly analogous to that described above for the villages associated with the Saar and DS3 villages. Demographic measures, particularly of mortality, suggest that the deaths of young children and women, while following a similar trend, were slightly less at Saar than at DS3. The exact degree of difference is hard to determine because of the possible bias present in the Saar sample but the magnitude is probably similar to that observed between the two Gambian villages. This then suggests that, apart from using Keneba as an alternative model of cause of death to be compared with the epidemiological data from DS3 and Saar, it may be that the differences in mortality that occur between Keneba and Manduar are comparable to those occurring between DS3 and Saar. An examination of pathology should go some way towards addressing the reasons for mortality differences.

The use of the two analogies: Khanna and Keneba, has been to demonstrate possible similarities to the Bahrain situation, and to propose possible models, particularly related to causes of death, against which the Bahrain data can be compared. These, however, are only probabilities. The villages of Gambia and the Punjab may have experienced similar demographic rates to the historical villages of Bahrain but there is no suggestion that the economic and social organisation in any one of these villages is identical to that which existed in the past on Bahrain. While the problems faced by all these populations may be similar, it is unlikely that their solutions would be identical, determined as they are, not just by demography, but by environment, history, politics, and culture. In addition, the Bahrain populations still vary in some significant respects from all comparisons, especially adult mortality. The issue is to explain why these variations occur and to explore further the implications of demography and disease for the people who actually lived on Bahrain.

Conclusion

The villages associated with DS3 and Saar, described here, experienced shorter life spans and generally higher mortality than many modern populations. This means that individuals in each group must have carried a heavy economic burden, and also that social structures were probably adapted to ensuring that such societies could continue.

The alternative explanations of course is that the data are simply wrong: that the samples are biased; or that methods of skeletal ageing of adults are extremely skewed towards the younger age groups. These are not, however, satisfactory explanations. Firstly the similarities between the two samples from two spatially separated cemeteries are striking; despite their differences the two groups are more like each other than like comparative groups. The implication then is that this reflects the same underlying trends.

Secondly, the argument concerning ageing is an inadequate explanation. As pointed out earlier, up to age 30, skeletal ageing is relatively precise since there are many additional indicators. Presumably therefore errors of under-ageing would apply most strongly to those currently aged 30 and above. Ageing errors too can be assumed to have a nearly normal distribution. That is the greatest probability of age misclassification is to the neighbouring age class rather than to more distant age classes (e.g. Table 5.2). Looking at this in terms of the two samples studied, misclassification would have little effect, especially on measures such as the dependency ratio; the population would still be much younger on average than modern populations and the greatest number of deaths would still occur between 20–30 years.

This is supported by using the full range of age estimates for each cohort: the fuzziness inherent in methods of age estimation makes little difference to basic demographic

parameters, and even less to the structure of the living populations.

It is more satisfactory at this point to see whether disease patterns support the interpretations based on demography. For example, in DS3 skeletons: is there evidence of early weaning, female-specific pathology, low accident mortality but signs of chronic diseases that affect older males and general evidence of high morbidity? Does Saar suffer generally less morbidity, no sex-specific differences in disease and low accidental mortality? Are the diseases found within these populations comparable to those observed in modern populations and is the resultant age at death comparable with known patterns?

Once it can be determined what elements of the population structure are supported by the analysis of pathology, then the question of why this particular age structure occurred can be at least partly answered, and its implications fully explored.

Chapter 7

Step 3: Childhood stress indicators

As indicated earlier, the division between specific pathological indicators and non-specific indicators of stress is arbitrary. Partly for the sake of convenience, but also because essentially the processes of analysis do differ, the description of palaeopathology has been divided into two sections. The first broadly covers indicators of stress. The second concerns those pathological conditions to which a specific cause can be tentatively assigned.

The stress indicators recorded in this population were enamel hypoplasia and the rate of childhood growth. These are indicative of childhood conditions: the number of stress episodes and recovery experienced by children less than six years of age, and the degree of growth disruption experienced by those who survived.

Each condition indicates, to varying extents, living conditions of the individual throughout childhood. They are by no means the complete picture and, as will become obvious, each indicator has associated problems of interpretation. Nevertheless they are a start to sorting out the question of past morbidity.

Linear enamel hypoplasia

Linear enamel hypoplasia (LEH) is a horizontal groove or undulation on the crown of a tooth caused by decreased enamel thickness (Skinner and Goodman 1992). This is caused by a cessation of enamel formation during calcification of the tooth over a period of several weeks to more than two months (Rose et al. 1985). Possible causes of this disruption may be hereditary, traumatic or systemic. Systemic LEH is the most common and can be distinguished from trauma which is generally localized (Goodman et al. 1980; Pindborg 1982). In contrast, systemic LEH affects the tooth's surface in a linear pattern corresponding to the chronology of tooth formation (Massler et al. 1941). Teeth appear to be affected differentially according to the pattern of calcification (Goodman and Armelagos 1985; Goodman and Rose 1990). Owing, however, to appositional crown formation, particularly in the cheek teeth, early hypoplasia may be obscured (Hillson 1996).

Numerous studies have identified causes of LEH (for a review see Pindborg 1982). Nikoforuk and Fraser (1974) suggest that the cause may be hypocalcaemia which is a frequent side effect of nutritional and infectious disorders. The synergistic effect of malnutrition and infection, however, makes it generally impossible to isolate a specific cause. At present, LEH is accepted as indicative of a period during childhood when the individual was under some form of severe systemic stress (Goodman and Rose 1991).

Since formation of the tooth enamel follows a regular pattern, the chronology of childhood stress episodes is recorded by the location of the hypoplasia upon the tooth (Sarnat and Schour 1942). The enamel is not remodelled so there is a perpetual longitudinal record of growth disruption and recovery (Rose et al. 1985). Such data can be analysed on both an individual and populational basis.

Numerous factors do, however, need to be taken into account. Firstly, there are individual variations in susceptibility to stress. A 100% correlation between infectious episode and the formation of a hypoplasic lesion does not occur (Via and Churchill 1959), rather, it seems that a 'threshold' effect operates (Goodman and Rose 1990). It is also possible that varying genetic background may make some groups less susceptible to LEH than others. In the present study, however, it is assumed that the Saar and DS3 populations come from a similar genetic background.

Secondly LEH is a sign not only of the onset of childhood stress but also of recovery. If the stress was severe the child may have died (Goodman and Armelagos 1989).

Method of recording

A hypoplasic defect was defined as a definite pit, groove, or line in the enamel's surface which was aligned horizontally (McHenry and Schultz 1976). To aid in recognition, a 10x hand lens was used in order to determine whether or not the surface was disrupted. Each defect was measured from the cemento-enamel junction following the methodology outlined by Goodman and Armelagos (1985). As these authors recommend, the anterior teeth were recorded since these are the most likely to display defects. The amended chronological scheme used by Goodman and Rose (1990) was used to record the timing of each disruption to a six month age unit.

The data was analysed on two levels: on an individual level, and on the basis of the number of six-month units of enamel. The multiple tooth method, whereby a line was only recorded if it was present on more than one tooth for the same six months, was used. This method avoids recording defects of traumatic origin.

Results

Enamel hypoplasia could be recorded on the anterior teeth of 95 individuals. An extremely high proportion of these had evidence of at least one episode of growth disruption (Table 7.1). There are no statistically significant differences in the number of individuals of either sex or age groups affected although fewer subadults than adults had hypoplasic lines in

the DS3 group. All of the Saar individuals had enamel hypoplasia.

Table 7.1
Percentage of individuals with LEH

	DS3			SAAR	
	%	n	% with 1+ defect	%	n
Subadults	77.8	18	72.2	(100)	1
20–30yrs	86.7	15			
30–40yrs	94.4	18			
40–50yrs	100.0	9			
50+yrs	100.0	2			
All Adults	93.8	48	75.0	100.0	16
Males	100.0	26	76.9	100.0	7
Females	86.7	30	70.0	100.0	7
Total Pop.	89.7	78	74.4	100.0	17

The average number of lines per individual was also calculated (Table 7.2). Since attrition could result in lines being lost near the incisal surface, averages were calculated both for all teeth and for those with all enamel units observable. The difference between the two sets of averages is not statistically significant so the following uses all teeth.

Table 7.2
Average number of lines per individual

	DS3			SAAR		
	av.	s	n	av.	s	n
Subadults	2.3	2.1	18			
3–6	1.3	1.8	6			
6–10	2.4	2.2	7			
10–15	3.2	2.4	5			
15–20	2.8	1.3	6			
20–30	2.0	2.3	15			
30–40	2.2	1.2	18			
40+	1.6	1.4	11			
Adults	2.1	1.6	54			
Males	2.1	1.5	26	3.0	2.3	7
Females	2.3	1.7	28	1.2	1.94	6
Total	**2.3**	**1.5**	**78**	**2.5**	**2.25**	**16**
T-tests:		**t**	**p**			
DS3:						
SA vs Ad		0.51	n.s.			
M vs F		0.29	n.s.			
Saar vs DS3		0.68	n.s.			

Individuals from Saar had more lines than those from DS3, but, considering the small sample sizes, this difference is not statistically significant.

Within DS3, the average number of lines is 2.1 per adult and 2.3 per subadult (Table 7.2). This difference is not statistically significant. A trend, however, is visible when the sample is divided into narrower age groups. The youngest age group, 3–6 years, has only 1.3 lines per individual, explicable in two ways. Firstly fewer enamel units, particularly the canines were observable in this group. This, however, is not the full story. Three of the six in this age group had no observable lines at all, compared to three of the remaining 13 older subadults. There appears to be a correlation between absence of lines and earlier age of death. Since line formation implies recovery, this could be seen as indicative of either children dying very abruptly (as in accidental deaths, acute infection), or children experiencing chronic disease with no periods of recovery.

The highest frequency of lines is observed amongst 10–15 year olds who experience an average 3.2 periods of growth disruption sufficient to cause hypoplasia. This does not vary significantly from the average 2.8 disruptions per 15–20 year old. Older adults experienced fewer episodes of disruption: the lowest frequency was observed amongst those who were more than 40 years at the age of death. This pattern of decreased number of lines with increased survivorship has been observed in other populations between subadults and adults (Goodman and Armelagos 1989).

Age seems to be a dominant factor in the frequency of lines in the DS3 group. With regards to the sexes, there was no significant differences between the frequency of lines on males versus females.

The average number of lines could be calculated separately for each sex in the Saar sample (Table 7.2). Females experienced fewer LEH episodes than males. The sample, however, was too small to further divide by age in order to test whether a similar pattern occurred.

The frequency figures indicate that the majority of individuals from DS3 and Saar experienced significant numbers of growth disruptions during the period of enamel formation. Apart from the very youngest age group which would include the most frail, the evidence suggests that those who died at younger ages experienced more numerous disruptive events than those who survived.

By assessing the age at which these growth disruptions occurred, it should be possible to isolate particularly stressful periods of childhood (Figure 7.1). In the DS3 sample the majority of disruptions occurred between 2.5–5.5 years of age, peaking between 2.5–3.5 years. At Saar the peak age appears to be later with the greatest frequency of lines occuring from 3–5.5 years. For both samples, however, the period from 2.5–5.5 years is the period of greatest frequency without evidence for a narrow time period of extreme frequency.

There are only minor differences in the distribution of lines when the DS3 sample is divided into subadults and adults. The distribution appears to be bimodal (Figure 7.2). This, however, tends to be an artefact of the method since at 4–4.5

years the incisors are no longer developing and the method of recording becomes reliant upon the canines. If only the incisors were observable the number of lines declines at this point, yet the number of enamel units remains high resulting in a lower frequency of hypoplasic defects. It is more realistic to see this decrease as a problem with incomplete dentitions rather than an age related phenomen. If all dentitions were complete, the frequency of lines between 4–4.5 years of age would tend to be midway between the frequency at 3–3.5 and at 4.5–5. In this case these percentages are nearly identical.

Figure 7.1
Age of LEH in DS3 and Saar

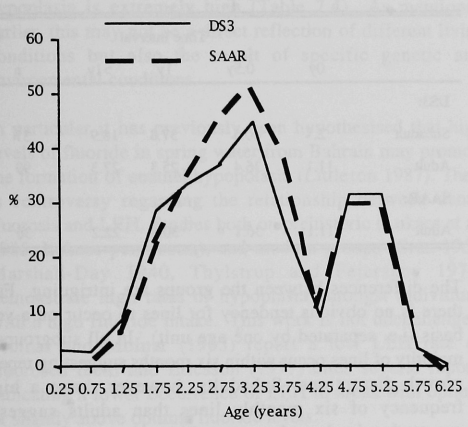
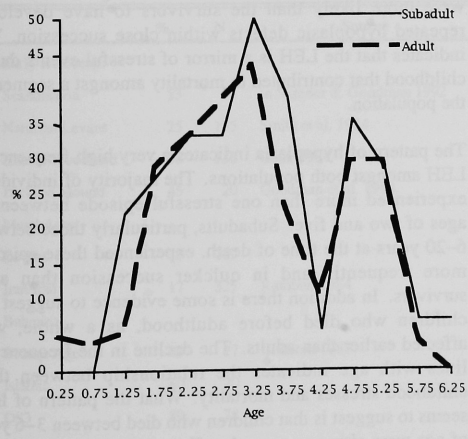


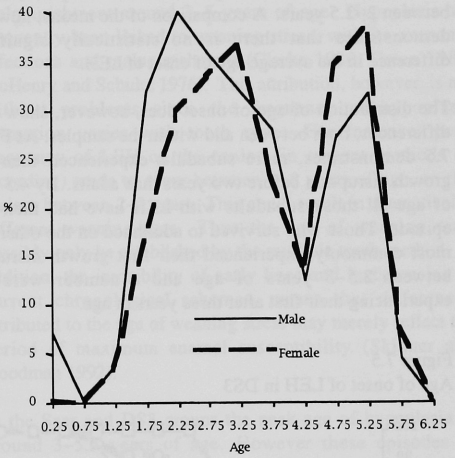
Figure 7.2
Age of LEH in the DS3 sample



The distribution demonstrates a slightly later and more prolonged occurrence of growth disruptions amongst those who died during childhood. This is consistent with the average number of lines data. Peak frequency for adults was 2.5–3 years, and for subadults 3–3.5 yerars. For both age groups, however, the period from 2.5 to 5.5 years appears to have been marked by growth disruptions.

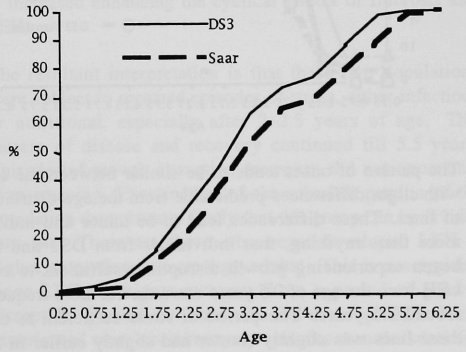
There are few differences in the pattern of LEH amongst males and females in the DS3 sample (Figure 7.3). Peak frequency of lines occurs between 2.5 and 3 years for both sexes. Males experienced more growth disruptions than females in early years (0–2 years) and females more in the 5–6 year age group. Despite the discrepancies at these ages, the overall distribution is extremely similar and differences between the two distributions are not statistically significant.

Figure 7.3
Sex-specific age distribution of LEH, DS3



The number of females in the Saar sample is too small to conduct a similar comparison. Male patterns are extremely similar with a peak frequency of lines from 2–4.5 years.

Figure 7.4
Cumulative occurrence of LEH in DS3 and Saar

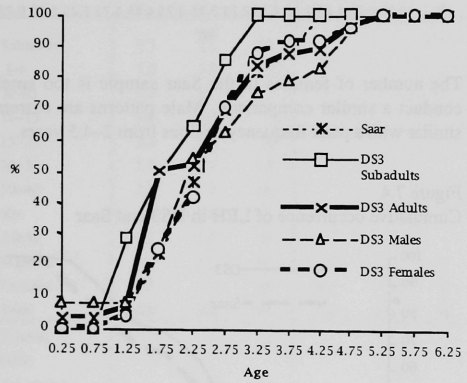


The similarity between the samples is obvious when comparing the cumulative occurrence of lines in the DS3 and Saar samples (Figure 7.4). This type of comparison highlights the differences within the DS3 sample: most episodes of growth disruptions amongst subadults occurred slightly earlier than amongst adults, and amongst males slightly earlier than amongst females. A statistical comparison of the cumulative frequency of lines indicates a significant difference between subadults and adults (Kolmogomorov-Smirnov: $p < 0.005$) with the maximum difference occuring between 2 and 2.5 years of age. There is no significant difference between males and females.

Since most individuals had more than one episode of growth disruption, the age of onset for LEH was calculated. In the DS3 sample this was mainly between 2.5–3.5 years; at Saar between 2–2.5 years. A comparison of the means, however, demonstrates that there is no statistically significant difference in the average age of onset of LEH.

The distribution of age of onset does, however, show some differences, both between and within the samples. As Figure 7.5 demonstrates, more subadults experienced their first growth disruption before two years than adults. By 4.5 years of age all those subadults with LEH have had their first episode. Those who survived to adulthood on the other had, most commonly experienced their first growth disruption between 2.5–3 years of age and a number were still experiencing their first after three years of age.

Figure 7.5
Age of onset of LEH in DS3



The pattern of onset tends to be similar between the sexes with slight differences predictable from the age distribution of lines. These differences tend to be minor and indicate, more than anything, that individuals from DS3 and Saar began experiencing growth disruptions sufficient to cause LEH from the age of 0.5 years onwards, but more frequently around 2.5 years. The period of stress sufficient to cause these lines was slightly shorter and slightly earlier in Saar than in DS3 but the difference is not statistically significant.

To some extent this age pattern is the result of problems with current methods. Different teeth possibly yield different peak ages due to buried striae in the enamel, which means that not all hypoplasic lines are visible (Skinner and Goodman 1992). The age peaks observed in the present samples correspond to those seen in other studies (Skinner and Goodman 1992).

Following the method devised by Goodman et al. (1980), the age distance between lines was calcualted in order to determine whether there was a tendency for lines to occur on a yearly basis, suggesting a seasonal round (Table 7.3).

Table 7.3
Age intervals between hypoplasic defects

	Time Interval				
	0y	0.5y	1y	>1y	n
DS3:					
Subadult	2.7	40.5	37.8	18.9	37
Adult	11.1	36.4	25.3	27.3	99
SAAR:					
Adult	11.1	36.1	30.6	22.2	36

The differences between the groups are intriguing. Firstly there is no obvious tendency for lines to occur on a yearly basis (i.e. separated by one age unit). In all subgroups the majority of lines occur within six months suggesting repeated growth disruptions. Subadults, however, have a higher frequency of six monthly lines than adults suggesting repeated episodes of stress within close succession. In addition more adults than subadults had only single episodes of growth disruptions, and specifically more males than females. Clearly what is indicated is that subadults who died were more likely than the survivors to have developed repeated hypoplasic defects within close succession. This indicates that the LEH is a mirror of stressful events during childhood that contributed to mortality amongst a segment of the population.

The pattern of hypoplasia indicates a very high frequency of LEH amongst both populations. The majority of individuals experienced more than one stressful episode between the ages of two and five. Subadults, particularly those between 6–20 years at the time of death, experienced these episodes more frequently and in quicker succession than adult survivors. In addition there is some evidence to suggest that children who died before adulthood, as a whole, were affected earlier than adults. The decline in the frequency of lines with age indicates the relationship between these childhood stresses and mortality. What the pattern of lines seems to suggest is that children who died between 3–6 years of age were either so severely affected by these stresses that they failed to experience any recovery before they died, or that they died of acute conditions; the remaining survivors experienced repeated episodes of stress. Those, however, who experienced fewer episodes sufficient to cause LEH or

who were less susceptible also seemed to have experienced these at slightly longer intervals. Survivors to 40 years were less affected by these conditions than those who died earlier which suggests that either adults were disadvantaged by childhood illness making them more susceptible to adult illness, or that a subgroup of the population was more profoundly disadvantaged than the remainder.

Discussion

The pattern of hypoplasia seen in these two groups, in particular the high frequency of lines, is comparable to other skeletal samples on Bahrain (Littleton 1987). Compared to other populations outside the Arabian Gulf, the rate of hypoplasia is extremely high (Table 7.4). As mentioned earlier, this may not be a direct reflection of different living conditions but also the result of specific genetic and environmental conditions.

In particular it has previously been hypothesised that high levels of fluoride in spring water from Bahrain may promote the formation of enamel hypopolasia (Littleton 1987). There is controversy regarding the relationship between dental fluorosis and LEH. Studies both on prehistoric (Lukacs et al. 1985; Lukacs pers. com.), and modern groups (Cran 1955, Marshall-Day 1940, Thylstrup and Fejerskov 1978) demonstrate high rates of hypoplasia amongst individuals with a high fluoride intake. This work is not unchallenged: Molnar and Molnar (1985) report a low LEH rate on fluorosed teeth and Ericsson (1977) cites several reports indicating a lower occurrence of LEH in areas with optimal or slightly above optimal fluoride levels.

Table 7.4
Comparative frequencies of LEH (no. of individuals)

	%.	N	Source
Early Holocene:			
Scandinavia	53	58	In Skinner & Goodman 1992
Natufian Levant	25	135	Smith et al. 1984
Illinois Woodland	58	130	Cook 1984
Dickson Mound	45	20	Goodman et al. 1984
Metal Ages +:			
Levant	70	282	
Iran/Iraq	17	327	Rathbun 1984
Bahrain:			
Bronze Age	89	27	Littleton 1987
Islamic	80	20	
DS3	89	78	
Saar	100	17	

It is hypothesised that fluoride interferes with calcium absorption (Fejereskov et al. 1977) and that this is one of the possible causes of LEH (Massler and Schour 1952; Nikoforuk and Fraser 1981). Cran (1955) also suggests that, in cases of nutritional disturbance, the effect of fluoride is accentuated so that the process is cyclical. It seems therefore, possible that in areas of high natural fluoride dental fluorosis will be accompanied by LEH. Amongst the skeletons examined, however, this relationship cannot be demonstrated since the frequency of hypoplasia on fluorosed teeth in the Bahrain sample was not statistically significantly greater than on normal teeth. It appears though, that in groups with dental fluorosis, growth disruptions are more likely to result in LEH. Thus care is needed in comparing the levels of hypoplasia in the Bahrain population with others. This does not, however, negate internal comparisons.

The levels of hypoplasia suggest that children may have experienced repeated infections or periods of malnourishment around 2–5 years of age. Hypoplasia has frequently been linked to weaning stress, with its attendant infections and malnourishment (Clarke 1980, Green 1982, McHenry and Schultz 1976). This attribution, however, is not without problems since the frequency of hypoplasia formation varies with tooth type. For instance, peak frequency of LEH on the canine (in current methods of recording) tends to occur between 4–5 years of age, on the incisor between 2–3 years. This could result in dramatically different weaning ages. Thus the age of weaning may possibly only be established by the multiple tooth method. In addition, the invisibility of early lines and inaccuracies in current chronological schemes, means that differences attributed to the age of weaning stress may merely reflect the period of maximum enamel susceptibility (Skinner and Goodman 1992).

In the Saar and DS3 groups the peak age of hypoplasia is around 3–5.5 years of age. However these episodes of repeated and frequent growth disruptions are initiated earlier: c 2–3 years, for those who survived adulthood, 1–2 years for those who died prior to adulthood. It is suggested, as argued previously (Littleton 1987), that age of onset of developmental defects may more closely correspond to the age of weaning, indicating an age of c 2–3 years for weaning. The link with dental fluorosis becomes important at this point since after weaning it is probable that a child's fluoride intake is increased enhancing the cyclical effects of fluorosis and LEH.

The resultant interpretation is that these two populations were subject to repeated episodes of stress, either infectious or nutritional, especially after 2–2.5 years of age. This pattern of disease and recovery continued till 5.5 years. Episodes of growth disruption, however, did not occur only after weaning. Examination of the subadult portion of the population demonstrates firstly that this group can be divided into two. There are those who never developed LEH (a higher proportion than amongst adults). This is unsurprising since LEH represents recovery from a period of growth disruption. Second are those who did develop LEH. These experienced more stress/recovery episodes than the surviving adults. However, not only did they experience more closely

repeated episodes than the survivors, they also experienced them earlier. A significant proportion of the group were suffering either nutritional or infectious disturbance between 1.5–2.5 years of age. Presumably this is a reflection of the pathogenic and nutritional environment of the society. It suggests that either a subgroup of society was less favourably treated during early childhood and subsequently died or that the population as a whole was subject to a stressful period of growth which lead to survival only of the fittest individuals. Since the effect is visible throughout every age group, the second scenario seems most probable, following the nontion of insult accumulation (Alter and Ridley 1989).

In both samples, there is little obvious difference between the sexes in the pattern of LEH. There is also little significant difference between Saar and DS3 although there is a tendency for individuals in the Saar population to have experienced their first stress episode between 2–2.5 years rather than 2.5–3 years and to have experienced stress for a narrower period of time during childhood. This could suggest a slightly different pathological environment; while the same episodes of stress were experienced, they were alleviated slightly earlier in the growth period. In turn this could tie in with the evidence suggesting slightly better mortality conditions in Saar.

It is intriguing to note that no deciduous enamel defects were found amongst these individuals despite high infant mortality. What appears to be happening is that during early childhood and infancy, the causes of death tended to be acute (such as diarrhoea). During weaning the same pattern continues with a high level of stress though possibly by this age it has become more chronic. There is no alleviation for those who subsequently died before six years of age. The severe and lingering effects of these episodes contributes to the mortality of older children and adults: the more episodes of growth disruption experienced, the greater the chance of premature mortality.

Examination of the patterns of growth, particularly in conjunction with these findings, should help in determining whether this is a valid interpretation.

Subadult growth

Subadult growth is subject to fluctuations caused by environmental effects, regardless of hereditary potential. Anthropometric variables are frequently used in assessment of the morbidity and nutritional status of modern societies. The same technique has been applied to skeletal samples (e.g. Hummert 1983). There are, however, four main problems which need to be recognised in any analysis of prehistoric growth.

Firstly, the data used in skeletal studies are, of necessity, cross-sectional rather than longitudinal. They are therefore not strictly comparable to modern longitudinal growth studies (e.g. Tanner 1978) which trace the course of growth of an individual. In skeletal populations there is no way of tracing individual patterns of growth retardation and catch-up growth.

More importantly there is no way of determining the chronological age of skeletons, so studies must rely upon dental ages which may be up to 3–4 years different from chronological age (Saunders 1992, Ubelaker 1987). This problem is partially avoided, however, by the necessity in many skeletal populations to use age groups rather than exact ages, particularly in higher cohorts where the variation between chronological and dental age could be assumed to be greatest.

Sex-specific patterns of growth are masked due to the inability to sex subadult remains. As a result there is reduced sensitivity in the method: it is impossible to determine whether the sexes are treated differently during childhood or have a different level of susceptibility. In terms of the actual growth curves pooling of sex probably makes little difference until adolescence when the growth spurt tends to occur earlier in females than males (Tanner 1978).

Finally, archaeological studies concentrate on that segment of the population which did not survive to adulthood. It is quite possible that growth in this segment is unrepresentative of the population as a whole. The degree of retardation appears to be related to the type of stress concerned, in particular acute versus chronic stress (Rowland et al. 1988; Saunders 1992). "Normal" growth in a skeletal sample may mask the fact that these children were subject to acute disease episodes, not necessarily experienced by the whole population.

These points need to be kept in mind and will be discussed further in the discussion of results.

Growth can be affected by stress in several ways: delayed maturation and an extended period of growth; episodic slowing of growth; decreased adult stature; increased variability of stature (Tanner 1978). Within skeletal groups, modifications in the timing and rate of growth are the most direct evidence for adaptation to stress. Delayed maturation is difficult to assess given the problems of ageing adult skeletons, while stature is also a reflection of hereditary potential, the effects of which are difficult to isolate.

Method

Subadult growth was recorded by standard long bone measurements of all subadult remains which could be aged by the degree of dental development. Sample sizes amongst the Saar skeletons were too small to be compared with the DS3 skeletons so the following discussion is related solely to DS3. The analysis is divided into two parts: foetal and neonatal size; and the growth of children.

Analysis is based upon comparison with other skeletal populations since these are aged by the same method rather than by chronological age. No direct comparison is conducted between modern chronological studies and the skeletal population. This factor, however, is incorporated into comparisons of velocity and the percentage of adult growth attained. Due to small sample sizes at older age groups, however, these latter analyses are restricted to the youngest ages where the convergence between chronological and dental age is greatest.

Growth the earliest period

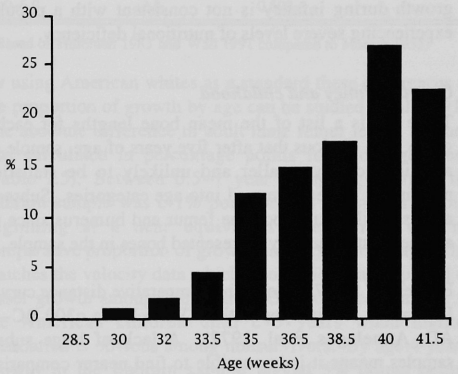
Foetal growth and the frequency of either premature or low birthweight infants are important indications of maternal health and environmental conditions. In addition premature and low-birthweight infants are at greater risk of dying within the first month of life or of experiencing later infections or nutritional disturbances which result in death during the first year (Puffer and Serrano 1973). Given this, it becomes important to know whether foetal growth was retarded, and whether any later evidence of slow growth amongst infants may reflect these conditions.

Unfortunately, as discussed in Chapter 5, the age of very young infants could not be obtained from dental age assessment. The only indication of subadult growth is the length of the long bones. Given that all infant bones were found separated from the skeletons of female adults, it is assumed that the very smallest bones represent actual births (though not necessarily live births). The category 'neonatal deaths' in this population includes, of necessity, possible stillbirths, premature and low-birthweight infants, as well as full-term live births.

It was argued in Chapter 5 that an average of c70 mm most probably represents a full-term birth. This figure is slightly lower than the average for modern populations for two reasons. Firstly, it is assumed that the most common length measurement amongst young infants is equivalent to the average length at birth (as per Stewart 1979). Secondly, there appears to be a historical tendency towards increasing size at birth (Tanner 1978). This approach may be unnecessarily cautionary. In studies of foetal growth, there is a general convergence around 75 mm as the average femur length at birth. As will be demonstrated later, there is no marked divergence between the Bahrain populations and others in growth attainment during the first six months of life. In addition, studies on populations with traditionally smaller sizes at birth demonstrate an increase in birthweight with improved nutrition (Lechtig et al. 1982). These factors all indicate the overwhelming impact of environment as opposed to heredity on size at birth.

Use of Scheuer and coworkers' regression of gestational age against the femur lengths recorded for Bahrain (Scheuer et al. 1980) demonstrates that most infants were between 38.5 and 41.5 weeks gestational age at the time of death (Figure 7.6). The remaining 33.7% were, by this calculation, less than 36.5 weeks gestation at the time of birth. These infants could be either premature births of full size for age, or low birthweight full-term births. As indicated earlier, in the absence of an independent method of ageing these conditions cannot be distinguished. Yet the implication is significant. In modern populations survivorship of premature but full size for age infants is close to that for full-size full-term births (Tanner 1978). On the other hand, low birthweight is a serious risk factor, not only in the first month of life, but for the first year (Puffer and Serrano 1973).

Figure 7.6
Distribution of infant femur lengths by age (wks). Estimated by Scheuer's regression*



*Scheuer et al. 1980)

Growth retardation amongst foetuses occurs most noticeably between c 33 weeks and 38 weeks gestational age (Sedaghatian et al. 1983; Tanner 1978). This tallies with the evidence from DS3 for the distribution of gestational ages, outside the full-term (38–41 week) period to be between 33.5 and 36.5 weeks. It could be assumed that the 3% of births of very small size (30–32 weeks gestation) are definitely premature infants who died, but those between 33 and 36.5 weeks are quite possibly low birthweight infants.

While this represents a significant percentage, an examination of modern populations indicates that this rate of immaturity amongst neonatal deaths (comprising low birthweight and prematures) is low. In the survey of South American childhood mortality, between 50–70% of neonatal deaths were of low birthweight infants (Puffer and Serrano 1973). Immaturity was an underlying or associated cause in over half of all neonatal deaths. These comparative figures are possibly inflated since they are based on hospital (not total) births. On the other hand, the Bahrain sample includes stillbirths while the comparative data are based on live births.

An accurate estimate of the rate of stillbirths, premature and low birthweight births is not possible for this skeletal population. Yet the comparison of associated mortality would seem to imply that, while immaturity was a significant

factor in neonatal mortality, its relative importance to mortality is less than in modern countries experiencing widespread nutritional deficiency. This is supported by the pattern of growth during infancy.

Alternatively, it could be suggested that, while the frequency of low birthweight was high, neonatal deaths are swamped by causes unassociated with low birthweight so that neonatal deaths are not biased towards immature births as in modern populations. There is, however, a close relationship between immaturity and the majority of other causes of infants death (one of the few exceptions is difficult labour), therefore it would be difficult to conceive of a cause of death sufficiently severe to cause this level of neonatal mortality which does not predilect low birthweight babies. Secondly, the pattern of growth during infancy is not consistent with a population experiencing severe levels of nutritional deficiency.

Growth infancy and childhood

Table 5.1 is a list of the mean bone lengths for each age group. It is obvious that after five years of age, sample sizes are significantly smaller and unlikely to be statistically meaningful unless grouped into age categories. Subsequent analysis will focus upon the femur and humerus since these are the most frequently represented bones in the sample.

Figures 7.7 and 7.8 shows the comparative distance curves of DS3 and a Nubian population (Wadi Halfa c300 BC–1500 AD; Armelagos et al. 1972). A lack of large subadult samples means it is impossible to find nearer comparisons. Significantly, there is little difference between these populations in mean adult stature suggesting that the differences in subadult growth among non-survivors are reflections of different rates of growth, not merely scale, although differing ageing standards may also play a role.

Figure 7.7
Comparative subadult growth for humerus

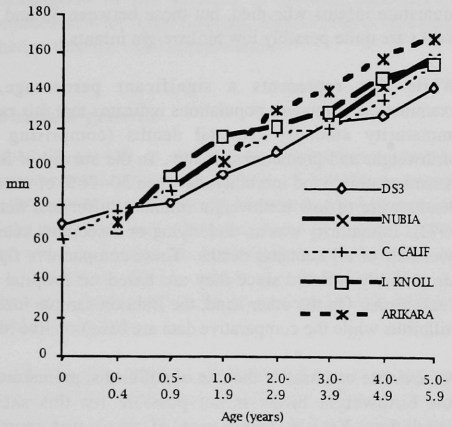
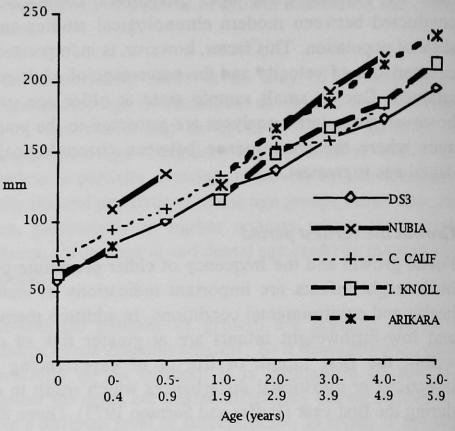


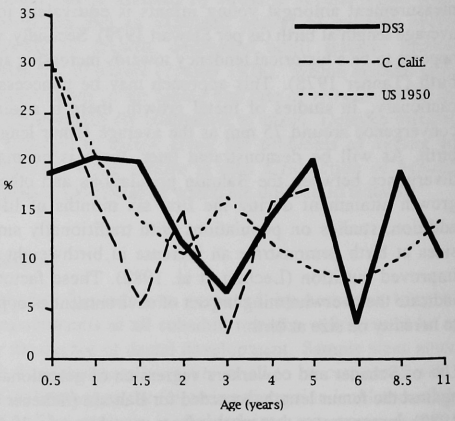
Figure 7.8
Comparative subadult growth for femur



At every age, especially above 3.5 years, the DS3 population lags behind despite the similarity of final height. Growth during infancy follows a similar curve for both populations, but the Bahrain group shows a slight lag at 1.5 years and a marked flattening of the curve between 4–6 years of age. A comparison of the means shows no significant differences between 0.5–4 years, but significant differences ($p<0.025$) at 4–5 and 5–6 years. After six years sample sizes are too small to test statistically.

Humeral growth (Fgure 7.7) shows the same lag at ages 1.5 and 3–6 although less obviously. This possibly mirrors the greater susceptibility of the lower limb bones to growth retardation (Buschang 1982, Tanner 1978).

Figure 7.9
Growth velocity of subadult bones



The rate of growth for the femur was calculated by converting the increment in length between the two age

groups into a percentage of growth attained/age cohort (Figure 7.9, Johnston 1969). Growth velocity is marked by an initial and sustained high, with a gradual slowing occurring until 3–4 years then a period of marked acceleration until 5–6 years, and then another decline (most marked in the femur, probably a reflection of small sample sizes). During the 7–10 year age period there is marked acceleration which flattens out slightly after this age.

A comparison of the rate of growth pattern with a modern white American sample (Maresh 1955) demonstrates the difference in growth of an optimally nourished group (figure 7.8). The velocity of growth in modern American males shows a gradual decline from a peak at 0.5–1.0 year of age. There is no evidence of the fluctuations in early childhood and the mid-childhood peaks as seen in the Bahrain sample of nonsurvivors.

Owing to the use of different age categories it was not possible to compare the velocity of growth on Bahrain with that observed in Nubia. Native American samples (Wall 1991), however, like Bahrain, show a slowing of growth around 2–3 years and the Arikara have a second slowing around 6–7 years as in the DS3 population. This appears to be mapping a similar pattern where in prehistoric groups decelerations at ages 2–4 and 6–7 are compensated for by marked acceleration. This is either a reflection of retardation and catchup growth, experienced by the whole population, or it is the evidence of those who died between ages 2–3 and 6–7 experiencing markedly slower rates of growth prior to death than those in successive age groups. Comparison with the distribution of pathological lesions, such as hypoplasia, may help to resolve this dilemma.

There is one major divergence between the DS3 sample and other skeletal samples: the pattern of growth during infancy. Despite the similarity between the populations in the distance achieved at early ages, it is obvious that growth during the first year, particularly the second six months of life, was markedly slower in the DS3 group than in other groups. This slow growth rate is not compensated for by a following period of acceleration. Rather between 1.5 and 2 years the rate of growth of children on Bahrain begins to mirror other archaeological groups. In addition to the decceleration of growth at c 3–4 years and again at 6–7 years which is indicative of periods of stress, or of periods where those with slowed growth are likely to die, growth of dying infants at DS3 was slow. This indicates that in Bahrain the second six months of life were particularly stressful, possibly either because of nutritional inadequacy or the onslaught of infection. The period of slow growth does not persist beyond the first year and after 1.5 years the fluctuations of growth rates are similar to those of other populations.

In order to account for genetic differences between groups, the rate of growth was also calculated as the proportion of adult femur length attained at each age (AFLA; following Mensforth 1985; Wall 1991).

Table 7.5
Absolute difference in AFLA

Age	Bahrain	Wadi Halfa	Central Calif.
0.0–0.4	0.6		-1.3
0.5–0.9	-1.5	0.242	-1.1
1.0–1.5	-1.9	0.299	0.9
1.5–1.9	-0.8		0.0
2.0–2.9	-0.5		-0.4
3.0–3.9	-5.3	0.36	-5.2
4.0–4.9	-3.2	0.428	-4.2
5.0–5.9	-7.0	0.486	-1.7
6.0–6.9	-2.8	0.503	

*(Based on Hummert 1983 and Wall 1991 compared to Maresh 1955)

By using American whites as a standard these differences in the proportion of growth by age can be studied (Wall 1991). The absolute difference in adult male femur length attained was calculated in percentage points for each age group (Table 7.5). Between 0.5–1 year of age, growth in the Bahrain sample was c 1% point behind the whites despite beginning at a near equivalent rate. There was no comparative proportion of growth until 1.5 years of age. This matches the velocity data which also demonstrate a period of lesser growth among late infants. Growth is comparable to the American children until c 3 years when marked retardation is obvious among nonsurvivors. By age 4 years, growth in the Bahrain sample lagged 5% points behind modern Americans, similar to the Central Californian sample. A second catch-up was visible at 6 years. By age 10 those who died at DS3 had only attained 64.6% of their adult femur length, compared to 71.1% in modern Americans. To isolate, however, whether the lag is due to sampling a chronically stressed group or to delayed growth in the whole population is impossible at the moment.

Overall the growth data, despite their limitations, do indicate slowed growth in the second half of the first year, decceleration of growth at age 3, with continued lack of recovery between 4–5.9, catch-up between 6–6.9 years, and possibly a second deceleration in mid-childhood, c 7–10 years, followed by a growth spurt. This pattern of growth differs from other groups, both archaeological and modern, in several respects: the lack of growth in the first year of life appears to be in response to particular stressors, either nutritional or infectious. As in other groups the ages around 3–5 seem to be stressful. Frequently this is ascribed to weaning (e.g. Wall 1991). In the Bahrain group such an attribution cannot be easily made since the effects of the decceleration appear to persist until 6 years of age. It suggests that the period of 'weaning stress' is unusually long or that the environment on Bahrain placed special burdens upon children of this age group. Finally it is possible that maturation in the group was delayed, or that chronic stress played a large role in the cause of death amongst children dying in late childhood/ early adulthood.

One comparison in the pattern of growth is, however, particularly close. This is the growth of children in several West African countries. In these populations, despite rates of growth similar to U.S. norms in the first six months of lfie, faltering occurs markedly in the second six months. In numerous groups this persists until 1.5 years. Thereafter, although annual increments tend to be similar to western norms, the leeway is not made up and the growth curve after two years runs parallel to these standards but well below (Rowland et al. 1988). A strong seasonality, less in height than in weight, is also visible in these populations (Rowland et al. 1988). A persistent difference between these groups and Bahrain is the slowed growth in Bahrain between 3–6 years.

A factor in these differences may be the bias inherent in measuring dead rather than living children. If it is assumed that small body size is associated with an increased risk of death, then a mortality sample (eg. an archaeological collection) will be, by its very nature, smaller than a sample of living subjects. However, these discrepancies may be too small to measure and work in Keneba, West Gambia, demonstrates that up to 3 years of age there is no significant difference between the weight and height of those who died and the survivors (Rowland et al. 1988). This reflects the lack of effect of acute disease episodes upon growth and the prevalence of chronic conditions. Above the age of three, however, there is no evidence concerning differences in height between survivors and nonsurvivors, therefore the dilemma still exists: did the whole population of DS3 or only a subset experience growth disruption? A comparison of the patterns of enamel hypoplasia with growth will address this issue.

The evidence of childhood growth demonstrates that a significant percentage of births were of preterm or small for age babies, although this figure is less than 50% of those infants who died at childbirth and is lower than in modern populations experiencing widespread nutritional deficiencies. In addition there was a period of slowed growth occurred between 0.5–1 year of age despite 'normal' rates of growth for the first six months and a longer period of growth retardation occurred between 3 to 5 years of age followed by partial recovery among nonsurvivors. Overall the population possibly experience delayed maturation or else chronic stress resulting in growth retardation. This retardation is significant when the mortality of late childhood and early adolescence is considered.

Conclusion

The relationship between linear enamel hypoplasia and subadult growth is not straightforward. This is not unexpected since growth disruption frequently is a response to chronic stress, while hypoplasia is the sign of recovery frequently from shorter periods of stress, possibly from as little as several days to two months (Rose et al. 1985:289). In addition, the information on subadult growth represents those children who died. As an examination of LEH demonstrated, those who failed to survive until adulthood suffered more episodes of stress resulting in hypoplasia in early childhood than those who did reach adulthood. It is therefore possible that the picture of subadult growth is not representative of the population as a whole. In this instance, the most accurate comparison is then between those subadults experiencing LEH and the pattern of subadult growth, and only secondly with the overall pattern of linear enamel hypoplasia.

When the age period covered (0–6 years) is analysed in yearly increments, a pattern becomes discernible. For the first six months of life growth appeared normal. From 6–12 months, long bone growth in nonsurvivors was marked by slow velocity and a drop in the percentage of adult femur length attained. This period is not matched by enamel hypoplasia since this is the least susceptible period of enamel to growth disruption.

Between 1–1.5 years, growth velocity was 'normal' or slightly above, compared with other populations, and there appears to have been a period of slight catch-up growth. Again there is only limited evidence of hypoplasia as this age.

From 1.5–2 years growth velocity declined slightly again but relative growth is sustained. A significant proportion of subadults began to develop enamel defects during this period, while this age period marks the initial onset of enamel hypoplasia amongst those who did not survive until adulthood.

During 2–3 years of age growth velocity continued to drop, with a gradual slowing compared to other populations. The number of hypoplasic lines amongst subadults continued to increase during this period. Amongst the survivors, 2.5–3 years marks the true onset of hypoplasic defects. This suggests a convergence of the pattern of growth between those who died early (as represented by the growth curves) and those who survived (represented in the analysis of hypoplasia).

The slowing of growth between 2–3 years continued between 3- 4 years with a slight pick-up. For both survivors and non-survivors this was the peak age of hypoplasia and the pattern of repeated stress episodes and growth disruption developed during 2–3 years continued.

From 4–6 years hypoplasia continued, and at the same age children dying demonstrate slow rates of growth. Recovery in growth finally occurs around 6 years. By this stage LEH is no longer a sensitive indicator of stress.

It appears there are two definite periods of rapid growth: between 0–2 years, and 2–5 years. Between 0–2 years, part of the population appeared to be suffering episodes of both acute and chronic stress. This was particularly centred on 0.5–1.5 years. This pattern of disruption ameliorated slightly between 1.5–2 years. After 2 years however, the majority of the population experienced a slowing of growth and more

frequent episodes of stress. This continued until 5 years of age when recovery in growth occurred. Even so, episodes of acute stress continued until 6 years of age.

A simple attribution to weaning stress as a transitory phenomenon is not possible. Firstly it is evident that those who died before 1.5 years of age were affected by episodes of growth disruption. The general pattern of growth retardation and hypoplasia between 2–4 years appears to have affected everyone and matches the pattern of 'weaning stress' seen in other prehistoric and historic groups (e.g. Hummert 1983; Milner 1982). In Tylos period Bahrain, however, recovery from this period is delayed with chronic stress, as represented by growth retardation, unalleviated until c 6 years of age, shorter episodes of disruption indicated by LEH also did not cease until this age.

The pattern of disruption seen in these samples seems to be similar between the two populations from DS3 and Saar. It is also highly specific to these two particular samples indicating a pattern of repeated stress throughout the first six years of life. Certain ages were particularly stressful with a high impact on mortality of a section of the population. At other ages the stress is more widespread. Analysis of specific pathological conditions should help to elucidate the causes behind the morbidity outlined by this examination of developmental defects.

Chapter 8

Step 3 cont.: Specific pathology

Some pathological conditions are more clearly associated with specific diseases. Differential diagnosis remains a difficult task. Yet even if all that can be achieved is a narrowing of the possible causes of skeletal lesions, we have at least come closer towards reconstructing the interaction of society and disease.

While the recording of the Bahrain material included resorptive, proliferative and traumatic lesions plus neoplasms, in reality not all of these conditions were found. During analysis a limited variety of pathological lesions were observed. These were: porotic hyperostosis (recorded as resorptive lesions); infectious lesions (primarily proliferative); bone deformity (recorded within resorptive lesions); and sclerosis with bone hypertrophy (recorded as a separate category). Less common conditions such as two cases of benign osteomata were observed. These are not included in the following discussion which concentrates upon those conditions associated either with significant mortality or morbidity in the two samples. Each of these conditions is described and analysed in turn. In Chapter 9 their combined impact on mortality is assessed.

One condition not discussed, yet recorded, is osteoporosis. The lack of inclusion is due to difficulties in recording the condition in a meaningful way. Cortical thinning and crush fractures of vertebrae were recorded, and on as many skeletons as possible the percentage of cortical bone was calculated. The lack of complete skeletons that could be both aged and sexed, however, means that the results are insufficient at this stage.

Porotic hyperostosis

Introduction

The term porotic hyperostosis is used to refer to porosity occurring in the outer table of the skull with attendant hyperplasia of the diploe (Angel 1967). The related condition, cribra orbitalia, refers to the same process occurring in the orbits (Stuart-Macadam 1989). Generally the orbits, parietals and occipitals are the most affected bones (Mensforth et al. 1978) although these are by no means the only bones to be altered. By radiology it can be seen that the process is one of thickening of the diploe with resultant thinning of the outer table. In more severe cases the trabeculae form a striated pattern resulting in 'hair-on-end' appearance (Reimann and Kuran 1973).

This definition of porotic hyperostosis includes porotic lesions of the orbits (cribra orbitalia) as well as proliferative lesions on the skull. Porotic lesions of the postcranial skeleton are described under the more general desciption of porosity.

Resorptive lesions included conditions marked by thinning of the outer cortex. Porotic hyperostosis was coded following the scheme of Nathan and Haas (1966). The final categories were:

1. porosity without swelling;
2. porosity/cribrotic with slight swelling;
3. trabecular with swelling; (these apply to cranial bones only)
4. lattice work cortical thinning (applying only to postcranial bones).

Cranial lesions

Cribra orbitalia and porotic hyperostosis were commonly found in the two samples. Forty percent of all individuals at DS3 (n=492) and 54% of those at Saar (n=48) had porotic lesions in the orbits (p=n.s.). Apart from the orbits, parietal lesions were most common (Table 8. 1).

Table 8.1
Frequency of protic hyperostosis by element

	DS3		Saar	
Element	**%**	**N**	**%**	**N**
Orbits	40	492	54	48
Parietals	25.3	249		
Maxilla	18.9	90		
Frontals	13.8	196		
Occipital	9.8	204		
Mandible	8.9	124		
Basicrania	7.9	101		
Temporals	7.7	181		

These lesions occur in distinctive patterns. In the case of parietal lesions, the area of reactive bone was generally located along the middle of the lambdoidal suture. A circular area of lesion was often found in this location with, much less commonly, a second circular lesion towards the frontal suture. With larger lesions the entire parietal could be affected, generally from the back towards the front, and only ever above the temporal line (Plate 4). Lesions were initially porous in nature with reasonably large holes penetrating the outer table. Even at this stage, however, traces of bossing were frequently visible. Larger lesions were often more vascular in nature, in extreme cases showing clear trabeculation and extensive thickening of the bone due to diploic expansion. This thickening was restricted to the bone above the temporal line, and generally did not affect the sagittal suture. Generally, only the outer table was affected.

Cross-sections of skulls have clear 'hair-on-end' appearance, the expanded diploe being arranged perpendicular to the

skull and the outer table obviously perforated (Plate 5). In cases where healing was apparent, the individual holes are still clearly delineated, with an obvious 'filled in' appearance, frequently with the cranial thickening still visible. The appearance of these healed lesions differs significantly from the superficial 'pinpricks' sometimes observed in the skulls of old adults.

Facial bones were affected differently. Apart from porosity which affects the entire maxilla, the maxillary sinuses are also altered. Amongst infants marrow hyperplasia of the maxillae is common, at times the sinuses are completely blocked. More generalized swelling of the facial bones is not, however, apparent.

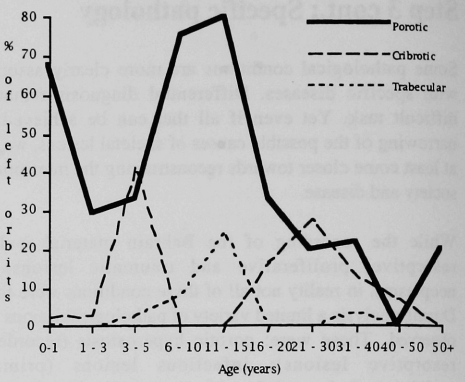
Table 8.2
Frequency of porotic hyperostosis amongst subadults, DS3

Age (yrs)	Cribra	P.H.	Occip.	Pariet.	Front.	Face
0.0–0.5	8.7	8.3	8.3	0.0	23.1	0.0
0.5–1.0	55.9	56.5	14.3	47.6	33.3	21.4
1.0–1.5	53.3	57.1	12.5	60.0	22.2	16.7
1.5–2.0	87.5	76.9	62.5	75.0	50.0	57.1
2.0–3.0	100.0	71.4	0.0	71.4	100.0	100.0
3.0–4.0	88.9	90.9	20.0	77.8	33.3	57.1
4.0–5.0	100	71.4	60.0	75.0	25.0	75.0
5.0–6.0	80.0	50.0	50.0	50.0	50.0	0.0
6.0–7.0	100.0	100.0	100.0	50.0	100.0	100.0

Table 8.2 demonstrates the age distribution of cribra orbitalia and porotic hyperostosis amongst the skeletons at DS3. A small proportion of those who died in the first year of life ahd signs of porotic hyperostosis. The frequency rose steadily to 100% of those who died at age 6. From 10 years on there was a decline in the frequency of the condition. All lesions were seen on at least a small proportion of infants, although most frequently among those who died in the second six months of life.

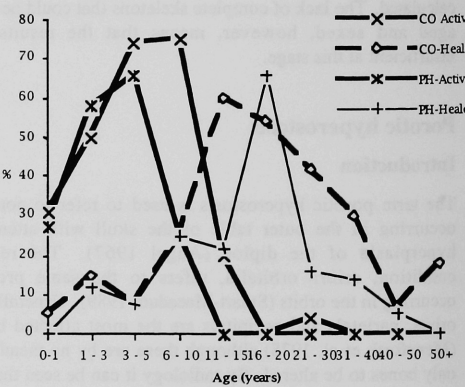
Cribra orbitalia was much more commonly found amongst the skeletons than the cranial lesions. This higher frequency persists into adulthood and is commonly observed in skeletal populations (Stuart-Macadam 1985). The age pattern of the two lesions is nonetheless extremely similar, especially under six years of age. This is supported by examining the relationship between cranial and orbital lesions in the same individual. Out of 235 cases of porotic hyperostosis only 6 did not have evidence of orbital lesions. On the other hand 69 individuals had evidence of cribra orbitalia with no accompanying cranial lesions (p=0.00) This relationship held regardless of which part of the cranium was compared to the orbits. It is apparent that in this population, at least, cribra and the cranial lesions probably reflect the same pathological process, one which most affects young children from 6 months of age to about seven years.

Figure 8.1
Severity of porotic hyperostosis, DS3



Severity of lesions was scored and the resultant pattern of age and severity is similar in both cribra orbitalia and porotic hyperostosis. A small percentage of infants had cribrotic or trabecular lesions on the parietals and orbits. This frequency increased until by three years of age severe lesions were more frequently represented among the dead than the milder porosity. After six years, the frequency of severe lesions stedily declines. This pattern of severity supports the evidence that porotic hyperostosis was most significant in deaths between one and six years.

Figure 8.2
Active and healed lesions, DS3



Examination of remodelled and active lesions supports this assumption (Figure 8.2). After 10 years no active parietal lesions and very few orbital lesions were found. Only three adults (aged 20–30 years) still had active cribra orbitalia at the time of death. The transition from active to healing states in the dead occurred between six to ten years of age.

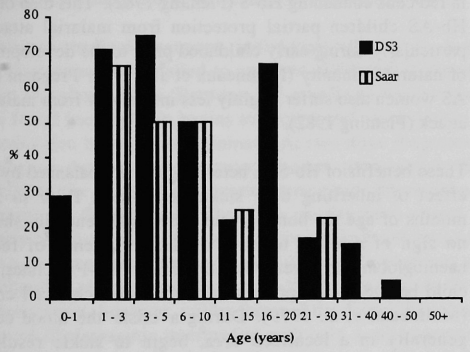
The lack of active cases after seven years indicates that cranial lesions are evidence of a childhood disease episode.

Differences between the sexes can be taken as reflecting either inherent differential susceptibility or possible differences in the childhood treatment of males and females. More females than males were affected by cribra ($p>0.01$; Table 8. 3) but there was no statistically significant difference in the frequency of calvarial lesions.

Table 8.3
Sex and cribra orbitalia, DS3

	Males		Females	
Age (yrs)	%	n	%	n
DS3:	37	78	50	88
15–20	50	4	75	4
20–30	57	7	63	27
30–40	67	15	31	16
40–50	33	12	18	11
50+	20	10	0	2
Saar:	35	23	59	27

Figure 8.3
Porotic hyperostosis at Saar and DS3



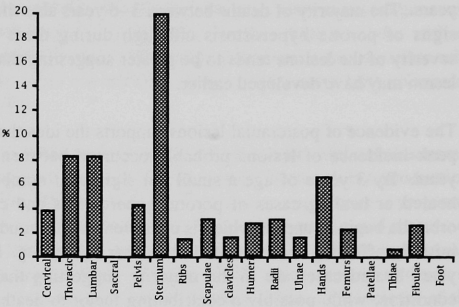
The frequency of cribra orbitalia is not significantly different between Saar and DS3 but significantly fewer cranial lesions were observed at Saar ($p=0.029$) (Figure 8.3). The difference in cranial lesions holds even when subadults and adults are considered separately although the results are no longer statistically significant. Reflecting the lower prevalence at Saar, no cases of facial, basicranial nor temporal involvement were found. Similar age and sex patterns were observed, however.

Postcranial porosity

A much smaller percentage of children had postcranial lesions of porotic hyperostosis (Plate 6). These lesions were identified by excessive thinning of the cortical bone leading, in severe cases, to a lattice like appearance of the affected bones. The skeleton was not evenly affected by porosity.

Figure 8.4 shows the distribution of porosity amongst the DS3 subadults (less than 6 years).

Figure 8.4
Postcranial porosity, DS3



Of individual subadults, seven had evidence of postcranial porotic hyperostosis (8%). These were one infant 0–6 months, one infant 6–12 months, one child 12–18 months, one young child c 12 months, one child 18m–2 yrs, a child 2 –3 years, and a child 4 years. Of the minimum six individuals affected who could be determined from multiple tombs, all were aged between 0 and 3 years, the majority less than 1.5 years (as judged by long bone length)

In all seven individual cases, evidence of porotic hyperostosis or cribra was seen on the crania. The youngest infant (0–6 months) had markedly porous temporal and facial bones but no cribra orbitalia, while one child (12–18 m) had no cranial lesions, only slight cribra orbitalia. The remaining 5 skeletons all had extensive cranial lesions and cribra orbitalia.

This connection between postcranial and cranial lesions is statistically significant ($p<0.001$). The number of cases, however, is too small to see any clear age related trends beyond the obvious fact that only a restricted age group was affected. Certainly the condition occurred early in life: in the first six months in one instance. No individuals who died after four years showed signs of postcranial lesions. In addition no healed lesions were observed. This suggests either complete remodelling (contradicted by evidence of remodelling in other populations) or that the condition represented by these postcranial lesions caused 100% mortality.

Despite finds of subadults with extensive cranial lesions at Saar, no postcranial lesions were found. Given the small size of this sample the difference is not statistically significant.

Summary of results

These three types of lesions appear to be closely related in the DS3 sample: postcranial lesions are found only amongst those with some form of cranial involvement, while cranial

lesions are connected with the presence of cribra orbitalia. In addition the age pattern of these lesions is closely related: a small number of cases appear amongst those who died between 0–6 months, although not amongst neonates. A significantly greater number are found between 6–12 months but the majority of lesions affect those who died between 1–3 years. The majority of deaths between 3–6 years also showed signs of porotic hyperostosis although during these ages severity of the lesions tends to be greater suggesting that the lesion may have developed earlier.

The evidence of postcranial lesions supports the idea that the peak incidence of lesions probably occurred between 1–3 years. By 3 years of age a small but signifcant number of healed or healing cases of porotic hyperostosis and cribra orbitalia begin to appear which is consistent with a condition initially affecting those in the 0.5–3 year age group. By 7 years healed cases are in the majority suggesting that the condition, while possibly a contributing factor to deaths, no longer reflects an immediate cause of death. The persistence of active lesions amongst a small number of adult females who died between 20–30 years also implies that the condition continued from childhood at least for these females.

Comparing the two cemeteries, it is apparent that the same syndrome occurred in both areas of the Island, affecting similar age groups. The evidence, however, points to a lower frequency and lesser severity of lesions amongst those buried in Saar Mound 5.

A possible diagnosis?

Until the seventies the aetiology of this condition was disputed, particularly in the case of cribra orbitalia. Inflammation of the lacrimal gland was originally suggested as a cause by Moller-Christensen (1953), who later suggested that the condition might be the result of the interaction of environmental stresses such as malnourishment and anaemia (Moller-Christensen and Sandison 1963). Other authors (Angel 1964, Hengen 1971, Nathan and Haas 1966) have come to similar conclusions. In particular, Angel pointed to the link between porotic hyperostosis and the congenital haemolytic anaemias, especially thalassaemia (Angel 1971, 1978). Yet even when doing so he still emphasised the possible role of nutrition in the pathology (Angel 1967).

Skull changes similar to those of the congenital anaemias are found in chronic iron-deficiency anaemia (eg. Lanzkowsky 1968) and this is most frequently interpreted as the cause of porotic hyperostosis in populations historically unaffected by abnormal haemoglobins (El-Najjar 1976, Stuart-Macadam 1991a). Disputes now centre over whether the iron deficiency is nutritional or due to infection (Reinhard 1990, Hern 1990, Stuart-Macadam 1991, 1991a).

The conditions which induce such marked skeletal changes are:

1. Sickle cell disease (and trait?).
2. Thalassaemias.
3. Hereditary spherocytosis.
4. Pyruvate kinase deficiency.
5. Polycythaemia vera and secondary polychythaemia.
6. Iron deficiency anaemia (Ortner and Putschar 1981).

Attribution of lesions to one or other of these causes requires consideration not only of the lesions produced by each disease, but also their frequency and pattern, the associated age distribution of mortality and morbidity, population incidence, and geography.

The congenital haemolytic anaemias include two major conditions: the thalassaemias and sickle cell anaemia. These disorders of haemoglobin synthesis are caused by the inheritance of a recessive gene affecting production of either the alpha or beta chains in haemoglobin.

Sickle cell anaemia?

Sickle cell anaemia is an inherited condition, generally occuring in malarial areas. A heterozygous individual (Hb-AS) has greater protection than other people from malaria. Hb-S operates in two ways: firstly parasitised red cells when deoxygenated sickle causing destruction of the parasites, and at the same time growth of the parasite tends to be inhibited in red cells containing Hb-S (Fleming 1982). This then offers Hb-AS children partial protection from malarial attacks, particularly during early childhood prior to the development of natural immunity (Molineaux et al. 1979). Pregnant Hb-AS women also suffer slightly less intensively from malarial attack (Fleming 1982).

These benefits of Hb-S in heterozygotes are balanced by the effect of inheriting both sickle cell genes. Prior to 5–6 months of age the homozygous individual generally shows no sign of disease, because of the persistence of foetal haemoglobin. However, when the level of Hb-F declines, the child becomes susceptible to anaemia and sickle cell crises (Molineaux et al. 1979). During a 'crisis' the blood cells, generally in a localized area, begin to sickle resulting increased blood viscosity, stasis, hypoxia and tissue infarction which then becomes a cycle (Kotoney-Ahulu 1974). In infancy most infarctions occur in the hands and feet causing 'hand-foot syndrome'. Up to 90% of homozygotes less than two years of age suffer from this syndrome which generally clears, but may be complicated by osteomyelitis (Effong 1982). In older children the site of infarcts is more commonly in the long bones and soft tissues (Effong 1982).

The most common cause of a crisis is infection, particularly an attack of malaria (Kotoney-Ahulu 1974). Thus mortality tends to be greatest during early childhood when infections are most common. For example, in the Garki district, Nigeria prior to medical intervention, while 2% of infants had Hb-SS, only 0.4% in the 1–4 year age group had the disease, and this had decreased to 0.05% by 9 years of age, none survived to maturity (Molineaux et al. 1979). As Fleming states

> wherever a haematology laboratory or a sickle cell clinic are first opened in an area of tropical Africa previously untouched by modern medicine, not many Hb-SS patients will be seen who have reached puberty, these few will be in poor health and show many of the acute and chronic complications of sickle cell disease (Fleming 1982:73).

It should, however, be pointed out that reports of survival rates show wide variability in the course of the disease (e.g. Perrine et al. 1981).

The basic changes to bone in homozygous individuals are the result of chronic anaemia and infarction.

Beta-thalassaemia?

Beta-thalassaemia is also accompanied by severe bone changes. The condition is recessive, causing major changes in homozygous individuals (Beta-thalassaemia major) with heterozygotes only marginally affected (Knox-Macauly 1982). The erythroid marrow of patients proliferates to 10–20 times normal, expanding into the most peripheral limits of the skeleton (Pootrakul et al. 1988). Despite this, the circulating red blood cells are still reduced to a quarter or less resulting in severe chronic anaemia. Like sickle cell disease the condition is apparently connected to malaria, although no direct mechanism has so far been proven (Kazazian 1990).

Prior to medical treatment, individuals affected by beta-thalassaemia major failed to survived childhood (Lehmann and Huntsman 1966). Between 3–5 months of age the level of foetal haemoglobin begins to decline and haemoglobin A production becomes predominant. At this stage progressive anaemia develops which soon becomes pronounced. The children tire quickly and experience recurrent infectious and nutritional disturbances. These are usually most severe in early childhood. The child who survives this period improves to a certain extent but maturation and sexual development are delayed (Lehmann and Huntsman 1966). Along with these severe cases, however, milder forms of the disease have been found (thalassaemia intermedia) with only a mild anaemia. This can occur in individuals with a persistence of high foetal haemoglobin levels (Huisman and Jonxis 1977).

Heterozygotes are significantly less affected, although the severity of symptoms can vary widely from mild to moderate anaemia. Beta thalassaemia minor, however, may become a severe haemolytic anaemia when extra strain such as malnutrition, parasitism, or chronic infections occur (Lehman and Huntsman 1966). Similarly the condition may be exacerbated in pregnancy.

The bone changes associated with beta-thalassaemia major are marked and result from marrow hypertrophy.

Alpha-thalassaemia?

The other class of thalassaemias consists of abnormalities of alpha chain synthesis. Inheritance patterns of the alpha thalassaemias, however, are by no means as straightforward as for beta-thalassaemia. There are four basic states. The silent carrier has three functional genes for alpha chain synthesis and is thus unaffected. Alpha-thalassaemia trait is seen in individuals with two functional genes; again there is minimal effect except possibly marginal anaemia. Haemoglobin-H sufferers have only one functional gene, and Barts hydrops, or Hydrops foetalis, cases are those with no functional genes. This latter condition is incompatible with life; most are stillborn or die shortly after birth (Kazazian 1990).

The heterozygous states (silent carrier, alpha-thalassaemia trait) are unaffected with normal haemoglobin production, while those with Hb-H disease experience symptoms somewhere between beta-thalassaemia minor and major. As in cases of beta-thalassaemia minor, the patients may have a pernicious slight anaemia which is accentuated at times of stress (Lehamnn and Huntsman 1966). Hyperplasia of the bone marrow may occur in these individuals but is generally not sufficiently severe to produce the characteristic and extensive changes seen in beta-thalassaemia major (Huisman and Jonxis 1977).

Other possible causes

Two other congenital conditions are associated with development of porotic hyperostosis: pyruvate kinase deficiency and hereditary spherocytosis. The onset of pyruvate kinase deficiency is at birth. The condition is associated with high mortality. Skeletal changes include bossing of the frontals and parietals although only a small percentage demonstrate thinned cortices, and the sinuses remain unaffected (Becker et al. 1973). Hereditary spherocytosis is a genetically determined haemolytic anaemia in which the erythrocytes are spheroid in shape and vulnerable to destruction. Bone changes occasionally occur but are generally limited to the diploe, rarely affecting the long bones (Ortner and Putschar 1981; Powell et al. 1965).

Two other uncommon conditions causing porotic hyperostosis are polycythaemia vera and secondary polycythaemia which cause an oversupply of red blood cells. Cortical defects are present in the long bones. These changes do not occur in all individuals but only in severe cases. The average age when these changes occur is 12 years although the age of sufferers ranged from 8–43 years (Ascenzi and Balistreri 1977; Ortner and Putschar 1981; Powell et al. 1965).

Iron-deficiency anaemia?

The most common condition of all is iron-deficiency anaemia. Amongst third world countries today this is most common particularly amongst infants, young children, and reproductive women. The condition develops when there is insufficient iron for the haemoglobin in the new red blood cell. This may arise due to dietary factors or inadequate dietary intake of iron; excessive intake of compounds which

inhibit the absorption of dietary iron (for eg. phytates; El Najjar 1976; Reinhold 1972); prolonged breast feeding and inadequate weaning diet (Jelliffe and Blackman 1962). Alternatively it can be the result of infection. During some infections the body sequesters iron in the liver as part of the body's defense (Weinberg 1974; Scrimshaw et al. 196). In parasitism, particularly with attendant blood loss as in hookworm infestation, anaemia may develop (Scrimshaw et al. 1964) and is also common in malabsorption states such as weanling diarrhoea (Gordon and Scrimshaw 1965). Finally increased demands for iron may not be met, for example the anaemia of prematurity or during pregnancy and lactation (Scrimshaw et al. 1964, Smith 1954, Manir and Khaleque 1969).

Naturally many of these causes are synergistic. In an individual with an iron-deficient diet, an infectious episode may tip the balance in favour of iron-deficiency anaemia (Stuart-Macadam 1991a). Generally today iron-deficiency anaemia due solely to an inadequate diet is uncommon, and most frequently the anaemia is produced by the intervention of a second factor such as infection, including parasitism (Passmore and Eastwood 1986).

The response of the body to anaemia is to produce more red blood cells. In young individuals with large deposits of haematopoietic marrow both in the long bones and skull, this causes marrow hyperplasia with attendant bone changes if the condition is chronic and relatively severe (Stuart-Macadam 1989). Generally in adolescents and adults the amount of marrow in the body can cope with increased demands and bony changes no longer occur (Stuart-Macadam 1985).

The exact frequency with which bone alterations occur in iron deficiency is not known. Two studies (Reimann and Kuran 1973; Agrawal et al. 1970) suggest that changes are not uncommon given chronic and severe anaemia. The majority of studies, however, are based upon individuals presenting for treatment or in hospitals, rather than large scale surveys, so these frequencies are probably elevated.

In a large study by Agrawal and coworkers (Agrawal et al. 1970), the most common lesion amongst individuals with radiological changes was osteoporosis of the long bone shafts (100%). Ninety per cent presented with atrophy, osteoporosis and thinning of the hand cortices, 46% had destruction of the trabeculae, 7% had coarse trabeculation, 4% 'hair-on-end' appearance. This suggests that long bone changes are reasonably common; several other studies, however, have failed to find any long bone changes (Shahidi and Diamond 1960; Britton et al. 1960). While the evidence overwhelmingly points to long bone changes, it should be noted that the majority of these are osteoporotic rather than the advanced or severe changes seen in thalassaemia major (Ortner and Putschar 1981). In addition it must be pointed out that both Agrawal and Lanzkowsky's series contained individuals with concomitant rickets and protein malnutrition, both conditions which can cause thinning of the cortices (Agrawal et al. 1970; Lanzkowsky 1968).

In the skull, changes indicative of anaemia also occur. There is an increase in the diploic space, and in some cases 'hair-on-end' appears. This occurred in 5% of cases with bone changes in the study by Agrawal et al. (1970). In a small survey by Aksoy and co-workers (Aksoy et al. 1966), 5 of 12 patients had mild to moderate expansion of the diploe but none had clear 'hair-on-end' appearance and no facial changes were apparent. Facial involvement, however, was visible in 33% of the group studies by Reiman and Kuran with deficiencies in form, size or aeration of the frontal sinus; 50% had abnormalities of the maxillary sinuses (Reimann and Kuran 1973).

It can be seen that there is a wide variability of manifestations in chronic iron-deficiency. It appears that in chronic clinical anaemia extensive bone changes can be seen similar in extent to those of beta-thalassaemia minor or the marrow hyperplasia of sickle-cell disease, but not as extensive nor as severe as those of betal-thalassaemia major. Obviously this makes differential diagnosis of porotic hyperostosis difficult. Consideration needs to be given to the geographical areas of the abnormal haemoglobins, the age distribution of individuals affected, and the associated mortaliity in addition to comparison of the lesions themselves.

A diagnosis?

The Arabian Gulf traditionally lies on the crossroads between Africa and Asia, Asia and the Mediterranean. In addition, until recently, it was heavily malarial in watered areas (see Chapter 3). Therefore it should come as no surprise that sickle-cell anaemia and both thalassaemias are present amongst modern populations in the area.

In the Eastern Province of Saudi Arabia current estimated gene frequencies are:

1.	Hb-S	c0.175
2.	Alpha-thal	c0.43
3.	Beta-thal	c0.20 (El-Hazmi 1982, 1986).

These frequencies, however, are highly variable, according to tribal group and location (Al Awamy et al. 1984; Ganeshaguru et al. 1988).

In a survey of infants based on Damman, eastern Saudi Arabia, some 20% were Hb-AS, 1.6% had Hb-SS, and 0.25% had Hb-S/B-thal. Alpha-thalassaemia was very common with estimates of up to 50% of the population carrying at least one alpha-thal gene. So far, however, Hb-H disease is extremely rare (El Hazmi 1986). Similarly the prevalance of beta-thalassaemia major is at this stage only an estimate, although Gelpi (1983) suggests that in some oases the true frequency of the disease may be close to 3% of the population.

Despite these relatively high frequencies, the incidence of severe forms of sickle cell disease or thalassaemia is

extremely low or undescribed (in the case of the thalassaemias). The course of sickle cell disease in Eastern Saudi Arabia and the Gulf is benign. While pronounced anaemia occurs in homozygous infants of 9–12 months of age, vaso-occlusive crises rarely occur and if they do are generally only slight (Perrine et al. 1981). 'Hand and foot' syndrome and skeletal deformities associated with sickle cell disease are not commonly reported from the Eastern Province of Saudi Arabia (El Hazmi et al. 1990). Of 42 young children with sickle cell disease, only one child had osteomyelitis, though there did appear to evidence suggesting elevated rates of infection among the children (El Hazmi et al. 1990).

The disease is widely variable even in the Eastern Province. Bennett and Namyak (1990) describe 57 referrals with Hb-SS. 74% had osteonecrosis, 61% had osteomyelitis (most frequently in the younger age group) and coarse vertebral trabeculation and 'codfish' vertebrae (i.e. vertebrae with depressed endplates) were most common. Yet only one patient had thickening of the vault, and that without hair-on-end. There is no or only very low mortality associated currently with sickle cell disease in the eastern province of Saudi Arabia.

The benign course of the disease appears to be due to the possible interaction of three factors: alpha-thalassaemia which ameliorates Hb-S due to the decreased concentration of Hb-S in the red cell; hereditary persistence of high levels of foetal haemoglobin; and possibly iron-deficiency anaemia which prevents red cells sickling (El-Hazmi 1979; El-Hazmi et al. 1990). On Bahrain Island, however, sickle cell disease is clearly associated with higher than normal maternal mortality suggesting that the course of sickle-cell anaemia amongst Bahrainis is not as benign as amongst the Eastern Province Saudis (El Shafei et al. 1988).

The high frequencies of abnormal haemoglobins in the area suggest their presence over a long period of time (Gelpi 1983, Lehmann et al. 1963) although gene frequencies are susceptible to environmental changes and population movements. Therefore it cannot be assumed that the relatively benign course of congenital anaemias seen in the area today also occurred in the past. Nor can it be assumed that, given the presence of thalassaemia and sickle-cell anaemis in the area today, these were also prevalent 2000 years ago, nor that every case of porotic hyperostosis represents an abnormal haemoglobin. But the possible identification of sickled cells in a Hellenistic adult skeleton from Kuwait (Maat and Baig 1990) suggests that at least one of the congenital anaemias was present nearby at a contemporary period.

A natural history of anaemia

In the skeletal samples examined the frequencies of people affected, the early age of onset, plus the extensive nature of the lesions rule against hereditary spherocytosis, pyruvate kinase deficiency, and secondary polycythaemia as being the cause. It is possible that a single case of polycythemia vera may be included in the number of individuals affected but this would not account for the large percentage of people affected. Table 8.4 compares the diagnostic features of the

Table 8.4
Diagnostic features of the anaemias

	Thalassaemia major	Thalassaemia minor	Sickle-cell anaemia	Iron deficiency	Bahrain
Skull	Marrow hyperplasia	Less severe, c50% with mild-mod changes	5% of patieitns with hair-on-end	Similar but less severe	4% severe, thickenning incuding hair-on-end
Facial Bones	Overgrowth of marrow, no pneumatization of sinuses	Less severe	Rare	Frontal sinuses normal	Marrow hyperplasia in sinuses in 0-3 age group
Vertebral Column	General osteoporosis	Less severe	Compression or cupping of vertebrae	Minimal osteoporosis, rare cases of compression	c4.1% in 0-6 years with osteoporosis
Metacarpals/ Metatarsals	Early: marrow hypertrophy and coarsening of trabeculae	Less severe	Similar	Rare	Excessive thinning, lacy appearance, at least 5% of 0-6 years
Ribs	Cortical thinning & medullary expansion	Less severe	No data	No data	Lacy, no rib within rib appearance
Long Bones	Premature fusion of epiphyses, cortical thinning	Less severe	Thrombosis, thickenned cortex	Possibly cortical thinning	Lacy apppearnce
Pelvis	Marrow hyperplasia	Less severe	Marrow hyperplasia, aseptic necrosis of femur head	Mild osteoporosis	Osteoporosis, cortical thinning
Infection			Increased frequency		Low percentage
Occurence	At birth or shortly after	-	Delayed onset 6m+	Delayed onset after birth 8m+	Postcranial changes 0–6 years; majority 6m–3y.

Source: Aksoy et al. (1966); Baker (1964); Burko et al. (1961); Steinbock (1976)

remaining anaemias with the results from Bahrain.

Diagnostic signs of sickle-cell disease are absent. There is no evidence of vertebral end plate cupping amongst adults and adolescents. Similarly no cases of aseptic necrosis were observed in the population, and only one case of an adult with prurulent osteomyelitis was found. This skeleton however is fragmentary. In addition, in sickle-cell disease 'hair-on-end' appearance is only observed in 5% of homozygotes (Ortner and Putschar 1981). In the DS3 population 'hair-on-end' appearance is much more common. Amongst those who died aged 1–6 years 4% had radial trabeculation. If this was indicative of sickle-cell disease, based on the frequency of 'hair-on-end' amongst homozygotes, nearly 80% of subadults who died before 6 years of age had sickle cell disease. Given a subadult mortality rate till this age of around 40%, this would mean 32% of the entire population were sickle cell homozygotes at birth, or a gene frequency of (0.56), far exceeding even maximum gene frequencies seen in African groups (c0.4). In addition sickle cell anaemia is not generally associated with the extreme thinning of cortical bone observed in these skeletons.

Several of the same arguments apply to alpha-thalassaemia. In any population, owing to the complex inheritance of the alpha-thalassaemia genes, the percentage of individuals showing signs of Hb-H disease will be extremely small. This is apparent in Saudi Arabia where, despite the 50% prevelance rate of alpha-thalassaemia, Hb-H is a rarity (El Hazmi 1986). In addition, in terms of disease, Hb-H is generally associated with an intermediate state incompatible with the extensive and extreme bone changes found in 4% of those who died during childhood.

This leaves us with two alternative explanations: beta-thalassaemia or chronic iron deficiency. Given the lack of survey data, the true frequency of bone changes in iron deficiency is unknown and estimates range from low to common (Ortner and Putschar 1981; Stuart-Macadam 1989). Studies in areas historically devoid of the congenital anaemias, such as North America, demonstrate high percentages of porotic hyperostosis attributable to iron-deficiency. The percentage of cases observed on Bahrain, while high, are not inconsistent with these results. No studies, however, on either archaeological or modern populations suffering from iron deficiency show the extreme changes seen so clearly in Plate 6. There are a few reports of osteoporosis and cortical thinning occurring (Cockshutt-Smith 1986; Palkovich 1987). These suggest firstly that changes tend to be osteoporotic, rather than actual destruction of the cortical walls, and secondly that such changes are not frequent. The other criterion frequently used to suggest thalassaemia rather than iron deficiency are the changes to the maxillary and frontal sinuses (Baker 1964). This no longer holds—a high proportion of individuals with skull changes due to iron deficiency also show at least some overgrowth or failure to pneumatise the sinuses (Reimann and Kuran 1973). Again whether this incorporates the extreme changes seen in thalassaemia major is a matter for conjecture.

Finally the possibility of diagnosing every individual as either iron-deficient or thalassaemic is impossible in a skeletal group given the possible coexistence of these conditions together with sickle-cell anaemia in malarial environments. Indeed malaria itself is suspected as a direct cause of iron-deficiency anaemia in sufferers due to the increasing iron demands of the parasite upon the body (Manir and Khaleque 1969). Despite these reservations, individuals homozygous for thalassaemia may still be visible in the sample.

Interpretation

In the DS3 group, 4% of subadults who died before 6 years of age had extensive and severe cranial lesions, including 'hair-on-end' appearance. Accompanying these changes were alterations to the facial bones. These changes were not merely increased porosity: extensive swelling of the bones, thickening of the palate, and trabecular deposits in the maxillary sinuses were frequently observed. A small number of individuals (the fragmentary nature means that not all can be connected to a cranium) also demonstrated extreme marrow hyperplasia including porosity, 'lacy' appearance and cortical erosions. These signs are all visible in people suffering beta-thalassaemia major. In addition the early onset of porotic hyperostosis in infants prior to six months of age suggests a congenital anaemia as opposed to iron deficiency which may develop in a child by about 3 months but not appear radiologically until after a lag of several months (Palkovich 1987; Stuart-Macadam 1985).

One means of confirming this possible diagnosis of beta-thalassaemia major is through an examination of the iron levels in diseased and normal bones (Zaino 1974). In iron-deficiency anaemia, iron stores are depleted resulting in a lower than normal percentage of iron in the total body. In contrast, in the haemolytic anaemias, particularly when diet is adequate, the body's stores of iron (non-circulating) are increased (Pootrakul et al. 1988). In infants this increase will probably be marginal due to the lack of time for accumulation to begin, but iron overload in older children is a serious problem. While normal iron levels for children are between 2–3 gms for total body weight, in beta-thalassaemia major these levels may reach up to 15 gms—the same levels as seen in pathological iron overload in adults (Bothwell et al. 1979).

In order to test the possibility of distinguishing between iron deficiency and iron loading, ten samples were submitted for atomic absorption spectroscopy (Table 8.5). Wherever possible normal and diseased bones were matched from the same chamber to give a comparative picture. Iron tends to accumulate in bones through diagenesis (Lambert et al. 1983). This is particularly true in wet environments where the soil contains naturally high levels of iron (Zaino 1974). However examination of samples taken from the same

chamber should give a relative, though not absolute, indication of the iron levels. In addition a soil sample from a burial chamber at DS3 was tested.

Table 8.5
Iron levels from bone at DS3

Location	Sample	Fe (ppm)
DS3:		
73/18	Porous rib	500
73/18	Porous metatarsal	700
73/18	Normal cranium	300
73/18	Porous vertebra	700
73/18	Normal metatarsal	600
83/30	Normal metatarsal	200
73/18	Normal ribs	600
73/18	Normal vertebra	700
83/30	Porous metatarsal	6100
Saar:		
5/102	Porous cranium	200
DS3:	Soil	5600
	Soil	0
Saar:	Soil	3600

Ignoring the last sample from Mound 83, chamber 30, the average percentage of iron in the normal samples was 480 ppm. compared to 525 ppm in the diseased samples. While this suggests that porotic hyperostosis was not associated with iron deficiency in this population, care needs to be taken with the figures. Values are highly variable and the difference is not statistically significant. The level that is highly significantly different is that of the six year old child from DS3, Mound 83, Chamber30, No. 9. The estimated percentage of iron for total body weight for this child is 12 grams—clearly a case of iron overload. There is, however, a strong possibility of sample contamination in this individual, even though bones from within the same chamber give low to normal iron levels. The soil samples taken demonstrate the extreme variability in iron levels and the problems of contamination.

On the other hand, x-rays of this four to six year old child from Mound 83, Chamber 30, do indicate the severity of the condition and support the possible diagnosis of beta-thalassaemia. As seen in Plate 5, the cranium has clear thickening in the frontal, parietal and occipital regions. In the parietal region the outer table is partially destroyed and radial spiculation indicative of 'hair-on-end' is present. At the same time the palatal plate appears thickenned. The cranium was covered with fine porosity, in particular the sphenoid and maxilla were very porous, and the maxillary sinuses were filled with trabecular bone. Trabecular cribra orbitalia was visible within both orbits with marked swelling of the normal bone contours. The skeleton was also affected postcranially with marked cortical thinning of the vertebral arches, ribs, pelvic bones, long and short tubular bones. The overall picture is of extreme marrow hyperplasia and cortical thinning affecting the entire skeleton. It is entirely consistent with descriptions of beta-thalassaemia major and certainly more severe and extensive than those described for either sickle cell or iron deficiency anaemia.

Frequency of thalassaemia

The possible diagnosis of beta-thalassaemia major in one skeleton at DS3 does not mean that all individuals with porotic hyperostosis and cribra orbitalia were thalassaemics. Following the method used by Angel (1971, 1978), gene frequencies can be estimated. If we assume that moderate-severe porotic hyperostosis indicates individuals with beta-thalassaemia major, 25.6% of children who died before 6 years at DS3, and 17.7% at Saar were homozygous for thalassaemia (or 10.2% of the whole population at DS3 and 7.1% at Saar). This would indicate a gene frequencies of 0.505 at DS3 (0.42 at Saar) at birth and that 42% of the population at DS3 (and nearly the same at Saar) was heterozygous. In the light of current gene frequencies of thalassaemia this is exceptionally high. If we assume that only those children with severe changes to the skull, especially hair-on-end, represent thalassaemics, this means 8.1% of dead children from DS3 and 5.9% from Saar. This lower percentage tallies with the 9% of individuals from single chambers at DS3 with postcranial changes. The resultant estimated gene frequency at birth is 0.284 with an estimated 40% of the population heterozygous. This of course is an absolute maximum since it is based on the frequency of homozygotes amongst those who died before six years. Yet homozygotes have no chance of survival beyond age 6 compared to the whole population with a 55% chance of survival at this age.

This factor can be added into the calculation. At DS3 between the ages of 0 and 6 years, 8% of dead children were homozygous, the remainder were either normal and heterozygous. Yet this is equivalent to only 40% of the total population between ages 0–6 years. Therefore, of the entire living population between 0–6 years, 3.2% were homozygous and the frequency of the thalassaemia gene (square root of the homozygous percentage) is 0.179. Therefore, at birth, 29.4% were heterozygotes, 3.2% were homozygotes and 67.4% were normal.

Similar figures for Saar cannot be calculated since the youngest age group is unknown. In total the percentage of affected individuals at Saar is less than at DS3, suggesting lower frequencies of abnormal genes.

Given that an estimated 29.4% of the population were heterozygotes and that approximately half of these could be expected to develop bone changes, the expected frequency of thalassaemia minor visible in the living population would be 14.7% during childhood. Due to the loss of homozygotes from the population and possibly a selective advantage for heterozygotes, this figure may have increased slightly by adulthood. Allowing solely for removal of homozygotes the expected frequency of heterozygotes with bone changes is 16% amongst adults. A comparison of the expected with the

observed frequencies among the dead immediately displays disparities.

1. 0–6 year:
   47.6% with porotic hyperostosis,
   56.4% with cribra
   compared to 22% (estd thalassaemics with bone changes)
2. Adults:
   11% with porotic hyperostosis,
   35.4% with cribra orbitalia
   compared to 20% (estd thalassaemics with bone changes)

This means that at DS3 about 25% of cases of porotic hyperostosis amongst children and 34% of cribra orbitalia are excess to thalassaemia and about 20% of cases of cribra orbitalia amongst adults are excess, so due to other causes. One explanation could be the coexistence of another defective gene such as sickle cell causing some cases of porotic hyperostosis. Given the discovery of sickle-cell anaemia on Failaka in the same period this is a possibility (Maat and Baig 1990). However, the individual on Failaka had no severe bone changes despite the presence of sickled cells indicating either that he was a heterozygote or that even 2000 years ago the course of sickle cell in the area was benign. (In this latter case a very small percentage of individuals would show bone changes, including osteomyelitis and aseptic necrosis. Yet amongst the skeletal populations from Bahrain not only are these conditions absent but there is high associated mortality with porotic hyperostosis, unexpected in a benign form.) Again, while sickle-cell anaemia does not appear to have had a major role, this does not say that the condition did not exist on the Island. It may well account for a small percentage of cases but, as today in this area, it was not a serious condition.

The most likely conclusion is that, concomittant with beta-thalassaemia and less severe congenital anaemias, there was iron-deficiency anaemia. As El Hazmi (1979) describes, this same interaction of factors is observed today in Saudi Arabia.

Anaemia and the Bahrain population

As discussed earlier, iron-deficiency anaemia may be caused by a variety of factors both nutritional and infectious. These are associated with characteristic patterns of mortality and morbidity (Mensforth et al. 1978; Puffer and Serrano 1973; Palkovich 1987). Generally in the first six months of life infants are protected by prenatal stores of iron. Only in premature infants, twins, low birthweight infants or infants with severely deficient mothers is anaemia visible within the first six months (Smith 1954). In these instances one would expect to see early porotic hyperostosis from about 2 months of age (due to the lag between onset and development), the development of which may be due to infectious episodes (Palkovich 1987). Accompanying this would be increased mortality in the second six months, probably due to the development of nutritional deficiency after repeated bouts of diarrhoea (Puffer and Serrano 1973).

The typical pattern of diarrhoeal disease and mortality is high death rates in the first three months of life followed by sharply decreased rates between 6–11 months due to the development of immunity (Puffer and Serrano 1973). This corresponds to the pattern of disease observed by Mensforth and colleagues (1978). Prenatal stores of iron are generally sufficient to maintain normal growth up to six months, but prolonged breast-feeding or inadequate weanling diets contribute to iron-deficiency after this age. Thus children become susceptible to infections. This results in poor absorption of iron, the body's sequestration of iron as a defense against pathogens, and heightened iron demands to maintain growth. The combination of factors results in the development of porotic hyperostosis among young children from six months to three years of age and for a second peak of mortality around 2–3 years.

Neither of these patterns of mortality and morbidity corresponds exactly to that observed in Bahrain. In this group mortality is high in the first month of life, peaks again in the second six months, and remains high, though declining, until some 3 years of age. Cribra orbitalia and porotic hyperostosis develop around six months of age, becoming increasingly prevalent until 3 years when healed cases begin to appear. Active anaemia continues until six years after which healed cases predominate. Significant points of contrast with mortality due to nutritional deficiency are the absence of early cases of porotic hyperostosis on Bahrain and high mortality in the first month of life with only moderate levels of low-birthweights. In contrast to the pattern for diarrhoeal disease, mortality rates are not decreased during the second six months of life and mortality, while high, does not peak around 2–3 years.

An alternative explanation does exist. The pattern of malaria morbidity and mortality follows a slightly different pattern to that of diarrhoeal disease. During the first six months of life the infant tends to be protected from infection by passive immunity acquired from the mother. Associated with this is low mortality due to malaria. From about 6 months until one year immunity to malarial attack is low, resulting in peak parasite rates during this age group, with associated mortality and morbidity. Between one and four years there is rising immunity from frequent and intense malarial attacks, but with great morbidity and mortality. After 5 years acquired immunity has developed and malarial attacks are less frequent (McGregor et al. 1961).

In a population containing homozygotes for the congenital anaemias, heterozygotes and normal individuals it is hypothesised that the following pattern would occur:

1. Neonates (Passive immunity):
   homozygotes—protected
   heterozygotes—protected
   normal—protected
2. 6m–1 year (low immunity):
   homozygotes—protected by homozygosity,
   development of symptoms (severe porotic hyperostosis, beginning of deaths)

heterozygotes—protected by heterozygosity (low levels of porotic hyperostosis)
normal—frequent and intense malarial attacks (concomitant porotic hyperostosis and mortality)

3. 1–4 years (rising immunity):
homozygotes—extreme morbidity and mortality due to thalassaemia (peak mortality, small percentage with severe porotic hyperostosis)
heterozygotes—protection by heterozygosity, malaria but attacks less virulent (level of porotic hyperostosis, but no mortality)
normal—malarial attacks declining over time (decreased mortality and increasing healed porotic hyperostosis)
4. 5 years + (acquired immunity):
homozygotes—death amongst thalassaemia
homozygotes, sickle cell disease, if benign, no mortality
heterozygotes—less frequent and less severe malaria (marginal porotic hyperostosis)
normal—less frequent and less severe malaria (healed porotic hyperostosis).

(based on Molineaux et al. 1979).

Malaria by itself is not necessarily the cause of significant mortality but it can be a significant contributing factor to mortality from other infections.

This pattern explains why mortality peaked on Bahrain during the second six months of life and why porotic hyperostosis became increasingly prevalent from this age unto 3–4 years, irrespective of any weaning peak. Thalassaemia homozygotes in the population would also die within this time period with severe porotic hyperostosis.

The model does not, however, explain the peak of mortality within the first month of life. Obviously a complex of factors is involved which will be explored more in Chapter 9. At this stage it appears that a combination of iron-deficiency anaemia and thalassaemia plus the possible presence of other congenital anaemias created significant health problems for the Bahrain populations, possibly more in DS3 village than in Saar. The pattern is probably not so very different from more recent historical groups in the area.

Infection

Only a small percentage of infections leave signs upon the bone. In addition, owing to the variability of physical response to disease, typical symptoms of the conditions do not always appear, while many changes may simply be non-specific in nature. Potential identification of the actual disease is reliant upon the most accurate possible recording of pathological lesions.

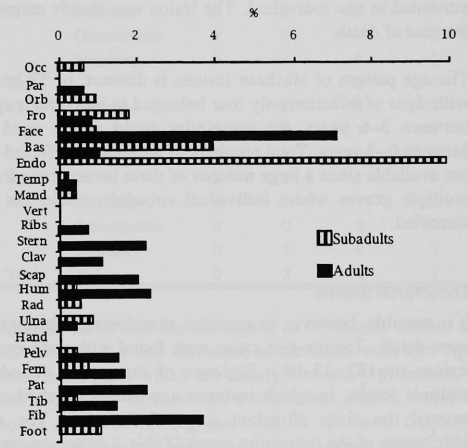
Proliferative/infectious lesions were categorised following a modification of the scheme proposed by Stothers and Metress (1976). These classes were:

1. porosity;
2. striations;
[1 and 2 comprise periosteal lesions]
3. primary osteomyelitis (including sclerotic lesions) with infection of the cortex and endosteum;
4. suppurative osteomyelitis (presence of cloacae and drainage areas);
5. secondary osteomyelitis (obvious traumatic origin).

Results

A small percentage of skeletons within the samples showed signs of infection. The distribution of these lesions at DS3 can be seen in Figure 8.5. Obvious differences occur between subadults and adults in the bones most frequently affected.

Figure 8.5
Distribution of infectious lesions



Subadult infections

Subadult infections were primarily seen in the form of periosteal lesions, with superficial pitting or roughening of the external bone surface. In some instances the apposition of new bone on the periosteal surface is apparent. These lesions were found throughout the skeletons with the exception of the thoracic area (Figure 8.5). Most common were periosteal lesions of the orbits, frontal and endocranium. These could be distinguished from porotic hyperostosis by the nature of the lesions. During porotic hyperostosis the outer bone table is destroyed by progressive expansion of the bone from within, in periostitis new poorly organized bone is laid down upon the external bone surface generally without its destruction.

Six cases of orbital periostitis were found. While in some cases the same area of bone was affected as in cribra orbitalia, in others change was restricted to the outer margin of the orbit. Two cases were also found where the supraorbital area was affected.

Endocranial lesions were the most common of all (Plate 7). The inner calvaria in these cases was covered either with fine porosity with a thickened, slightly roughened bone surface, or else there was clear apposition of fine striated bone though the original bone surface was always present. In a few cases the surface of the new bone showed cicatricial markings presumably following vascularity.

In the long bones, the affected surfaces were always the shafts rather than metaphyses. Only one instance of infection involving more than the periosteal surface was found. The affected bones were found in a multiple grave which means that the complete skeleton could not be identified, but one radius, ulna and femur were found with signs of marked bone destruction. In each case the original bone shaft was partly surrounded by an involucrum of new bone through which drainage holes were visible (Plate 7). These bones were the only bones found with such a lesion and are therefore attributed to one individual. The lesion was clearly active at the time of death.

The age pattern of all these lesions is distinct: of 57 bones with signs of infection only four belonged to individuals aged between 3–6 years, the remainder could all be aged to between 0–3 years. Total numbers of individuals affected are not available since a large number of these bones come from multiple graves where individual subadults could not be identified.

Endocranial lesions

It is possible, however, to examine at endocranial lesions in more detail. Twenty-five cases were found with endocranial lesions (n=187, 13.4%). Eighteen of these were found in multiple tombs, in which instance age cannot be attributed beyond the class of infant (or c 0–2 years). The age distribution of the remaining seven (Table 8.6) demonstrates that the lesions occurred primarily within the first year of life, at a slightly lower frequency in the second, and not at all by five years of age. There were no cases of healed lesions.

Table 8.6
Age distribution of endocranial lesions, DS3

Age (years)	N
0.0 – 0.5	1
0.5 – 0.75	2
0.75– 1.0	1
1.0 – 1.5	1
1.5 – 2.0	1
2.0 – 3.0	0
3.0 – 4.0	1
Total	7

The pattern of occurrence is distinctly different to porotic hyperostosis where the age range 1–6 y is most affected. The endocranial lesions appear to be an earlier phenomenon occuring even in the 0–6 month age group. Yet in all instances of endocranial lesions some form of porotic hyperostosis, either cranial or orbital, was found. In four cases (those marked with an asterisk) the porotic hyperostosic lesions were only just beginning to show at the time of death, while the endocranial lesions covered 25–50% of the internal surface of the bone. This suggests that periosteal lesions of the endocranium, while related to porotic hyperostosis, may possibly reflect a condition contributing to its development.

Amongst the Saar skeletons only one case of subadult infection was found. This was an infant aged 6–9 m with porosity covering a large area of the endocranium. Externally there were slight signs of active but mild cribra orbitalia without more extensive cranial lesions.

Adult infection

The percentage of adult bones in the sample affected by infection is similarly low (less than 7.5%) although, based on individual skeletons, the percentage affected is comparable to agricultural populations (Table 8.7). The distribution of these lesions both in DS3 and Saar can be seen in Figure 8.6. The pattern of skeletal involvement is similar in both cases: the bones of the lower leg, along with the facial area, were most frequently affected. The rest of the skeleton was less involved (between 0–2.5% of bones).

Table 8.7
Comparative frequencies of infection

Site	%	N	Source
Iran/Iraq:			
Paleolithic	17	6	Rathbun 1984
Pre-Neolithic	0	-	
Neolithic	30	56	
Chalcolithic	26	30	
Bronze& Iron Age	3	540	
U.S.A.:			
Georgia*			
Preagricultural	4.5	156	Larsen 1984
Agricultural	15	374	
Ohio River Valley:			
Late Archaic	10.8	111	Perzigian et al. 1984
Middle Woodland	28.6	49	
Fort Ancient	13	792	
Bahrain:**			
DS3	24.3	152	
Saar	14.8	54	

*Tibiae only; **Individual adults only.

Periosteal lesions (i.e. superficial porosity and striated bone) predominated, with a smaller number of bones showing evidence of suppurative lesions. Ten cases of infection wererelated to trauma (both fracture and dislocation). In these cases the extent of the lesion is clearly localized to the damaged area. In only one case was the lesion active at death. This reflects the low prevalence and lack of severity of

trauma, as indicated on the bone, within these two populations.

Of the remaining 31 cases of infection found at DS3, six involved extensive suppurative or sclerosing lesions, the remaining 25 are superficial lesions of the bone probably due to inflammation of the periosteum. Table 8.8 lists the involvement of bones in each case. It is obvious that, where evidence is available, a high proportion are symmetrical and involve more than one area of the skeleton. Specific locations on the affected bones appear to be particularly susceptible: the majority of tibial and fibular lesions involve the shaft; facial lesions are concentrated on the palate and around the nasal area; pelvic lesions most commonly occurred around the acetabulum.

Figure 8.6
Differential involvement of the skeleton, Saar and DS3

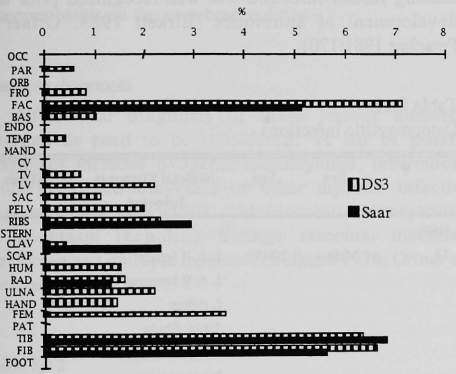


Table 8.8
Infectious lesions at DS3

	N	Single	Multiple	Symmetr	Unilat.
Crania	5	2	1		
Face	8	4	2(1*)		
Clavicles	3	3(3*)	-		3
Ribs	4	-	4(4*)		
Vertebrae	2	1	1(1*)		
Innominate	8	2	2	1	3
Humeri	4	2	2	0	2
Radii	3	1	-	0	2
Ulnae	4	1**	3	0	1
Femora	5	1	4(1*)	3	1
Tibiae	14	3	9	3	7
Fibulae	12	2	9	5	2

Columns do not tally owing to missing data
*Osteomyelitic lesions; ** Secondary lesions.

The symmetrical and extensive nature of the lesions suggests that it may be possible to isolate the specific pathoses causing the disease.

Owing to the fragmentary nature of the majority of remains it is not possible to calculate the percent of affected individuals in the entire population. It is possible, however, by looking superficially at the individual cases to determine age and sex trends (Table 8.9).

Table 8.9
Age and infection, DS3

		Female		Male	
		Active	Healed	Active	Healed
15–20	Periosteal	1	0	1	0
Total		1	0	1	0
20–30	Periosteal	3	2	–	–
	Osteomyelitic	0	1	2	–
	Secondary	0	0	0	2
Total		3	3	2	2
30–40	Periosteal	3	3	2	2
	Osteomyelitic	0	0	0	1
	Secondary	0	1	0	1
Total		3	4	2	4
40–50	Periosteal	1	2	0	2
	Osteomyelitic	0	0	1	0
	Secondary	0	3	0	0
Total		1	5	1	2
50+	Periosteal	0	1	1	0
	Osteomyelitic	0	0	0	0
	Secondary	0	1	0	2
Total		0	2	1	2

At DS3, infectious lesions appear to be clearly neither age nor sex specific, and incidence accumulates with age. Apart from those cases which are clearly secondary to trauma, the rest apparently demonstrate a consistent pattern where the bones of the lower leg and maxilla are most frequently affected. Often the lesions tend to be symmetrical. These cases are more extensive and involve several areas of the skeleton, with more severe lesions including distinct areas of suppuration.

Distinguishing the pathogens involved, however, is much less simple since there are a wide variety of infectious organisms and their effects tend to be non-specific. It is, however, important at least to isolate the possible organisms since they are an important reflection of living conditions within a community (Powell 1991).

The number of cases of infection is much smaller at Saar, making it difficult to determine any clear patterning. The pattern of bones affected is similar to DS3 (Figure 8.6), especially when secondary infections due to trauma are excluded (i.e. clavicle, radius and rib). This leaves three cases of infection–two involving the tibia and fibula, one the maxilla. As at DS3, equal numbers of males and females were involved with two between 40 and 50 years and one unaged. The infections causing skeletal lesions did not have a great impact on mortality; the majority (2/3) were healed at

the time of death. On the basis of the evidence here there is nothing to suggest different infectious organisms affecting DS3 and Saar.

Differential diagnosis of infection

Differential diagnosis is difficult given the number of infectious organisms which cause changes to bone, and the often non-specific nature of these lesions. As emphasised earlier, however, the task is necessary if the social implications of disease in ancient populations is going to be satisfactorily addressed.

The material from Bahrain, as described earlier, can be divided into three categories based on the nature of the skeletal lesions: secondary infection following trauma; destructive lesions of the entire bone (osteomyelitic infections); and periosteal lesions. Diagnosis, however, can be undertaken with a degree of certainty only if the majority of the skeleton is present, thus isolated bones will not be included in the following discussion unless they are particularly pathognomic.

Secondary infection

Ten cases of secondary infection were found in the population. These are summarised in Table 8.10.

Table 8.10
Secondary infection

	Female	Age	Skeletal Elements Invovled	Status
DS3:				
1.	Female	40–50	Innominate	Healed
2.	Male	45+	R ulna	Healed
3.	Female	40–50	R clavicle	Healed
4.	Male	30–40	L radius	Healed
5.	Female	40–50	R radius	Healed
6.	Male	45+	Innominate	Healed
7.	Male	25–35	L ulna	Healed
Saar:				
8.	Male	20–30	L clavicle	Healed
9.	Female	50+	R radius	Healed
10.	Female	35–45	Rib	Healed

These cases were all either healed or healing by the time of death. In the majority of instances the infection was probably caused by the entry of microorganisms into the area at the time of trauma. The successful healing is indicative of both the relatively minor nature of the trauma and the level of health amongst these adults whose immune systems were sufficiently robust to fight the infection successfully.

Osteomyelitic infections

Severe infections involving the entire bone, not just the outer layer adjacent to the periosteum, occurred in several individuals (Table 8.11).

The primary feature of many of these instances is extensive destruction of bone without concomitant proliferation of periosteal bone. The obvious destruction of the mandible (No. 13) appears to be related to the direct spread of infectious bacteria from the mouth, probably through dental decay or abscessing. The resultant infection has caused violent destruction of the bone, forming a drainage abscess and superficial reaction of the surrounding bone. The porosity around the palate and nasal area is probably also due to the localized effects of infection. The severity of the lesion and the fact that it was active at the time of death suggest that this infection may have contributed to death. The danger of dental disease, particularly abscesses, and their role in causing further infection was well-recognized prior to the development of antibiotics (Birkett 1983; Ortner and Putschar 1981:120).

Table 8.11
Osteomyelitic infections

	Sex	Age	Skeletal Elements Invovled	Status
DS3:				
11.	Male	20–30	L & R femur	
			L & R humerus	
			L radius	
			Tibia, fibula	
			Ribs, vertebrae	
			Basicranium	Active
12.	Male	40–50	Ribs, sternum	
			Vertebrae	
			Tibia, fibula	Active
13.	Male	20–30	Mandible, maxilla	Active
14.	Male	30–45	R humerus, l ulna	
			L clavicle	
			Ribs, sternum	Healed
15.	Male	15–20	Ribs, vertebrae	
			L & R femur	Active
16.	Female	20–30	Femur	Healed?
Isolated bones:				
17.	–	0–3	Temporal	Active
18.	–	0–3	L & R femur	Active
19.	–	0–3	Parietal	Active
20.	?	Adult	Saccrum	
			2 thoracic vertebrae	Active

The swollen femur (No.16) without external signs of infection other than swelling of the contours may be an instance of sclerosing osteomyelitis (Ortner and Putschar 1981). Given the incompleteness of the material, however, diagnosis is extremely difficult.

Common features

Of the remaining five cases several points of similarity occur, suggesting a common disease process. Of the four that can be sexed all are male, one age 15–20, one 20–30, one 30–40 and one 40–50 years. The lesions are highly destructive involving lytic cavities with little proliferative reaction. The affected bone is thickened and has coarse trabeculation. Lesion location is, with the possible exception of no. 11, concentrated on the ribs (though from current data not all ribs), lower thoracic and lumbar vertebrae, as well as the sacrum. Number 11 is a departure from this in that the proximal femur is severely affected, possibly on both sides of the body, and that there appears to be destruction in the basicranial area. In this individual there is scattered periosteal reaction on the other long bones. In two other individuals there is also slight periosteal reaction on the tibiae, although the relationship of these lesions to the major focus of disease in unknown since the reaction in the lower limb tends to be scattered and obviously superficial compared to the overt osteoperiostitis on the vertebrae and ribs.

Possible diagnosis

In differential diagnosis of these lesions numerous alternatives need to be considered. A list of possible diagnoses includes pyogenic osteomyelitis, tuberculosis, brucellosis, actinomycosis or other mycotic infection, treponematosis, sarcoidosis, echinococcosis, histocytosis-X, or neoplasm including Ewings sarcoma, metastatic carcinoma and multiple myeloma (Buikstra 1976; Ortner and Putschar 1981).

Several of these possibilities can be disregarded. Multiple myeloma and metastatic carcinoma most commonly affect individuals over 50 years of age (Ortner and Putschar 1981). Since all possible cases in the current sample are concentrated in younger age groups this does not seem to be a likely diagnosis.

Ewing's sarcoma similarly may be discounted. While adolescents and young adults are affected by this disease, the diaphysis of bones (especially in young adults) is affected with either the development of parallel layers of bone under the periosteum (onion peel appearance) or 'sunburst' type of growth (Ortner and Putschar 1981), contrary to the lack of exuberant periosteal reaction seen in the above examples.

Sarcoidosis is similarly unlikely since this disease predominantly affects the fingers and toes, none of which were found to be affected in the Bahrain samples. In addition, vertebral lesions usually involve multiple and sometimes widely separated vertebrae (Ortner and Putschar 1981), contrary to the localized nature of the lesions seen in the Bahrain cases.

Solitary and multiple destructive lesions may be seen in the collection of diseases called histocytosis-X. Affected age groups are children, adolescents, and young adults. The skull is the most common location for lytic lesions and the disease is marked by a definite lack of reactive bone formation (Lichtenstein 1953). Neither of these characterise the disease observed in the Bahrain material.

Table 8.12
Diagnostic features of osteomyelitic infections compared to Bahrain

	Bahrain	Osteomyelitis	Tuberculosis	Brucellosis	Fungal Infection	Actinomycosis
Age	Y Adult	Child/Adult	Adolescent/ Young Adult	Children and adults	Population subgroups	-
Sex	M>F	No difference	M>F?	M>F	M>F?	M>F
Nature of Lesion	Minimal proliferation	Involcrum, not bilateral	Resorptive, lytic lesions	Resorptive	Resorptive, possibly periostitis	Skeletal rare; periosteal, "wormeaten"
Vertebrae	Anterior only	Rare, only 1–2 disc, spinous process	Lower thoracic/ lumbar, limited no.	Collapse uncommon, osteophytosis when healed	All parts	Localized, often transverse process
Ribs	Lytic, slight periostitis	–	Variety of lesions, periostitis with lytic	Occasional abscess	–	–
Femur	Trochanter destroyed	Primarily suppurative	Destruction of joint with trochanter, no metaphyseal involvement	Long bones rare but knee common at endplate, no collapse	–	Reactive, periosteal
Pelvis + Saccrum	Destruction of body	Supurative	Destruction due to abscessing	Hip joint affected but no collapse	–	–
Cranial	One destructive lesion	Involved	Lytic, sharp borders	Not significant	Outer table only	Cervical and facial
Sequestra	None	Sinus, present but majority only one	–	–	Fistula common, small bone involvement	Rare

Source: Buikstra & Williams (1991); Daniel (1981); Kelley & Micozzi (1984); Ortner & Putschar (1981)

Echinococcosis infection which occurs through tapeworn infestation needs to be considered since the disease is common in Iran (Ortner and Putschar 1981). The bone marrow may become permeated by cysts causing localized necrosis. In the vertebrae, which form almost 50% of cases, the transverse processes and posterior elements of the spine can be involved (Ortner and Putschar 1981) which is contrary to the pattern seen in the Bahrain material.

The elimination of these diseases on the basis of epidemiology and lesion nature leaves four conditions which need to be more thoroughly considered in a diagnosis for the Bahrain material: tuberculosis, pyogenic osteomyelitis, brucellosis, and actinomycosis, or some other fungal infection. Treponematosis will not be considered here since the most common response in this disease is periosteal, particularly involving the face, tibiae and fibulae. Obviously while the disease is relevant to a consideration of the periosteal lesions seen in the sample, it is less relevant in a discussion of a resorptive pathology.

Table 8.12 outlines the features of the Bahrain material and compares these to the five possible diagnoses.

Each of these diseases bears some similarity to the cases described. Actinomycosis seems unlikely, however, given the lack of transverse spinous process involvement in the Bahrain cases as well as the location of most actinomycosis lesions in the cervico-facial region, and the frequency of periosteal reaction in this condition.

Brucellosis appears incompatible with the degree of bone destruction, especially in case no. 11. In addition vertebral collapse is uncommon in Brucellosis while it appears to be imminent in two cases from Bahrain.

Pyogenic osteomyelitis also seems inapplicable to these cases. While the age and sex distribution accords well with the Bahrain material, the skeletal distribution of lesions does not match. In only 2% of osteomyelitis cases are the vertebrae affected and when they are the spinous processes are also affected. Also contrary to a diagnosis of osteomyelitis is the lack of periosteal reaction, complete absence of sequestra and lack of involucrum formation.

Fungal diseases, while similar in effect to the lesions seen on the Bahrain material, appear to be ruled out by the lack of involvement of the small bones, and limitation of cranial lesions to the outer table. They do however remain a possible diagnosis.

Given these points tuberculosis appears to be the most likely condition responsible for these skeletal lesions. Firstly the age distribution agrees with the known historical pattern of the disease (Daniel 1981) especially when three isolated lesions in young child are also incorporated. In one multiple tomb subadult ribs with slight periostitis were found, possibly indicative of pulmonary tuberculosis. Although there have been reservations expressed regarding the equation of rib lesions with pulmonary tuberculosis (Kelley and Micozzi 1984 cp Buikstra and Williams 1991) more recent work suggests that people with tuberculosis are more likely to have these lesions than other people (Roberts et al. 1994).

In another tomb an isolated sphenoid bone was found with lytic circular lesions on the outer surface (Plate 8.). No other destructive lesions were found on facial bones from this grave which suggests that the lesion was not malignant carcinoma which presumably would have spread further (Ortner 1980). At the same time the smooth rounded nature of the lesions suggests a slower process than carcinoma, which accords well with a case of possible tuberculosis described by Ortner from Bronze Age Jordan (Ortner 1980).

The third possible subadult case is an infected temporal bone of a child (Case no. 17). This lesion is clearly lytic and perforates the outer table of the skull directly above the mastoid process. Middle ear infection due to tuberculosis is common in the first year of life (representing about 50% of all middle ear infections in one sample cited in Ortner and Putschar 1981). This may result in destruction of the outer bone and the mastoid process.

Including these three cases as possible tuberculosis, this would result in an age distribution of the disease amongst young children and younger adults. One contradictory point to the diagnosis of tuberculosis, however, is the male predominance of lesions (amongst adults: four males and one unknown). While two samples studied report a predominance of males over females in the development of this disease (Daniel 1981; Ortner and Putschar 1981), other studies are less definite (Buikstra 1977, Kelley and Micozzi 1984). The significance therefore of only males affected amongst the sexed skeletons is difficult to determine. Statistically the sample is too small to be significant but it must remain as a question-mark over this possible diagnosis.

On the other hand there are several points strongly in favour of tuberculosis as a possible diagnosis of these cases. The small numbers of vertebrae affected in each individual plus their location in the lower thoracic/upper lumbar area accords well with tuberculosis, as does the involvement of only the anterior portions of the vertebral bodies. The same point applies to the ribs: the selective involvement of only 2–3 ribs matches with osseous tuberculosis. Most importantly, however, the destruction of the trochanter, while uncommon in tuberculosis, is "the most identifiable tuberculous bone lesion of the adult with the exception of tuberculous spondylitis" (Ortner and Putschar 1981:154). Buikstra and Cook describe a similar case of trochanter destruction due to the presence of an abscess (Buikstra and Cook 1981: Figure 2).

Given the pathognomic nature of this sign, the possible diagnosis of tuberculosis in the Bahrain population appears more secure. The reservations have been noted above: the male predominance of lesions and the involvement of the

basicranium which is uncommon (Ortner and Putschar 1981). By a process of elimination the only possible alternative would be a fungal disease which fails to accord with either the observed age distribution (expected to be more age accumulative in prehistoric groups) or the skeletal distribution of lesions. The assumption is made that, until further evidence is accumulated in favour of an alternative diagnosis, tuberculosis was probably present amongst the populations from both Saar and DS3.

Tuberculosis in the Middle East

The diagnosis is unsurprising given the long history of tuberculosis in the Middle East. Elliot-Smith and Ruffer (1910, cited in Ortner and Putschar 1981) described a case of tuberculosis from Egypt while Ortner identified two individuals (one child and one adult) with tuberculous lesions from Bab edh-Dhra in Jordan (Ortner 1980). Even today tuberculosis is a major health problem in the Arabian Gulf (Hansen 1968; Musaiger 1987).

The disease is spread primarily by person to person contact, either through the respiratory tract, infection of the oropharynx lymphoid tissue, through the gut, or, least frequently, via skin wounds. Alternatively it may be contracted by drinking infected milk from domestic animals, particularly cattle. The disease is density dependent but the persistence of infection amongst small Amazonian tribes demonstrates that in conditions where the primary tuberculosis infection, occurring in childhood, reactivates during adulthood, survival of the disease is assured (Black 1975).

Historically initial infection tended to occur during childhood. In one study 87% of cases in a population were in children less than 14 years of age (Kaplan 1952). Peak mortality is also around childhood: c5 years of age. Generally the primary infection is pulmonary, but during the course of this initial infection the tubercule bacilli disseminate via the blood system, or possibly directly as in the case of the ribs, (Kelley and Micozzi 1984) to other organs including the bone marrow. These dispersed foci of infection may not be active during the primary disease but, especially in conditions of stress, may reactivate. The majority of osseous changes occur with this secondary bout of infection. Amongst infected individuals, only 5–7% will develop bony lesions. In addition, only 50% of these lesions may be typical further reducing the expected frequency of recognisable pathological skeletons in a sample (Buikstra 1976).

Given the mixed nature of the Bahrain material (multiple and individual skeletal groups) it is difficult to estimate the true frequency of skeletal cases. Based, however, on the number of infected vertebrae (since all of our adult cases involve vertebrae), the percentage of tuberculous skeletal lesions amongst adults is 1–2%. Allowing for the number of atypical cases, this would be c2% of the living population. Ultimately this would mean that c40% of the population had contracted tuberculosis. This is an overestimate since it is based on the assumption of equal survival probabilities yet tuberculosis sufferers are more likely to form part of the skeletal sample. In small populations from Brazil the frequency of tuberculosis varies from between 3–86% (Daniels 1981).

Table 8.13
Periosteal lesions

	Sex	Age	Skeletal Elements Involved	Status
DS3:				
21.	Female	15–20	L temporal	Active
22.	Female	20–30	L humerus & ulna	
			L & R fibula	Active
23.	Female	40–50	Palate	Active
			Innominate	Healed
24.	Female	30–40	L tibia & fibula	Active
25.	Female	40–50	L ulna	Healed
26.	Male	40–50	Fibula	Healed
27.	Male	30–40	L Ulna, ribs	
			Maxilla	Healed
28.	Female	30–40	Maxilla	Healed
29.	Male	30–45	Maxilla	Active
30.	Male	30–40	L tibia & fibula	Healed
31.	Infant	0–0.5	L tibia	Active
32.	Male*	40–50	Frontal	Healed
33.	Female	30–40	Parietals	Healed
34.	Female	25–40	L tibia & fibula	Active
35.	Female	20–30	L & R fibula	
			L femur, tibia	
			Innominate	Active
36.	Female	45+	Frontal	
			R femur, l humerus	Healed
37.	Female	20–30	L tibia	Healed
38.	–	Adult	L tibia & fibula	Healed
39.	Female	30–40	Frontal	Healed
40.	Female	20–30	L tibia, sphenoid	
			Maxilla	Healed
41.	Female	30–40	L & R fibula	Active
42.		2–3	R femur	Active
43.		c3	L frontal	Active
44.	Male	30–40	Innominate	Active
45.	Male	50+	l & R tibia	
			L fibula	Active
46.	Female	20–30	L tibia	Active
47.	Female	40–50	L femur	Healed
48.		Adult	R humerus	Active
Isolated bones				
49.	–	0–3	Frontal	Active
50.	–	0–3	Frontal	Active
51.	–	0–3	Frontal	Active
Saar:				
52.	Female	45+	L & R tibia	
			Fibula	Active
53.	Male	40–50	Maxilla	Healed
54.	–	Adult	R tibia & fibula	Healed

*Iron Age

Associated mortality in the past was high. In historical urban centres up to one-fifth of all deaths could be attributed to tuberculosis (Ortner and Putschar 1981). The sectors of the population more affected were children and young adults. While the childhood cases (least likely in any case to develop skeletal lesions) are lost in the mass of childhood mortality, the condition would have been a significant contributor to young adult mortality. One question, however, is whether females were equally affected as males; skeletal studies suggest that sex-specific mortality from tuberculosis was near identical, while historical studies suggest higher male mortality. This question is unresolved but it does emphasise that while a possible diagnosis of tuberculosis accounts for some of the young adult mortality seen in the Bahrain samples, especially at DS3, it fails to account for the excess

female mortality compared to male mortality amongst young adults.

Periosteal infections

Thirty-two individual and three isolated groups of bones show evidence of primarily periosteal reactions, though at times the dividing line between these reactions and osteomyelitis is fine (Table 8.13).

The periosteal lesions observed on the bone are indicative of numerous diseases including direct trauma and non-specific infectious organisms. As described, however, the clear patterning of lesions, the involvement of more than one area of the skeleton, and bilaterality suggests a more systemic cause in several cases.

The most common are lesions of the tibia and fibula, either symmetrical or of the two bones on one side (see Table 8.8). Other long bone lesions are predominantly single occurrences, no symmetrical lesions are recorded. Maxillary lesions and skull lesions occur in isolated cases but this may be more a reflection of preservation than underlying pattern.

Possible causes

Given the possible diagnosis of tuberculosis amongst this sample, the possibility needs to be considered that these lesions also reflect the disease. This, however, seems unlikely given the primarily periosteal nature of the lesions and their location on the diaphyseal surfaces of the lower leg, rather than joint surfaces and vertebrae.

Osteomyelitis is also a possibility but the symptomatic signs are absent. There is no evidence of sequestrum formation, nor is there any trace of an involucrum surrounding the bone. In addition, while osteomyelitis may attack the shafts of the long bones, it more commonly affects the metaphyseal surface. The lack of joint involvement also rules out septic arthritis.

As for tuberculosis, neoplasms, with the exception of Ewing's sarcoma, can be disregarded as a possible cause since the age of occurrence does not correspond to the age distribution seen here. Ewing's sarcoma can affect young individuals; yet the characteristic pattern of onion-peel lamellation is absent on the Bahrain bones.

The other lesions considered in differential diagnosis of tuberculosis do not apply to the last set of cases since the pathology described here is periosteal and proliferative rather than resorptive.

Two other diseases not considered for tuberculosis do need to be considered however. These are leprosy and Paget's disease. Leprosy is an extremely disfiguring condition associated both with the resorption of the extremities plus, less commonly, periosteal reaction (Moller-Christensen 1967). The reason for considering it, albeit briefly, is that leprosy is associated with destructive changes of the nasomaxillary area. These changes, however, are ultimately more destructive than those seen in the Bahrain material. While the skeletons from DS3 and Saar do show signs of infection in the facial and palate region, the lesion tends to be more superficial to the bone with only marginal destruction of the borders. In addition, the changes to the hands and feet seen in leprosy are absent in this population.

The other disease associated with periosteal reaction is Paget's disease which in its chronic form is associated with rapid bone remodelling and enlarged and distorted bones (Cook 1980). This disease, however, rarely occurs prior to 40 years of age and produces bowing and distortion, rather than the clearly defined elevation of bone seen in some of these samples.

Endemic syphilis?

One disease considered earlier in the context of differential diagnosis of osteomyelitic lesions does appear to fit the pattern of lesions seen in the Bahrain populations. Endemic treponematosis, or bejel, is associated with periosteal elevation of the tibiae and fibulae and with nasomaxillary destruction. The destructions on the tibiae and fibulae are marked by plaque-like apposition of porous and striated bone, often along the anterior crest of the bone. While sequestrae rarely occur, occasionally gummatous lesions may cause localized penetration into the cortex, though the disease is distinct in that these lesions penetrate from the outside in (Hackett 1981; Ortner and Putschar 1981).

This matches the pattern of periostitis seen in some of the Bahrain material but is not sufficient to prove that endemic treponematosis did exist in this population. In support of the diagnosis, however, are the cranial lesions. One of the earliest signs of bejel is infection centred around the nasal cavity and the palate. Initially this occurs without structural abnormality but eventually destruction may ensue. Secondly, cranial signs of endemic treponematosis, while not as extensive as venereal syphilis, include superficial cavitation and the development of circular porotic lesions which eventually become, in the healed phase, depressions surrounded by a margin of sclerosed bone, possibly with radiating lines in the centre (Hackett 1981).

There are four skulls in the present sample, plus one Iron Age skull, which have small isolated circular depressions (the Iron Age skull has two). The diagnosis of these as infectious rather than due to trauma is difficult and uncertain, but in two cases sclerosis was observed around the edge of the lesion; these, however, are very indefinite. More promising are two child skulls, one with a small circular porous lesion on the frontal. The lesion appears penetrative and is more marked on the outside surface than on the inner. This is different to the lesions of tuberculosis which are marked by internal erosion. A second child has a roughly circular erosive lesion of the frontal where the outer cortical

bone has been destroyed but the inner bone is unaffected. The lesion is not surrounded by porosity.

All of these cases however are questionable in some degree since the uneven nature of preservation means that they are not associated with complete skeletons. Yet several points do favour the possible diagnosis of treponematosis:

1. The bilateral nature of the lesion on the lower limbs affecting not only the tibia (liable to trauma) but also the fibula.
2. The apposition of striated bone, sometimes with small nodes, which in only one case penetrated the outer cortex.
3. The patterning of lesions which, on a population basis, are clearly concentrated on the tibiae, fibulae and maxillae
4. The fact that most lesions were healed by the time of death.
5. The age accumulative nature of the pathology and the tendency for females to be affected more than males, and for at least some young children to be affected.

The pattern is clearly most similar in some individuals to endemic treponematosis. The individuals with extensive periosteal response in the lower limbs including superficial cavitation and in particular the one individual in which this is also associated with maxillary lesions are classed as probable bejel (Nos. 40,41,45,36). Those cases where the maxilla and palate or the cranial bones (with sclerosis or porosity) are affected have been classed as possible treponematosis (Nos. 23,27–29,32,33,39,43,53). Similarly those with symmetrical periostitis of the tibia and fibulae are classified as possible treponematosis. For the remaining individuals the isolated nature of the periostitis, while possibly due to incomplete preservation, is not sufficiently strong evidence for the diagnosis, even questionable, of treponematosis.

It is suggested, however, that despite the presence of some cases of periostitis which are non-specific, the remainder accord well with the pattern expected in a population affected by syphilis. In particular the pattern matches that expected in endemic syphilis. Venereal syphilis tends to be marked by more extensive cranial symptoms, and more significant shaft expansion and irregular surfaces of the long bones. The age distribution also matches endemic rather than venereal syphilis, with the absence of congenital signs of syphilis (notched teeth etc.) amongst young children, female predominance and age accumulation of cases.

Endemic syphilis in the Middle East

As far as the Bahrain material goes, the skeletal lesions seen in this group are similar to those seen in neighbouring countries today (El Serafy 1972; Erdelyi and Molla 1984; Hamada and Rida 1972; Hudson 1958; Kanan and Kandil 1971). In addition, their similarity to suspected treponematosis cases from America (Powell 1991; Reichs 1989), suggests that endemic syphilis was well and truly established in the Middle East in its endemic form by the time of Columbus. The description of a medieval skeleton from England and one from Italy also supports this contention (Henneberg et al. 1992, Stirland 1991).

In the Bahrain populations it appears that about 2% of the mortality sample had skeletal signs of bejel. Since there is little assoicated mortality with bejel, this figure is possibly reflect of the living population and would represent some 7–10% of the affected (seropositive) population. It suggests, therefore, that about 30% of the population had contact with endemic syphilis. This level of infection is currently classed as mesoendemic (Vorst 1985).

In a community the disease tends to be contracted in early childhood. The spirochete may be passed on from other children or through communal drinking vessels (Hudson 1958). The initial infectious lesions are often mucous patches in the mouth which may in some patients be associated with a generalized non-irritating rash. These early lesions are frequently non-destructive although the stages of the disease tend to be overlapping (Hudson 1958). The disease may reactivate many years later. One precipitating factor is the reinfection of a woman from her breast-feeding child (Hudson 1958; Kanan and Kandil 1971). In this second stage, which does not occur in all patients, gummata of the skin, bone and cartilage may develop, sometimes with bony destruction particularly of the nose and palate (Erdelyi and Molla 1984). Those affected may experience nocturnal pain in their lower limbs which will sometimes be sufficiently severe to interfere with work and other activities (Hudson 1958; Powell 1991).

In general the disease is associated with only low to moderate mortality but high morbidity. As Powell describes, the disease was 'no doubt regarded as one of life's regular nuisances' (Powell 1991:179), though in cases where the secondary infection became widespread and septic no doubt high mortality could occur. In the Bahrain populations, however, its severity, except possibly amongst the very young, must be doubted. Firstly 50% of the lesions were healed by the time of death and secondly the disease is clearly age accumulative which is inconsistent with high levels of associated mortality.

In fact many of the same living conditions which would sustain tuberculosis in a population would also sustain endemic treponematosis. Close contact between individuals associated with dense occupation of an area, shared drinking and eating vessels, and personal contact particularly between children of different families all favour the spread of infection. In addition, infectious stress (such as caused by malaria and pneumonia) or even nutritional stress could precipitate lowered resistance and thereby cause the reactivation of infection (Hudson 1958).

The effect of the two conditions, however, is markedly different. Tuberculosis was a serious disease and cause of mortality especially amongst children and young adults. Treponematosis, while not a high cause of mortality, would

have placed an added burden upon young children, particularly if already fighting other infectious organisms. It would have resulted amongst adults in sporadic periods of decreased work capacity. It may have had an effect on increasing old adult mortality through the weakening effects of chronic infection, though impact of bejel on mortality prior to that cannot be expected to have been significant.

The other point of course in all of the discussion on infection is that the vast majority of these cases, with the exception of the possible tubercular cases, were healed by the time of death (61.5% of all infections were healed). Obviously while these individuals lived in conditions suitable to the spread of infection and a reasonably high proportion were affected by it, those who lived long enough to develop bone lesions also had good resistance. This suggests that nutritional stress may not have been significant in the population. Two age periods, however, still remain a problem. These are the youngest age group, especially 0–6 months, and young adult females, where the high level of mortality is unaccounted for by infectious skeletal lesions.

Endocranial lesions

In relation to young children the aetiology of endocranial lesions also needs to be considered. Opinion seems to be divided over the nature of these lesions. Initial work by Angel (Angel 1971) suggested that lesions on the internal cranium are the result of anaemia in very young children. Angel suggested that the lesions were the result of expansion of the diploe, destroying the inner table of the skull (Angel 1971). Mensforth and co-workers, however, described these same lesions as endocranial periostitis, stating that infection may cause inflammation of the periosteum allowing the storage of pus under the periosteum (Mensforth et al. 1978). This interpretation, however, is problematic since in the case of an acute infection such as neonatal or infant osteomyelitis it is difficult to see how fatal meningitis did not occur before bone lesions could develop (Discussion to Rose and Hartnady 1991). This would mean that possibly the cause of the lesion is not an acute pyogenic disease but a slower process such as congenital treponematosis, although the evidence for this is seen as very slender. In addition, congenital transmission of treponematosis is specifically ruled out in the case of endemic syphilis, as in the Bahrain groups (Grin 1956; Hudson 1958).

Against the diagnosis of periostitis is the specific age relationship of the lesion. Other periosteal reactions occur into adulthood in a similar distribution as amongst children, however in the Bahrain skeletons endocranial lesions are only observed amongst infants and young children.

Closer examination of the lesion helps little: due to the thinness of infant crania it can be difficult to distinguish between the inner and outer tables. In addition, the endocranial lesions observed in the Bahrain material are extremely variable. Some consist of spiculated bone radiating from a solid bone table (Plate 7) others are more cicatricial markings on the inner cranium, specifically in the area of the cranial boss with no real destruction of the inner table (Mensforth and coworkers described these as healed, Mensforth et al. 1978).

Location tends to be primarily in the area of the parietal and frontal bosses and the upper occipital, sometimes, though not necessarily, with clear radiating lines. At other times depressions from the meningeal arteries are visible. In the Bahrain examples there is no evidence for trauma and no relationship with periostitis on other parts of the skeleton.

The clearest and most statistically significant relationship in the Bahrain material is between these lesions and cranial porotic hyperostosis, the same pattern noted by Angel (1971). In addition it is noted that even in those individuals with incipient or minor ectocranial porosity, marked marrow hyperplasia has already occurred without bossing but with regular expansion. It is suggested that two possible causes are rickets and anaemia.

Rickets may be accompanied by the deposition of subperiosteal bone and the destruction of both cranial tables 'so that the entire thickness of the cranial vault has the porous appears of the diploe' (Ortner and Putschar 1981:274). The age distribution of endocranial lesions does in fact match the expected patterns of rickets —from 2 months to 4 years of age with the highest frequency between 6 months and 2 years. In addition the cranial changes tend to precede the characteristic long bone changes of rickets (Stuart-Macadam 1989). Against this possibility is the spiculated pattern of bone observed in many of the Bahrain skulls. It may however explain one case, which will be described later, where there are postcranial signs of rickets.

The alternative is anaemia. Evidence is equivocal on whether the marrow hyperplasia of severe anaemia affects the inner table. Reimann and Kuran (1973) suggest that the development of 'hair-on-end' pattern is the result of pressure of red blood cells on the trabeculation of diploe forcing outwards expansion and thickening of the inner table. Ortner and Putschar described the possibility of inner table destruction in thalassaemia major but state that this occurs much later and is always more limited than the outer table (Ortner and Putschar 1981). The photo they display, however, does demonstrate the same spiculated pattern seen in the Bahrain examples.

In addition, Lawson and coworkers note that in thalassaemia major the meningeal arteries become enlarged and tortuous, clearly visible on x-rays (Lawson et al. 1984). This is due to the increased activity of the marrow. The pressure of these enlarged and distorted arteries on the endocranial surface may explain the characteristic appearance of some endocranial lesions representing increased blood production rather than healed periostitis.

None of these possibilities present a full answer to the problem of endocranial lesions. The possibility of periostitis

cannot be ruled out, given the preservation of at least the outer table, and possibly the internal table of bone in some cases. However, if the lesions are to be accepted as periostitis, explanation is needed as to which organisms are causing the infection and through which portal. Obviously the infection was subacute, allowing time for bone changes to develop, and equally it must be specific to infants and young children.

Rickets and the deposition of osteoid appears to explain at least one of the Bahrain cases though it fails to account for the 'hair-on-end' appearance of other cases. Expansion of the meningeal arteries due to anaemia and marrow hyperplasia may be the cause, and is the most probable cause of cicatricial markings on the inner vault. Whether it is the cause of the more extensive spiculated lesions is still a matter for discussion. Whatever the diagnosis, however, in the Bahrain material the condition clearly operates in a synergistic fashion with porotic hyperostosis rather than with diffuse periostitis.

Therefore, rather than counted as an infectious lesion in this population, endocranial lesions will be included as part of the syndrome leading to porotic hyperostosis amongst young children. The seriousness of this sign cannot be ignored since in this one Bahrain population it is only seen amongst young children who died; no healed cases were found in children over four years of age, or for that matter in any children.

Conclusion to infection

It must be emphasised that amongst the Bahrain material there is a generally low prevalence of infectious lesions. Of the conditions identified and their associated diagnoses, three are associated with significant mortality: endocranial lesions (whether or not due to anaemia), osteomyelitis (affecting two individuals) and possible tuberculosis. The remaining conditions–periostitis, sclerosing osteomyelitis, secondary infection and possible treponematosis–are associated with significant levels of morbidity but not necessarily mortality.

This pattern of skeletal infections is comparable entirely to what is recorded in this same region historically and today. What needs to be accounted for are the infections which do not affect bone but do exist in the living conditions implied by these differing diagnoses. Prior to this, however, two groups of non-infectious lesions need to be discussed.

Bone deformity–rickets?

Description

As described in the discussion of endocranial lesions, one possible case of rickets was observed. In addition a minimum of four infants from DS3 had postcranial evidence of pathology other than that described as either porotic hyperostosis or infection. The cases are listed in Table 8.14.

Table 8.14
Bone deformity amongst DS3 subadults

	Location	Skeletal Elements Involved	Age
DS3:			
1.	73/18	L & R femur, L & R tibia	0–6ms
2.	70a/2	L & R femur, R tibia	2–3yrs
		R radius	1–2yrs
3.	83/30	Vertebrae, humerus	6–12ms
4.	83/29a	Ulna	0–6ms

The lesions consist of flaring and cupping of the metaphyses of both long and short bones, compression of the vertebral endplates, and distortion of the bones, including swelling near the distal ends of long bones. These changes consistently affected individuals between the ages of 0 to 3 years at the time of death.

Given the fragmentary nature of the burials and the disassociation of the bones it is difficult to distinguish any pattern of skeletal involvement other than that long bones are involved, and possibly the vertebrae.

The lesions are common to all cases and most marked upon the distal metaphyses, with the exception of the proximal humerus. The metaphyseal surface appears sunken and very porous while the surrounding layer of surface cortical bone, which has obviously continued to grow, is poorly organized, thin and striated in appearance. The resulting bone is flared at the metaphyses. The shafts are tubular with a squat appearance.

The lack of clearly associated cranial and other postcranial material (such as ribs) makes it impossible, not only to outline skeletal involvement completely, but also to calculate the percentage of individuals affected in the population. Percentages can, however, be estimated on the number of bones of individuals less than 6 years of age at death (Table 8.15) compared to other conditions such as porotic hyperostosis, this was an uncommon pathology affecting only a small proportion of those who died.

These pathological lesions are characteristic evidence of rickets or a related disorder (Ortner and Putschar 1981). Vitamin D is required for proper calcification of cartilage and mineralization of osteoid. When the individual has an inadequate supply of Vitamin D, the osteoblasts maintain their normal function, but because of the lack of mineralization and calcification, a mass of osteoid and decalcified cartilage builds up in the growth plate adjacent to the metaphyseal surface, resulting in cupping and flaring of the metaphysis (Pitt 1981).

Table 8.15
Frequency of bone changes amongst subadults less than 6 years

Bone Affected	No.	%
Femur	4/169	2.4%
Tibia	3/146	2.1%
Radius	1/124	0.8%
Ulna	1/173	0.6%
Humerus	1/250	0.4%

The location of these deformities on the skeleton is determined by the age of the child. In very young children the first signs are seen upon the skull, which at this stage is growing very quickly. The result is craniotabes–areas of softened bone due to the deposition of osteoid. This is followed by the characteristic cupping seen at the sternal ends of the ribs (rachitic rosary), distal radius and ulna, distal femur, tibia and fibula, and proximal humerus. Signs are sometimes visible on vertebrae as biconcave depressions on the upper and lower bodies (Pitt 1981).

Table 8.16
Diagnostic features of rickets and their presence at DS3

Rickets*	Presence at DS3
Craniotabes	nd
Rachitic rosary	nd
Pelvic deformity	nd
Cupping and flaring of metaphyses (in order):	
distal femur	x
proximal femur	x
proximal humerus	x
proximal and distal tibia and fibula	x
distal ulna and radius	x
Tubular appearance of long bones	x
Bowing of long bones	?
Concave depressions in vertebral endplates.	x

*Based on Ortner & Putschar (1981); Pitt (1981); Stuart-Macadam (1989).

A comparison of the signs of rickets and those on the DS3 skeletons demonstrates their conformity, where the relevant skeletal parts are present, with the diagnostic charactistics of this condition (Table 8.16).

It is evident that rickets is the most plausible cause of the changes in the DS3 population. This is consistent with the age distribution of lesions amongst the DS3 skeletons. Rickets is most common in early childhood, particularly during periods of rapid growth (Underwood and Margetts 1987).

Causes of rickets

Rickets can be caused by several factors:

1. most important, Vitamin D deficiency from inadequate UV radiation;
2. poor maternal stores, especially in premature, low birth weight children (neonatal rickets);
3. inadequate diet (generally secondary to 1);
4. nontropical sprue/mild chronic malabsorption;
5. hereditary disfunction of the renal tubules;
6. biliary fistula and biliary statis.
   (Oke 1972; Pitt 1981; Pitt 1991; Smith 1972)

The last three conditions tend to be uncommon. It is unlikely that they would be responsible for rickets in the percentages seen here at DS3, especially considering the absence of deformities amongst older children, and the absence of signs (in the case of hereditary conditions) amongst the smallest infants (Pitt 1981). Table 8.17 lists other specific signs of pathology related to rickets, none of which were found in the DS3 population.

Table 8.17
Rickets-related pathology

Renal osteodystrophy—signs of hyperparathryroidism.
X-Linked Hypophosphatasia—usually only moderate to mild changes of growth plate, minimal deformity.
Hypophosphatasia—wormian bones, craniosynostosis, advanced demineralization in newborn children.
Metaphyseal chondrodysplasia of the Schmid type—multiple small bone projections extend from growth plates.

(Based on Pitt 1981, Smith 1972)

This leaves three remaining causes which are interrelated: inadequate sunlight, prematurity, and inadequate dietary intake.

'Inadequate sunlight' is unexpected for the Middle East. Modern studies, however, demonstrate a high prevalence of rickets in several populations of the region (eg. Costeff and Breslaw 1962, Salimpour 1975, Underwood and Margetts 1987). The primary cause observed in these groups is that small children are rarely taken outside (to avoid the 'evil eye'), live in dark housing, and are heavily clothed in all seasons.

These cases of rickets in the Middle East, however, frequently occur as a result of a combination of causes. Apart from low exposure to ultraviolet radiation, the presence of marasmus exacerbates the condition; the combination of the two conditions results in apathetic and frequently immobile children, particularly until about 3 years of age (Underwood and Margetts 1987). Poor maternal health and low birth

weight of children also contributes to an increased frequency of rickets (Costeff and Breslaw 1962), as do frequent infections and diarrhoea (Salimpour 1975, Serenius et al. 1984, Underwood and Margetts 1987). Finally, a high cereal diet, particularly with a high level of phytic acid which interferes with calcium absorption may predispose children to vitamin D deficiency (Reinhold 1972, Berlyne et al. 1973).

Rickets, in itself, is not a major cause of mortality, rather it is a debilitating and possibly deforming disorder in young children (Jelliffe 1968). The lack of deformation seen amongst older children in the DS3 sample confirms that the deficiency is restricted to only a small percentage of the population who either did not survive or who healed completely prior to major deformation.

Rickets takes two basic forms:
hypertrophic: observed in well-nourished children with good muscle tone. The bone cortices are porous but thick, with narrow marrow spaces; distortion of bones is common.

porotic: seen in generally undernourished children. The bones are thin with porous cortices, and susceptible to fracture (Ortner and Putschar 1981).

A study of children with rickets in Tehran (Salimpour 1975) confirms the presence of these two forms along with intermediate cases.

The limited evidence from Bahrain makes it difficult to state clearly which form is present since, while the bones tend to be thick and tubular in shape, there is no obvious sign of the bowing which is often most severe in relatively well nourished children (Stuart-Macadam 1989). On the other hand, in severely malnourished children rickets becomes visible only in the recovery phase since it is a disease of growth (Jain et al. 1985). It appears from the skeletal evidence of rickets in Bahrain, combined with the presence of both acute and chronic growth disruptions, that early childhood was a time of particular stress. The fact that signs of rickets are apparent suggests that the cause is not severe malnourishment but rather a combination of causes such as frequent infectious and diarrhoeal episodes, along with inadequate exposure to sunlight.

The importance of rickets in this skeletal group is first, that it is evidence of the nature of childhood stress. Secondly, rickets is closely associated with respiratory and gastrointestinal infections, both as cause and effect, and with anaemia. Thus it is evidence of part of the cycle of morbidity in early childhood which ultimately results in the death of a high proportion of children.

Bone sclerosis and hypertrophy

Pathological conditions were recorded in several categories, none of which, strictly speaking, are mutually exclusive. These are traumatic, resorptive (porotic hyperostosis, osteoporosis), proliferative (periostitis, osteomyelitis etc.) or sclerosing/hypertrophic. Initially it was expected that this final condition would include neoplasms. In the entire sample, however, only two cases of benign osteomata were found. Instead it was observed that a large number of bones showed signs of excessive ossification of the ligaments and interosseus membranes, accompanied by some disorganization of the bone cortex. As a result more systematic recording was carried out, grading each bone into one of four categories:

1. Normal.
2. Marked accentuation of muscle markings, increased robusticity of the bone.
3. Ossification of ligamentous insertions and interosseus membranes, with protruding masses of bone, the beginning of joint ankylosis.
4. Ankylosis of joints, near complete ligament ossification.

Both each bone and each individual skeleton were scored according to this scale. In the case of individual cases attribution to either slight, moderate, or severe classes was on the basis of the majority of bones.

Description

Since increases in the rugosity of muscular attachments can be a generalized phenomenon of age, the analysis concentrates upon the moderate-severe changes where actual ossification of non-bony structures had occured. Table 8.18 demonstrates the distribution of these lesions throughout the adult skeletons from DS3. No subadult bones were found with any evidence of skeletal hypertrophy. Only bone with extensive muscular attachments showed signs of excessive ossification.

Table 8.18
Frequency of moderate-severe entheseopathy and ankylosis

Bone	% Mod-Severe	Bone	% Mod-Severe
Vertebrae:		Humerus	4.1
Thoracic	1.5	Radius	2.2
Lumbar	2.3	Ulna	3.9
Sacral	3.4	Hand	0
Innominate	5.1	Femur	1.2
Ribs	6.7	Patella	2.9
Sternum	12.7	Tibia	2.6
Clavicle	0.4	Fibula	3.6
Scapula	1.4	Foot	7.7

The skeleton was differentially affected. Major effects were seen in the thoracic area (7% of ribs; 13% of sterna). Fusion, or near fusion, of the sternocostal joints was frequent (12.7% of sterna). Apart from the articulations with ribs, the surface of the sternum was also affected with progressive ossification of the anterior ligaments covering the body.

In the vertebral column, the probability of bone ankylosis and ligamentous ossification increased down the spine. This

ankylosis could be readily distinguished from osteophytosis since the first ligaments to show signs of ossification were the supra- and interspinous ligaments along the neural arch. In addition osteophytic growth along the edges of the vertebral bodies was vertical rather than horizontal, the resultant effect being of 'candle wax' outgrowth rather than the 'parrot-beak' seen in vertebral osteophytosis. In addition there were no accompanying signs of joint degeneration such as porosity or eburnation of the articular surfaces.

The pelvis was also frequently affected, 5.1% of these had more definite signs such as ligament ossification and joint ankylosis, primarily at the sacroauricular joint.

In comparison the limbs were less affected. Changes in the humerus were marked by a general increase in midshaft diameter, increased robusticity of the deltoid tuberosity, gradual ossification of the ligaments at the sites of insertion, and ossification of the muscle attachments around the olecrannon fossa. In the radius and ulna ossification of the ligament insertions was generally obvious, particularly on the olecranon of the ulna and around the radial tuberosity. Marked ossification of the interosseus membrane was also observed, although complete fusion of the two bones was not found.

The most characteristic changes were seen in the bones of the leg. The foot was most frequently and severely affected with 7.7% of feet showing evidence of ossification, particularly of the talo-calcaneal ligament. Changes on the femur were generally non-specific consisting of an increased rugosity in the attachment of the linea aspera and the insertion of the patellar ligament. Changes in the patella were evidenced by the gradual ossification of the anterior patellar ligament so that, in severe cases, spicules of bone protruded vertically from the anterior surface. Some of the most obvious changes were seen in the tibia and fibula. These included ossification of the interosseus ligaments and marked ossification of the patellar ligament insertion. In severe cases spicules of bone protruded from each of these locations resulting in 2.6% of skeletons with ankylosis of the tibia and fibula.

Intrapopulation comparisons

When this condition was analysed by age a clear trend was immediately obvious (Table 8.19). No one from the 20–30 yr age group was affected. In the 30–40 yr age group only the pelvis, ribs, sternum, humerus, ulna and fibula had any evidence of moderate-severe changes. An increase was seen in the 40–50 yr age group but severe changes were only seen in a small number of bones. A much higher proportion of these bones were moderately to severely affected in the 50+ age group.

This clear age relationship is seen in an examination of the numbers of individuals affected. Cases of extensive joint ankylosis were only observed in the oldest age group. The condition is clearly age-related: not only did its frequency increase with age but severity is also clearly age-related suggesting a disease that slowly and steadily develops over time.

Table 8.19
Age distribution of moderate-severe changes

	30–40	40–50	50+
Cervical Vertebrae	0	0	0
Thoracic Vertebrae	0	0	14.3
Lumbar Vertebrae	0	0	21.4
Sacrum	0	0	15.4
Pelvis	1.3	9.4	12.1
Ribs	5.6	6.7	27.3
Sternum	11.1	0	57.1
Clavicle	0	3.4	0
Scapula	0	5.6	4.3
Humerus	3.7	0	14.3
Radius	0	0	16.7
Ulna	4.2	0	25.0
Hand	0	0	0
Femur	0	0	7.4
Patella	0	0	12.5
Tibia	0	0	15.8
Fibula	2.3	0	16.7
Foot	0	16.7	22.7

In order to account for age differences in the male and female samples, the frequency of bones affected by all degrees of sclerosis were compared for the 30–40 and 40–50 year age groups. (The youngest and oldest age group were ignored because of the small frequency of affected bones in the youngest group and the small sample size, particularly of females, in the oldest age group.)

The frequency of bones affected in the 30–40 year group was inconclusive since so few individuals experience changes by this age. In the 40–50 year age group (Table 8.20), the pattern of involvement of the corpus was similar between the sexes but overall females were less frequently affected than males in this age group. Statistically significant differences in frequency occur in the humerus, ulna and fibula.

Table 8.20
All categories of changes in the 40–50 age group by sex

	Males	Females
Cervical Vertebrae	0.0	0.0
Thoracic Vertebrae	12.5	22.2
Lumbar Vertebrae	12.5	11.1
Sacrum	28.6	25.0
Pelvis	61.1	50.0
Ribs	28.6	25.0
Sternum	60.0	20.0
Clavicle	44.4	25.0
Scapula	50.0	20.0
Humerus	64.3	16.7
Radius	75.0	41.7
Ulna	78.6	40.0
Hand	40.0	0.0
Femur	42.9	50.0
Patella	38.5	0.0
Tibia	22.2	12.5
Fibula	63.6	33.3
Foot	30.0	50.0

The condition is obviously linked initially to age. In its severe form, it was a disease of the oldest members of the society though there was obviously a progression in both prevalence and severity during adulthood. Secondly, even accounting for age, women tended to be less frequently affected and certainly less severely than males. The sexes tended to be differentially affected as well: both showed signs in the thoracic and pelvic area, but involvement in men much more frequently included the limbs and sternum. This suggests that, rather than a simple difference in the level of predisposition, there is also a possibility of either genetic or environmental factors affecting progression of the disease between the sexes.

Saar

There is no statistically significant difference between Saar and DS3 in the percentage of individuals affected by excessive ossification (Table 8.21).

Table 8.21
All changes at DS3 and Saar

	DS3	Saar
Cervical Vertebrae	3.4	2.2
Thoracic Vertebrae	11.4	5.7
Lumbar Vertebrae	6.9	7.4
Sacrum	16.1	16.7
Pelvis	31.8	24.7
Ribs	24.4	22.9
Sternum	27.3	21.7
Clavicle	18.3	5.7
Scapula	24.8	13.8
Humerus	32.9	21.2
Radius	26.6	25.3
Ulna	27.3	27.3
Hand	12.8	21.4
Femur	26.3	12.5
Patella	23.2	5.3
Tibia	20.2	11.9
Fibula	29.7	41.7
Foot	27.5	39.1

At Saar, like DS3, slightly more males than females are affected and moderate–severe cases are restricted to males. This is true even when age is accounted for.

Within the two samples, therefore, the same epidemiological pattern is prevalent. The condition is clearly age-related . Onset may occur within the early twenties at DS3 or within the thirties at Saar but moderate to severe cases do not occur until the 40+, particularly 50+ age group.

Summary

Overall, even when accounting for age men were both more frequently and more severely affected than women. Finally, differences occur between the sexes and between populations in the pattern of skeletal involvement. Trunk involvement tends to be similar in all groups but there are dissimilarities in the pattern of limb involvement. This all suggests a chronic and steadily developing disease, affecting eventually the majority of the population, men preferentially to women and which is related to some extent to environmental factors since differences in involvement exist not only between the sexes but also between different populations.

Differential diagnosis

Plate 8 is an x-ray of both a severely affected spine (from T8 to L4) compared to a normal vertebral column. While the pathological spine has been damaged postmortem, the difference in radiodensity is not due to preservation or the presence of dirt. The two specimens came from the same cemetery and both were cleaned prior to x-ray. Several characteristic features of this condition are demonstrated. There is a marked development of thick osteophytes from the central anterior body with additional ossification of the anterior spinal ligaments. Complete ankylosis has occurred on several vertebral bodies, especially L2–L4 with encroachment upon the intervertebral disc space. All intervertebral disc spaces show signs of marked narrowing. The posterior spine on T8 has a roughened surface with exostoses developed along the lower border. The most obvious difference between the two spines, however, is the degree of radiodensity. The pathological case is near opaque with markedly greater density of bone, indicative of osteosclerosis.

The pathological changes on this spine demonstrate the characteristic changes seen in skeletons classed as "severe". Diagnosis must obviously be based upon a consideration of those orthopaedic conditions accompanied by a progressive and generalized increase in bone production, ossification of tendons and ligaments, and joints, especially spinal, ankylosis.

Differential diagnosis needs to consider five possible causes of these conditions:

1. Osteoarthritis and vertebral osteophytosis.
2. Paget's disease.
3. Diffuse idiopathic skeletal hyperostosis (DISH).
4. Ankylosing spondylitis and other sero-negative arthropathies.
5. Skeletal fluorosis (Bullough and Vigorta 1984; Rogers et al. 1987).

Possible causes

The later stages of Paget's disease may result in an increase in the radiodensity of bone and coarsening of trabeculae. However, this thickening is localized often affecting only the outer borders of the vertebral body and resulting in a 'picture frame' appearance (Bullough and Vigorta 1984). In addition the disease is often limited to the involvement of one or two bones rather than the entire skeleton, and does not involve extensive calcification of tendons and ligaments (Ortner and Putschar 1981).

Severe vertebral osteophytosis is marked by the development of beak-like osteophytes which project laterally from the vertebral body. Ankylosis, however, tends to be rare and

generally affects vertebral bodies in isolation. Osteophytosis is generally not associated with generalized ossification of tendons and ligaments. Osteoarthritis similarly tends to affect joints in isolation rather than the whole skeleton and very rarely results in ankylosis (Bullough and Vigorta 1984; Manchester 1982).

DISH, or Forrestier's disease, is a disease primarily of older individuals, especially men (Rogers 1982). The condition is diagnosed by the presence of thick bridging osteophytes especially along the right antero-lateral aspect of the thoracic spine. This may result in the ankylosis of several contiguous vertebral bodies (Vernon-Roberts et al. 1974). While the cervical and lumbar spine may be affected, the condition begins in the thoracic area with the calcification and ossification of the paraspinal ligaments. The posterior longitudinal ligaments may become ossified and there is sometimes ankylosis of the apophyseal joints (El Garf and Khater 1984). In general, however, the intervertebral disc space is preserved without marked narrowing (Vernon-Roberts 1974, but see Harris et al. 1974) and the facet joints are almost always normal (Rogers et al. 1987). There may be involvement of other joints apart from the spine. The most commonly affected areas are the pelvis, upper femur, heel and knee. On the pelvis, 'whiskering' along the ilia is prevalent, and there tends to be para-articular osteophytosis with calcification of ligaments such as the sacrospinous, calcaneal and patellar ligaments. In a large sample surveyed (El Garf and Khater 1984), skeletal findings were restricted to the spine and lower extremities. The condition rarely encompasses osteoporosis and costochrondral ossification is similarly rare (Manchester 1982). The prevalence of the disease in an autopsy population is between 6–12% of all individuals. In this same series 65% were male and 88% were more than 50 years of age (Rogers 1982).

In contrast, ankylosing spondylitis primarily affects men in late adolescence/early adulthood (Riley et al. 1971). It is marked by gradual ankylosis of the spine beginning initially in the lumbar region. The vertebral bodies are thickened along the anterior aspect and thin vertically directed syndesmophytes unite contiguous vertebral bodies. This results finally in exaggerated kyphosis and obliteration of the intervertebral discs so that eventually the spine moves as a single unit. Ankylosis however, often intially occurs in the sacroiliac joint and only later in the disc (Bullough and Vigorta 1984). Characteristically the joint surfaces show signs of erosion. In addition the vertebral bodies have a 'squared' appearance and osteoporosis is common. About 20% of cases show evidence of peripheral joint involvement, predominantly in the lower limbs, including the metatarsophalangeal joints (Manchester 1982, Riley et al. 1971). The changes tend to be arthritic in nature with destruction of the joint surfaces.

In other seronegative spondylo-arthropathies the major changes are erosive in nature. Vertebral ankylosis in Reiters syndrome is similar to AS but frequently "skips" vertebral bodies. Changes in other joints are assymmetrical (Rogers et al. 1987). In psoriatic arthropathy the small joints of the hands and feet may be affected with severe erosive changes (Rogers et al. 1987; Zias et al. 1996).

The final possible diagnosis is skeletal fluorosis caused by the ingestion of excessive fluoride, often from water sources. Fluorosis is marked by increased bone production, thickening and coarsening of bone trabeculae, with a corresponding increase in bone density resulting in a 'ground glass' appearance on x-rays (Bullough and Vigorta 1984). The entire skeleton is affected. The cranium becomes thick and heavy with gradual obliteration of the diploe, while ossification of ligaments results in the development of irregular margins around the foramen magnum. Postcranially the bones become heavy and irregular with abnormal prominence of muscle and tendinous insertions. Ossification of spinal ligaments is common especially the ligamenta flava, intratransverse and interspinous ligaments. The vertebral bodies appear larger than normal and eventually there is complete fusion of the vertebrae by thick osteophytes. These changes are most common in the cervical and lumbar regions. The ribs appear large and have roughened surfaces due to ligament ossification. Ankylosis of the costo-vertebral and costosternal joints may occur with consequent 'freezing' of the chest cavity in an aspirated position (Jolly et al. 1969). Calcification of ligamentous and tendinous insertions is most marked on those areas of the skeleton subject to strain, especially the linea aspera, the interosseous membrane between the radius and ulna and the tibia and fibula, the sacrotuberous and sacrospinous ligaments (Moller and Gudjonsson 1967). In advanced cases ankylosis may occur in these locations (Teotia and Teotia 1988). Finally teeth are also affected within the population, though not necessarily within the same individual, with mottling, brown staining and pitting of the enamel (Moller 1982). The disease tends to affect older adults and more frequently men than women (Jolly et al. 1969).

Each of these diseases has similarities to the condition described at DS3 and Saar. Osteoarthritis and Paget's disease are least likely since they do not involve extensive ossification of tendons and ligaments, nor multiple joint ankylosis. The erosive lesions of Retier's syndrome and Psoriatic anthropathy also have not been observed in the Bahrain material. Table 8.21 lists the chief diagnostic features of the remaining conditions as compared to the Bahrain material. While the vertebral changes closely resemble DISH, the change in bone density, the involvement of the vertebral facets and the symmetrical nature of alterations in the thoracic vertebrae rule it out as a probable cause. The skeletal material from Bahrain demonstrates all the skeletal signs of fluorosis especially considering the large number of cases where there is increased but non-specific rugosity of the bone.

Table 8.22
Diagnostic features of possible causes compared to Bahrain

	DISH	Ankylosing Spondylitis	Fluorosis	Bahrain
Age	>40 yrs	c 20 yrs	Older adults (prolonged habitation, except in extremely high F areas).	>40 yrs
Sex	M > F	M > F	M > F	M > F
Vertebral Ankylosis	Thoracic	Late total	Lumbar first, later total	Lumbar, then total
Vertebral Bridge	Thick o'phytes	Thin syn'phytes	Thick osteophytes	Thick o'phytes
Lateral	Thoracic: R>L	Central	Central	Central
Squaring	Absent	Present	Absent	Absent
Vertebral facets	Unaffected	Fused	Fusion	Fusion
Costovert/sternal joints	Ligament calcification	Arthritic	Ligament calcification	Ligament calcification
Bone density	Normal	Osteoporotic	Osteosclerotic	Ostesclerotic
Peripheral joints	Lower mainly	Lower primary	Lower and upper	All
Dental mottling	Absent	Absent	Present	Present

Source: based on Manchester (1982), Jolly et al. (1969), Riley et al. (1971), Vernon-Roberts et al. (1974).

This is further confirmed by the results of analysis of fluoride levels within bone from several individuals (Table 8.22). In order to examine the effects of diagnetic change, samples were taken from both the outer surface of the bone and from a mid-section. While the bones from one individual had no detectable fluoride, the other two adults had abnormally high levels, corresponding to those recorded for fluorotic adults. Both of these individuals demonstrated only slight evidence of fluorosis: an increased rugosity of skeletal markings. A severely fluorosed skeleton from a Bronze Age cemetery on the Island had bone levels of 1000 ppm (Frohlich et al. 1989) The analysis of soil confirms that these levels are not a result of contamination. In all, the chemical analysis confirms the possible diagnosis of fluorosis.

Table 8.23
Fluoride levels from adult bone samples and comparisons

	Age	Sex	Sample	% dry wt
Fluoride (ppm):				
<1	Adult		Iliac Crest	0.019–0.073*
0.2			Bone	0.053**
1.0				0.138
2.6				0.267
4.0				0.413
Normal	Adult			0.062–0.92
Bahrain				
BS40	40+	Male	Femur	1.000***
70A/13	Adult	?	Mid-section	0.000
			Outer surface	0.000
81/38	Adult	?	Mid-section	0.210
S5/38	45+	Female	Mid-section	0.210
			Outer surface	0.280
70a/13			Soil	0.000
S5/38			Soil	0.000

*Zipkin et al. (1958); **McClure et al. (1958); ***Frohlich et al. (1989).

Fluorosis

Skeletal fluorosis results from the ingestion of excessive amounts of fluoride. Intoxication can result from acute doses, often in industrial situations, but more commonly the condition is chronic due prolonged intake of high levels of fluoride. The most common natural source of fluoride in these cases is water, especially well and spring water where the surrounding substrata contains fluoride (Teotia and Teotia 1988).

Fluoride is rapidly absorbed into serum within the stomach and upper intestine. The absorbed fluoride is carried to the bone where it is deposited in the bone producing fluoroapatite. This occurs most rapidly in the trabecular portion of the bone (Teotia and Teotia 1988). The abnormal bone has an increased density visible in cases of excess intake. In addition the fluoride stimulates the formation of new irregular bone at the sites of tendon and ligament insertions, resulting in gradual ossification. In the presence of adequate dietary calcium, the main picture is of osteosclerosis; in cases however where dietary calcium is inadequate the fluoride may result in secondary hyperparathyroidism leading to bone loss, so that bone density may include areas of both sclerosis and porosis (Teotia and Teotia 1988). This osteoporotic type of skeletal fluorosis (genu valgum) occurs in children and younger adults, particularly in areas with extremely high levels of fluoride in water (Christie 1980; Krishnamachari 1986).

Most commonly the osteosclerotic type of fluorosis only develops after 15 to 20 years of continuous exposure to water containing high levels of fluoride (Jolly et al. 1969). The minimum water level ever reported as causing changes is 0.7ppm (Misra et al. 1988) but there is not a clear linear relationship between the amount of fluoride in drinking water and the development of skeletal fluorosis (Chibole 1987). Studies in temperate, developed countries have demonstrated no significant signs of skeletal fluorosis in communities with water levels of 4 ppm (McClure et al. 1958), yet work in some Indian villages has demonstrated skeletal fluorosis when the water level is much lower (Moudgil et al. 1986; Teotia and Teotia 1988). The influencing factors other than water level appear to be period of exposure, climate, other trace elements in the water, dietary intake of fluoride, nutritional status, water storage methods, work patterns, and tea drinking habits (Jolly et al. 1969).

Temperature is a major factor in fluorosis both due to increased water intake (Galagan and Lamson 1953) and evaporation of standing water (Nanda et al. 1974). The chemical constituents of water also have an effect on the physiological uptake of fluoride. Calcium and magnesium in water tend to inhibit fluoride ingestion while high sodium levels and alkalinity promote fluoride ingestion (Pinet and

Pinet 1968). Similarly a low dietary intake of calcium and phosphorus is implicated in elevated levels of skeletal fluorosis. General nutritional status also plays a role since malnourished individuals appear to be more prone to develop skeletal fluorosis (Massler and Schour 1952).

Sources of fluoride other than water also play a role. Plants irrigated with water containing fluoride contain low levels of fluoride. Fish also may contain high levels of fluoride from sea water, as does sea salt which is often used in cooking (Moller 1982).

Finally, work in India has demonstrated that manual labourers are more likely to develop skeletal fluorosis than their sedentary counterparts (Pandit et al. 1940). This is probably because people working outside tend to drink more water. Moreover it appears that the development of new fluorotic bone occurs at those sites most subjected to strain and minor trauma (Jolly et al. 1969). These factors may all predispose certain communities towards fluorosis.

Fluoride and Bahrain

This current study is not the only discovery of skeletal fluorosis amongst archaeological populations from Bahrain. Frohlich et al. (1989) describe a Bronze Age skeleton from Bahrain with advanced skeletal fluorosis and a bone level of 1% fluoride. Similarly dental fluorosis has been recorded in a series of skeletal samples from the Island (Littleton 1987). In addition there are modern cases of dental fluorosis documented on the Island (Barnes 1981), while cases of skeletal fluorosis have been described from nearby Qatar (Azar et al. 1961), coastal Saudi Arabia (Walters 1954), Sharjah, and Abu Dhabi (Fitzgerald-Finch 1981).

The traditional source of water on the island is spring water arising from natural aquifers which originate in eastern Saudi Arabia. A variety of modern tests upon this ground water have produced for various areas range between 0.5 and 1.5 ppm (Matter 1985, Musaiger and Khunji 1990). This variability in water levels between locations tends to be due to local variation in the substrata plus the depth of water and degree of sedimentation. There is an error involved in translating these levels back into the past since differences in average temperatures in the past, the amount of rain and the water levels of the acquifers would all cause variation in the fluoride levels.

In terms of geomorphology, however, significant variations in the level of fluoride in underground water would not be expected over this period, unless massive clearance and erosion had occurred resulting in substantial leaching of fluoride from surface soil. It is suspected that 2000 years is not sufficient time for these changes to occur (M. Bird, pers. com.). At the same time there is no evidence on Bahrain for this sort of activity, while the direction of the change, resulting in an increased level of fluoride in the present, would not explain the past frequency of fluorotic lesions.

There are two ways of dealing with this: one is to examine the prevalence of fluorosis in comparable living populations with varying water levels, and the other is to look at the levels of fluoride in bones from a variety of areas.

Table 8.23 lists comparative percentages of skeletal fluorosis with the comparable water levels. There is wide variation in the percentage of people affected in areas of similar water levels. In general, however, the levels of dental fluorosis observed amongst historical groups in Bahrain are comparable with levels seen at fluoride contents of between 1–2 ppm. The levels of skeletal fluorosis are less clear. Between 1–2.5 ppm, the percentage of individuals with skeletal fluorosis varies from 2.4 to 40.4% of the population. The percentage of affected individuals in the Bahrain sample would seem to imply either higher levels of fluoride in the water then as compared to now (c2.4 ppm rather than 0.5–1.5 ppm) or the presence of several predisposing factors amongst historic Bahrain communities.

Table 8.24
Levels of fluoride in water with the incidence of fluorosis

Area	F (ppm)	% fluorotic	% severe	Source
Punjab	0.3–1.4	30	3.6*	Jolly et al. 1973
Sahara	<1.5	0		Pinet &Pinet 1968
	1.5–4.0	30		
Punjab	1.4		2.4	Jolly et al. 1969
	2.3	10.0	23.0	
	3.0–3.6	19.6–33.1		
	5.0	60.0		
	8.5–9.7	58.9–80.7	10.0	

*x-ray evidence

Part of the disparity, however, can be explained by the nature of the Bahrain sample which is comparable to an autopsy population rather than a living population. Since chronic skeletal fluorosis is a severe disease, sufferers may have a shortened life expectancy, thus increasing their likelihood of being represented in the skeletal sample. In addition recognition is substantially greater on dry bone than among living individuals which may account for the higher than expected number of cases. This would explain the high proportion of severe cases compared to all cases in the group. It fails to explain, however, why there are **any** crippling cases at all, given the modern water levels.

Fluorosis is categorised into three stages:
1. blurring and coarsening of trabeculae;
2. merging of trabeculae, narrowing of the medullary cavities, early ossification of ligaments;
3. marbled bone, irregular periosteal thickening and more extensive ossification (Jolly et al. 1969).

These can be compared to the three stages used for recording the degree of lesions amongst the Bahrain skeletons. Thus Jolly's Stage 3 of crippling fluorosis can be seen as roughly

equivalent to the Stage 3 of skeletal recording. It was initially claimed that crippling fluorosis required water levels of more than 10 ppm before it occurred (Jolly et al. 1969). More studies, however, have demonstrated that in many populations crippling occurs above 3 ppm, and can occur at water levels of 1.35–1.5 ppm (Pinet and Pinet 1968) given the presence of predisposing factors.

On Bahrain itself, it can be hypothesized that some of these factors applied. Firstly high temperatures, particularly in summer, plus the shallowness of the subsurface water, would all increase the fluoride level in water, as would any storage of water in pots which presumably would have occurred in the case of household water. Since the level of sodium in the water tends to be high, fluoride intake would be increased (Musaiger 1990).

In addition sea water in the Gulf contains between 3.36–8.72 ppm resulting in a high fluoride content in fish (Azar et al. 1961). Since fish traditionally is a major source of protein on the Island this would also be a reason for elevated levels of skeletal fluorosis. With regard to nutritional adequacy it is difficult to draw any conclusions, but historically diets within the Middle East region tend to be low in calcium which would predispose the population, particularly those doing heavy agricultural labour, to fluorosis (El Tannir 1959; Walters 1954). It is obvious that, despite only moderate levels of fluoride in the ground water, the number of predisposing factors present on Bahrain means that skeletal fluorosis was a health problem for this community. The levels of fluorosis observed are equivalent to those found in areas of low chronic endemicity in India and the Sahara (Teotia and Teotia 1984; Pinet and Pinet 1968).

The effects of fluorosis

In skeletal fluorosis there is only a loose link between the degree of skeletal lesions and disability except in extreme cases (Chen-Yueng et al. 1983). There is however, a general progression in the clinical course of fluorosis. Early signs are vague pains and arthralgia. This generally progresses to backache, pain in the spine, and signs of stiffness and rigidity as well as constipation. With increasing calcification of tendons and ligaments there is a limitation of joint movement and inability to close the fists. The final stages of the disease are associated with Stage 3 and include difficulty in walking, with a generalized attitude of flexion and ankylosis until the the spine and chest become fixed and the sufferer is crippled (Teotia and Teotia 1988). In rare cases neurological complications occur due to compression of the spinal cords and radiomyelopathology (Misra et al. 1988; Naidu et al. 1988).

Faccini and Teotia (1974) described ten patients with typical signs of moderate to severe skeletal fluorosis. While three had flexion deformities of the spine none were completely immobilized despite extensive ossification. In addition they state that 'all were able to perform, at least, domestic work' (Faccini and Teotia 1974:47). This disparity between the physical signs of fluorosis and its effects is important to remember when gauging what effect fluorosis has upon a population.

A second factor to be accounted for is that the degree of disability experienced is often related to physical strain through life (Pandit et al. 1940). It appears that the bones most subject to stress are most likely to develop lesions. Thus in India male agricultural workers tend to develop exostoses in the lumbar region and lower limbs whilst amongst females changes are most common in the wrist, shoulder and neck (Jolly et al. 1969).

Applying these two factors to the skeletal population from Bahrain, it is obvious that the effects of fluorosis would not be apparent except amongst those over 40 years of age and, in particular, males. In the DS3 group, 2.6% of adults showed evidence of joint ankylosis. These were all men over 50 years of age at the time of death. There are no signs of quadriplegia as evident in the Bronze Age skeleton recorded by Frohlich and co-workers (Frohlich et al. 1989). Nevertheless, in terms of heavy labour it is unlikely that, given the extensive vertebral ossification, these men would have been full participants in the workforce, though there may have been little limitation in their performance of lighter tasks. In the younger age groups, apart form increasing stiffness, there may have been little impact of this disease. The same applies for the Saar population where only 1.5% of the population had signs of joint ankylosis.

The relationship between sites of skeletal involvement and work strain is important given the variation in the pattern of male and female involvement at DS3. While spinal lesions are similar between the sexes, the males had more upper limb involvement, particularly of the clavicles, scapulae, and humeri; women more frequently had lesions on their feet. It suggests that there were sex-specific tasks performed where men were involved in manual work involving the upper body. There are two possible explanations, neither mutually exclusive, for the pattern of male involvement. First, date palm cultivation requires men to work amongst the tops of trees both harvesting and then fertilising the plants, and presumably places strain upon the arms while the result of the body is supported. Secondly fishing, in particular hauling on nets, could also place strain upon the upper skeletal girdle. It may be supposed that, while men were primarily involved in these two tasks, women were responsible for the gardening of crop plants between the date palms and for household tasks, both occupations which would place strain upon the lower limb. Again these explanation need not be mutually exclusive, while it cannot be assumed that these patterns of labour were exclusive to either sex.

The importance of skeletal fluorosis is the fact that it may have meant that older adults were not as economically independent as younger members of the society. At least some would have had difficulty in moving freely, making their performance in heavy labour difficult, and in severe cases placing a burden upon younger members. It does,

however, appear that given the low life expectancy this most extreme instance only rarely arose.

Conclusion

The examination of pathological lesions and description in the light of differential diagnosis has identified four major conditions affecting the two Bahrain populations. These are thalassaemia, probably in conjunction with iron defiency anaemia, which affected infants and young children with high associated mortality. Treponematosis and periostitis affected the population at all ages but, while responsible for periods of sickness, did probably not account for much of the mortality. Tuberculosis, on the other hand, would have had a significant impact upon mortality, particularly of young children and young adults. Skeletal fluorosis obviously affected a large proportion of the adult population but since its severest effects were limited to the oldest age groups again associated mortality would be low. Rickets is not counted as a major condition since it only affected both a small percentage of those who died. Its importance lies with the evidence from hypoplasia and growth retardation of repeated infectious stress during infancy and childhood, particularly after the first six months. In order to determine the exact impact of these diseases, however, we need to know clearly how they did interact with each other, their specific relationship to mortality and at what ages, and their impact upon an individual's life and therefore village survival. The first three questions can be addressed by examining the causes of death, the last two by analysis of survivorship.

Chapter 9

Step 4: The causes of death

Introduction

While the analysis of palaeopathology has identified possible diseases which, on the basis of epidemiological models, had a varying impact on mortality, it fails to determine the extent and direction of this impact in any detail. Cause of death analysis, as devised by Preston (1977) and modified by Palkovich (1978) for skeletal populations, begins to address these issues. Basically this form of analysis attempts to measure 'the actual contribution made by a disease to mortality, including whatever impact it may have on mortality from underlying causes' (Preston 1977:19). It is based upon the realisation that much of the variability in mortality patterns between populations with the same crude death rate is the result of differences in the causes of death.

Preston, in analysing mortality, was primarily interested in creating a model of average experience, i.e. particular causes of death with particular life expectancies (Preston 1977). The usefulness of the concept in this analysis, however, is that it helps to address the issue of whether differences in the age structure of mortality in skeletal samples can be explained by different causes of death. In relation to the two Bahrain samples the issues that need to be addressed are: what are the causes of death, does the cause of death pattern explain the particular structure of mortality, does skeletal pathology reflect mortality or are the skeletal indicators too non-specific, are the causes of death the same in the Saar and DS3 populations, and what are the implications of these causes of death for environmental and social reconstructions?

Cause of death analysis allows assessment of the impact of disease and assessment of the interconnections between pathology. Since it allows acknowledgement of these interconnections, it becomes possible to isolate more specific epidemiological patterns within skeletal groups.

Method

Following Palkovich (1978), the relationship of individual skeletal pathology to mortality was examined by analysis of variance (ANOVA). This statistical test calculates the amount of variance in one variable (age-at-death) which can be explained by the combined effects of other variables (eg. porotic hyperostosis, fluorosis, localized periostitis, widespread periostitis and osteomyelitis). The resultant r-squared value is the amount of variance in the dependent variable which can be attributed to pathological categories. Multiple R is a measure (from 0 to 1) of the association between the dependent variable (age at death) and all independent categories (pathological conditions) combined. Further analysis using multiple classification analysis measures the average age effects of each pathological class, for example the presence or absence of porotic hyperostosis. A grand mean is calculated which, in this instance, represents the average age of mortality in the cases analysed. Taking the adjusted deviations for each category (eg. Class 1, Porotic Hyperostosis) from this age indicates the average age at which each of these categories of pathology had their greatest impact. A deviation close to zero indicates that the age distribution of the pathology mirrors the age at death structure of the whole population. Beta scores generated in the analysis are a measure of the relative effect of each individual condition on age at death, after allowing for interactive effects between conditions (Nie et al. 1986). This allows for statistically non-significant pathology to be identified. ANOVA and multiple classification analysis was run using SPSSX

Results

Using both samples, an ANOVA was run testing for the effects of porotic hyperostosis, fluorosis, localized periostitis, widespread periostitis, osteomyelitis and postcranial porosity upon the age distribution of mortality (Table 9.1). Categories such as trauma were not used due to the small numbers of individuals affected, while cribra orbitalia was not included at this stage due to the strong relationship between it and porotic hyperostosis. The combined effect of these conditions on mortality was significant ($p(F)=0.000$) although widespread periostitis and osteomyelitis did not have any significant independent effect. These conditions, in total, explained only 43.5% of the total variance in age at death indicating that while the chronic conditions these lesions represent were significant they failed to account entirely for the pattern of mortality.

The average age effects of each variable were also calculated as the adjusted deviation for each variable class from the grand mean (Palkovich 1978). The average age of death for all individuals combined was 25.8 years. Minor deviations from this indicate that the average age effect of this particular condition varied little from the overall age distribution of the group, but major differences are indicative of distinctive age relationships. Thus porotic hyperostosis, if present, had its average effect on those around 15 years of age, reflecting its impact on subadult mortality. Localized and widespread periostitis, on the other hand, affected a slightly older age group than average (around 30 to 35 years) which is indicative of the chronic nature of these two conditions. Osteomyelitic lesions, in contrast, were most severe on those around 20 years of age, a slightly younger age group than average. Fluorosis was strongly associated with a much older age group: c 44 years. In contrast postcranial porosity had a stronger effect on a much younger age group (10 to 15 years).

Table 9.1
ANOVA analysis for all individuals from DS3

Source of variation	Sum of squares	All Individuals DF	Mean square	F	Sig. of F
Main effects	32908.3	6	5484.7	28.7	.000
Porotic hyperostosis	5527.2	1	5527.2	28.9	.000
Localised periostitis	2544.5	1	2544.5	13.3	.000
Widespread periostitis	199.8	1	199.8	1.0	.308
Ostemyelitis	190.4	1	190.4	1.0	.319
Fluorosis	14105.3	1	14105.3	73.8	.000
Postcranial porosity	1251.1	1	1251.1	6.5	.011
Explained	32908.3	6	5484.7	28.7	.000
Residual	42799.1	224	191.1		
Total	75707.4	230	329.2		

784 cases were processed; 533 were missing

Multiple Classification Analysis

Age by
Porotic Hyperostosis
Localized Periostitis
Widespread Periostitis
Osteomyelitis
Fluorosis
Postcranial Porosity

Grand mean= 25.75

Variable and Category	n	Unadjusted deviation	Eta	Adjusted for independents	Beta
Porotic Hyperostosis					
0: Absent	178	3.97		2.83	
1: Present	53	-13.34		-9.49	
			.40		.29
Localized Periostitis					
0: Absent	208	-1.29		-1.12	
1: Present	23	11.70		10.10	
			.21		.19
Widespread Periostits					
0: Absent	218	-.47		-.23	
1: Present	13	7.86		3.87	
			.11		.05
Osteomyelitis					
0: Absent	226	.03		.14	
1: Present	5	-1.35		-6.14	
			.01		.05
Fluorosis					
0: Absent	191	-4.44		-3.70	
1: Present	40	21.18		17.66	
			.54		.45
Postcranial Porosity					
0: Absent	222	.94		.49	
1: Present	9	-23.25		-12.04	
			.26		.13
Multiple R squared					.435
Multiple R					.659

The above analysis indicates the close relationship between pathological conditions of the skeleton and the cause of death. Considering the relative effects of each condition, fluorosis and porotic hyperostosis contributed most to the observed pattern of mortality. These relationships need not be causal but a reflection of the age distribution of chronic disease.

In order to examine the relationship between the nature of the lesion (whether active or healed), three conditions were examined in greater detail: cribra orbitalia, infection (incorporating the three types of lesion), and fluorosis (Table 9.2). These three conditions account for even more variability than the preceeding analysis: 52.4% of the variance in age at death. Each variable contributed a significant amount to the explanation of the mortality pattern. Average age effects mirror the preceding analysis. Active cribra orbitalia most affected subadult age groups, while those with healed cribra orbitalia were slightly older on average. Healed infectious lesions affected an older age group although sample numbers are very small. The chronic effects of fluorosis are evident with the pattern of increasingly severe lesions matched with an increasing age at death.

Since fluorosis and porotic hyperostosis have such strong effects on either end of the age spectrum, it was decided to examine the relationship between pathological lesions and age at death for subadults and adults separately.

Table 9.2
ANOVA analysis for all individuals

Source of variation	All Individuals Sum of squares	DF	Mean square	F	Sig. of F
Main Effects	38285.3	10	3838.5	20.8	.000
Cribra Orbitalia	11144.4	2	5572.2	30.3	.000
Infection	2322.4	5	464.5	2.5	.030
Fluorosis	15220.5	3	5073.5	27.6	.000
Explained	38285.3	10	3828.5	20.8	.000
Residual	34718.0	189	183.7		
Total	73003.2	199	366.9		

784 cases were processed; 584 cases were missing

Multiple Classification Analysis

Age by
Cribra Orbitalia
Infection
Fluorosis

Grand mean= 24.99

Variable and Category	n	Unadjusted deviation	Eta	Adjusted for indepdendents	Beta
Cribra Orbitalia					
0: Absent	95	5.25		3.45	
1: Present	47	-17.48		-13.76	
2: Healed	58	5.56		5.50	
			.51		.40
Infection					
0: Absent	163	-2.21		-1.48	
1: Local Periostitis	14	9.66		8.79	
2: Healed Periostitis	9	5.23		1.78	
3: Widespread Periostitis	2	-11.49		-2.12	
4: Healed Widespread Periostitis	8	16.89		8.09	
5: Osteomyelitis	4	16.26		10.29	
			.29		.18
Fluorosis					
0: Absent	164	-5.14		-4.21	
1: Slight	26	22.32		17.87	
2: Moderate	8	25.95		21.93	
3: Severe	2	27.51		25.54	
			.58		.47
Multiple R squared					.524
Multiple R					.724

Table 9.3
ANOVA for subadults only

Source of variation	All Subadults Sum of squares	DF	Mean square	F	Sig. of F
Main Effects	101.6	4	25.4	3.614	.034
Cribra Orbitalia	85.4	2	42.7	6.076	.014
Postcranial Porosity	5.5	1	5.5	.786	.391
Endocranial Lesions	6.5	1	6.5	.931	.352
Explained	101.6	4	25.4	3.614	.034
Residual	91.4	13	7.0		
Total	193.0	17	11.3		

297 cases were processed
279 cases were missing.

Multiple Classification Analysis

Age by
Cribra Orbitalia
Postcranial Porosity
Endocranial Lesions

Grand mean=3.22

Variable and category	n	Unadjusted deviation	Eta	Adjusted for independents	Beta
Cribra Orbitalia					
0: Absent	2	-2.47		-3.17	
1: Present	15	-.26		-.12	
2: Healed	1	8.78		8.08	
			.68		.67
Postcranial Porosity					
0: Absent	15	.28		.25	
1: Present	3	-1.38		-1.27	
			.19		.17
Endocranial Lesions					
0: Absent	12	.51		.45	
1: Present	6	-1.02		-.90	
			.22		.19
Multiple R squared				.526	
Multiple R				.726	

Table 9.4
ANOVA analysis for all individuals from DS3

Adults only

Source of variation	Sum squares	DF	Mean square	F	Sig. of F
Main effects	8953.5	11	814.0	8.6	.000
Porotic hyperostosis	1328.8	2	664.4	7.0	.001
Infection	957.3	6	159.5	1.7	.129
Fluorosis	5666.2	3	1888.7	19.9	.000
Explained	8953.5	11	814.0	8.6	.000
Residual	13744.8	145	94.8		
Total		22698.3	156	145.5	

281 cases were processed; 124 cases were missing.

Multiple Classification Analysis

Age by
Porotic hyperostosis
Infection
Fluorosis

Grand mean= 36.5

Variable and category	n	Unadjusted deviation	Eta	Adjusted for independents	Beta
Porotic hyperostosis					
0: Absent	138	1.20		.99	
1: Present	3	-16.50		-15.68	
2: Healed	16	-7.28		-5.64	
			.29		.25
Infection					
0: Absent	122	-.60		-.54	
1: Local perios.	12	5.79		5.57	
2: Healed local	8	-3.06		-3.06	
3: Widespread periostitis	2	-11.50		-8.83	
4: Healed widespread.	8	5.38		5.78	
5: Osteomyelitis	4	4.75		1.08	
6: Healed Osteo.	1	-11.50		-8.83	
			.23		.21
Fluorosis					
0: Absent	115	-3.91		-3.66	
1: Slight	31	9.79		9.01	
2: Moderate	9	12.67		12.28	
3: Severe	2	16.00		15.55	
			.54		.51
Multiple R squared					**.394**
Multiple R					**.628**

Three conditions: cribra orbitalia, postcranial porosity and endocranial lesions explained 52.6% of the total observed variance in subadult age at death, although only cribra orbitalia is statistically significant (Table 9.3). Sample sizes are markedly reduced which means that the results need to be treated with some caution. Calculation of the average age affects, however, match the predictions of the differential diagnosis. The absence of cribra orbitalia had an average age effect upon the youngest age group (0–0.5 years) mirroring the fact that cribra lesions did not develop until after six months of age in this population. The distribution of active cribra lesions did not vary significantly from the population as a whole (c 3.2 years). This reflects both the age at which cribra orbitalia occurred, and the overall high frequency of the lesion amongst subadults. Healed cribra orbitalia, on the other hand, affected a substantially older age group amongst subadults (c11 years). Postcranial porosity and endocranial lesions both affected a younger than average age group (c 1–2 years).

Amongst adults only, porotic hyperostosis, infection, and fluorosis explained 39.4% of the variance in mortality with signficant age effects caused by hyperostotic and fluorotic lesions (Table 9.4). The percentage of variance explained is much lower than for either subadults only or all ages combined. This reflects the greater variability in the cause of death amongst adults. For all adults combined the average age of death was 36.5 years. Each condition, particularly porotic hyperostosis and fluorosis, affected a different age group. Active porotic hyperostosis had its greatest impact on a significantly younger age group, c 20 years, while healed porotic hyperostosis also affected a slightly younger age group, c 30 years. This is indicative of the long-term effects of porotic hyperostosis which are not restricted to subadults. Infectious lesions affected the population in a similar pattern to adult mortality as a whole. There are, however, some variations: active periosteal lesions tended to affect a slightly older age group (possibly reflecting the non-serious nature of these infections) but healed lesions were observed most among a younger age group. The reason for this is not clear. The data for osteomyelitic lesions are based on small sample numbers and need to be treated with caution. Fluorosis, as per earlier analyses, had the greatest affect on adult mortality (Beta=0.51) and the chronic nature of the condition is emphasised by the increasing age with increasing severity of lesion.

In summary, porotic hyperostosis and related lesions had their most significant affect upon younger age groups especially subadults. Their effect, however, was not restricted to subadult ages only: amongst adults those with active, or even healed, porotic hyperostosis had a younger than average age at death. Infection tends to follow the pattern of a chronic disease: localized and widespread periostis both affected, particularly if healed, a slightly older than average age group. Osteomyelitic infection, especially

when the whole population is considered was a significant cause of death amongst younger adults. In contrast, fluorosis had only a signifcant relationship with mortality above 40 years of age. The analysis of cause at death supports the notion of three syndromes: porotic hyperostosis (possibly reflecting, amongst other anaemias, thalassaemia); infection (tuberculosis with a significant age impact and syphilis causing morbidity without associated mortality), and fluorosis affecting the oldest members of the society.

The combination of porotic hyperostosis and infection amongst young children can be identified as the interaction of infection with both iron deficiency and possible thalassaemia, particularly amongst those over six months of age. Amongst adults, the infectious lesions associated with trauma and possible syphilis, plus fluorosis, are most closely linked to the pattern of mortality. These relationships between the observed lesions and age at death need not be a causal relationship, however, but a reflection that these conditions are chronic, age-accumulative, and, in the case of fluorosis, frequent. Therefore their age distribution is similar to the population as a whole while diseases such as possible tuberculosis, while a significant cause of mortality, is only observed on a small percentage of the skeletons.

Correlations between pathological conditions

The interrelationship between these pathological conditions isolated as underlying or associated causes of death (porotic hyperostosis, periostitis and osteomyelitis) and other lesions can be identified by analysis of correlation (Table 9.5). Porotic hyperostosis, for instance, does not operate independently of other skeletal lesions. The common etiology of cribra orbitalia and cranial lesions has already been discussed (Chapter 8). Analysis of correlation also demonstrates a significant statistical relationship between cranial and postcranial lesions. Similarly endocranial lesions are related to porotic hyperostosis in the correlation matrix (Table 9.5). Apart from these associations which are expected given the common cause, porotic hyperostosis is significantly related to localized periostitis (p=.018).

This relationship reflects two factors. Localized lesions are most commonly found amongst young children during the same ages as porotic lesions of the skeleton. Secondly, it is indicative of the synergistic relationship between infection and iron deficiency anaemia where infection may precipitate anaemia in individuals with marginal iron levels (Scrimshaw et al. 1967). The reverse condition also holds: children with anaemic conditions may be more susceptible to certain infections. Obviously after six months of age and the depletion of foetal iron stores many infants are susceptible to this train of events.

It is noteworthy that neither widespread periostitis nor osteomyelitic lesions are correlated with porotic hyperostosis. This is primarily because, while these conditions do affect younger individuals, it takes time to develop chronic signs of the disease. In addition, in both treponematosis and tuberculosis, evidence amongst children tends to be more localized, therefore less distinguishable from non-specific lesions. Therefore amongst young children these conditions may well interact with iron deficiency anaemia. Amongst adults, however, considering the low percentage of individuals with skeletal lesions, and the healing of porotic hyperostosis, no relationship between the two conditions is visible.

In both samples however widespread periostitis is correlated with fluorosis. This is explicable given the lack of mortality associated with bejel (endemic syphilis) and the long time over which fluorosis develops. Thus the frequency of both widespread periostitis and fluorosis is greatest amongst the same group–50 years and older.

Table 9.5
Correlations between different pathological conditions

	PH	PCPH	Endo.	LP	WP	Osteom.	Trauma	Fluor.	LEH
Porotic hyperostosis	–	**.44**	.15**	.18*	-.02	-.01	-.01	.03	-.16**
Postcranial porosity		–	**.25**	**.34**	.01	.02	-.02	-.04	-.13*
Endocranial lesions			–	.15*	-.00	.05	-.02	-.03	-.06
Localised periostitis				–	-.10	-.07	-.05	-.08	-.11
Widespread periostitis					–	-.06	.03	**.36**	.05
Osteomyelitis						–	.03	.01	.14**
Trauma							–	-.08	.06
Fluorosis								–	.13**
Hypoplasia									–

(.44: p<.001; **: p<.010; *: p<.050)

Linear enamel hypoplasia is significantly related to porotic hyperostosis and postcranial porosity in both samples. However the correlation is negative; hypoplasia is least common amongst those with porotic hyperostosis. Again this is not a simple relationship. Firstly deciduous hypoplasia, reflecting intrauterine conditions, was extremely uncommon in this population in contrast to hypoplasia of the permanent dentition, so that individuals of the youngest ages are scored absent for the presence of LEH. This means that children dying with porotic hyperostosis were less likely than the entire population on average, to have hypoplasic defects. Secondly hypoplasic lines represent stress and recovery, not just stress. The mortality associated with active porotic hyperostosis may be reflected in a negative rather than positive correlation with hypoplasia.

Hypoplasia is extremely common in the two samples; it is not expected therefore that there would be clear relationship between it and each pathological condition. Interestingly, however, there are significant relationships in both samples between hypoplasia and both osteomyelitis and fluorosis. This does not reflect age effects since, apart from children, osteomyelitis was most frequently observed in young adults, a different age group to fluorosis sufferers. It appears to demonstrate a more significant relationship between childhood development and these two conditions.

Tuberculosis most seriously affects young children, particularly between 1–6 years, the period of permanent tooth formation. The significant correlation observed between hypoplasia and possible tubercular lesions, therefore, appears to reflect, in part, episodes of attack and recovery in the course of this disease. (This of course, is not the only cause of LEH but one of many.) The analysis also suggests that tuberculosis and porotic hyperostosis may not be clearly related: porotic hyperostosis had a negative relationship with LEH either because it affected a younger age group (not entirely true) or because of associated mortality. Tuberculosis, on the other hand, was probably contracted slightly later in childhood, (during the same period as peak enamel formation) affected a smaller percentage of the population, and had a bimodal mortality peak with the strongest skeletal evidence for the second peak.

The possible relationship between fluorosis and hypoplasia was discussed in Chapter 7. The significant correlation found between skeletal fluorosis and hypoplasia appears to support the existence of such a relationship. The alternative explanation is far more difficult to support: that people with LEH had a significantly better life expectancy than those without. While this may hold amongst very young children, it is less likely, as demonstrated by the link between LEH and tuberculosis, to occur amongst adults. The more parsimonious explanation is that fluorosis and disruption of enamel formation occurs synergistically.

General discussion

In these two populations there were a complex of pathological conditions acting in varying combinations upon different age groups.

The reason for high neonatal mortality is unexplained by the observed pathological lesions. The only evidence is negative: while stillbirths, premature and low birthweight births do occur, these do not occur in frequencies suggestive of nutritional disturbance. High mortality of women in reproductive years does, however, suggest that perinatal deaths, including those due to complications of childbirth, did occur.

From six months to three years there is evidence of repeated episodes of stress, either infectious or nutritional, amongst all individuals. Deaths during this period are clearly reflected in the frequency of periostitis and porotic hyperostosis. The latter became of increasing importance in the 3–6 age period. During this period, also, tuberculosis probably had an impact upon childhood mortality. The lingering effects of porotic hyperostosis are observed during the 6–10 year period, at the same time mortality significantly declined.

Osteomyelitic lesions, possibly caused by tuberculosis, are signficantly associated with mortality in the young adult years. Yet it must be remembered that only about 26% of the variance in mortality could be explained by these skeletal lesions. In comparison with other populations (eg. those used for the Weiss Life Tables; Weiss 1973) the Bahrainis, particularly females, experienced higher than expected levels of mortality during young adulthood. Tuberculosis goes some way to explaining this but is obviously not the only cause. Unassociated with deaths in this period are traumatic lesions, widespread periostitis, and fluorosis.

The latter two conditions, however, are associated with deaths during the oldest age groups. Based on differential diagnosis these are probably associated causes of death rather than direct causes.

This pattern of mortality and morbidity is a reflection of living conditions within the society. The causes of death are indicative of the environment, but at the same time, ill-health, especially when resulting in death, places stresses upon a community. This stress is experienced not just in terms of individual loss of labour and increased food demands; while these problems are experienced at a household level, their impact is felt by the community as a whole. The impact is not merely economic and demographic but psychological and social.

Analysis of mortality and morbidity via survivorhsip identifies at what age these stresses occur and their nature. From this further analysis the population composition and its socioeconomic features may be reconstructed.

Chapter 10

Step 5: Growing up on Bahrain Island

Analysis of survivorship is particularly useful with stress indicators as a means of assessing the relative impact of pathology. Unlike cause of death analysis, survivorship does not offer absolute answers but a comparative perspective of groups within the population. This viewpoint is important because it indicates the impact of disease upon the living; the analysis is therefore complementary to cause of death. The two analyses partly overlap since the causes of mortality (eg specific diseases) affect deaths past the age of their occurrence. The link with the living, however, is often less explicitly stated. Therefore, in this instance, survivorship is not used to measure the impact of disease (as per Goodman and Armelagos 1988) but for a cross-sectional view of the society at various ages. The intimate link between morbidity, mortality and social structure is thereby reconstructed.

To examine the impact of disease upon a society it is necessary to remember the changing balances between the conditions over an individual's life span. For example in early childhood porotic hyperostosis and localized periostitis are important, but by the early twenties a disease contracted during this same early age period, tuberculosis, is more significant in terms of adult mortality. Given that only some 20–30% of mortality is directly explained by skeletal pathology, however, it is necessary to look at the effect of disease by examining the age interrelationships of both chronic and acute markers of stress while keeping in mind the presence of conditions which do not leave traces upon the skeleton.

Method

Assuming a single birth cohort of 100 people (50 males and 50 females) makes it possible to determine disease impact from skeletal remains by survivorship. This procedure is based on the same assumptions that underlie palaeodemographic analysis: that the sample is cross-sectional, and that it is representative of a single stable population thereby encompassing for each successive age group those who died and those who survived beyond this age. This is only a model based upon the life table notion of a single cohort or generation passing through life. It is not a cross-section viewpoint of the village population at a single point in time.

The analysis concentrates upon the DS3 sample, merely pointing out where the Saar sample diverges from this pattern. The caution in using Saar is due to the small size of the sample and the underrepresentation of infants in this group.

The experience of each age class

0–1 years

With a cohort of 100, given a probability of dying of 0.385 during the first year of life there were c38 infants who died, 62 surviving. Of these 38 children, approximately 11 died between 0 and 3 months of age. While there were no signs of skeletal pathology on any infants dying at this early age, approximately one-third of these children were possibly low birth weight or premature infants. Growth amongst the remainder appears to have been normal.

Low birth weight and prematurity are important underlying causes of infant mortality in the neonatal period (Puffer and Serrano 1973). This suggests, therefore, that the prevalence of low birth weight and/or premature infants amongst the dead is greater than in the entire population, although an estimate of the incidence at birth is not possible.

Causes of low birth weight and prematurity are numerous. Infection during gestation, maternal dietary deficiency, age of mother are some of the possible causal factors. These factors, of course, do not operate independently of each other; eg. anaemia during pregnancy can be precipitated by malarial or other parasitic attack. The link between maternal dietary deficiency and low birth weight is less apparent than the possible interaction of infection with borderline nutrition (Lechtig et al. 1982; Solimano and Vine 1980). In addition, as the demographic analysis earlier pointed out, close birth spacing, along with early childbearing was probably the norm. Infants of both young primaparas and of grande multiparous women are liable to be smaller and therefore more subject to complications accompanying premature or small for gestational age births (Puffer and Serrano 1973).

It is notable that, according to dental age estimates, growth of some of these children who died soon after birth was normal. The neonatal period is most vulnerable in a child's life. Apart from complications during birth, the child is also susceptible to several conditions causing early mortality. While resistant to many infections due to maternal immunity and protected from others by breastfeeding, diseases such as neonatal tetanus and simple infections caused by droplet infection may have devastating effect. For instance, in rural India death rates from neonatal tetanus range between 16–72% of all neonatal deaths (Maru et al. 1988). Certainly the decline of deaths after the first three months indicate that conditions concerned with birth were the most stressful on the new infant. Similarly the lack of skeletal lesions suggests acute rather than chronic conditions which would be expected in populations with severe nutritional deficiency (Palkovich 1987). In the case of food shortages one could expect the presence of porotic hyperostosis amongst this young group of children.

During the second three months of life conditions ameliorated slightly. Of the remaining 89 children a further seven died. Based on the frequency of porotic hyperostosis in the total sample (14%) one or two of these had radiological signs of anaemia at death. For these children who died growth was still normal, but a small percentage already had endocranial porosity, associated in this group with porotic hyperostosis.

As discussed earlier, the early presence of anaemia in this group may be associated with thalassaemia. Most anaemias, including thalassaemia, do no manifest until after six months of age when foetal stores of iron are finally depleted. In this population, however, some at least of the mothers were frankly anaemic (three women who died between 20–30 had active cribra orbitalia at death), so that the onset of iron-deficiency or thalassaemia may have been slightly earlier than expected. Yet the number affected was small and the absence of such evidence at an earlier age suggests that nutritional problems, while probably present, were not extremely serious. The maintenance of normal growth amongst these infants supports this interpretation.

Similarly amongst the survivors only 3% experienced episodes of stress resulting in an enamel hypoplasia. The episodes could be a bout of infection or a period of dietary deficiency. This of course is an underestimate since at this early stage teeth are less susceptible to hypoplasia. During 3–6 months of age, however, conditions were obviously better than in the preceding three months.

The second half of the year, however, was the most dangerous after the neonatal period: twenty infants of the original cohort died during this time. The increased death rate is matched by an increased number with activite porotic hyperostosis: approximately half of the twenty had cranial porosity. In addition growth had slowed amongst these children suggesting that stress had become chronic amongst those dying. A small number were affected by rickets, apparently occurring in conjunction with iron deficiency. Approximately five percent also had evidence of infection upon the long bones. Amongst those who survived this age, however, only about 3% again experienced stress resulting in a hypoplasic defect.

It has been estimated earlier that about 8% of children who died between 0–6 years were thalassaemic. Thus thalassaemia would account for only a small number of the deaths in this age period, especially when it is remembered that only half the children who died had skeletal evidence of anaemia.

The increase in death rates, however, appears to be associated with the transition at six months from iron stores gathered in utero to dependence on those accumulated from the diet. At the same time inherited immunity to infectious disease, including malaria, becomes less significant, while exposure to environmental pathogens is greater. Thus children become susceptible to an increased array of diseases. Weaning is often associated with increased death rates, particularly from infectious disease and frank malnutrition (Gordon and Scrimshaw 1965; Wood 1983; Wills and Waterlow 1958), but it is often accompanied by an increase in LEH amongst the survivors in a skeletal group. In the present case, however, no increase in LEH is seen at this age. The cycle of infection and nutritional deficiency, particularly of vitamin D and iron, appears to be primarily restricted to the children who died during this age group.

It is suggested that the increase of death rates in this second six months, and the interplay of this increased mortality with growing evidence of chronic stress amongst the same group, reflects either early weaning, dietary inadequacy (possibly because of nutritionally deficient mothers or delayed supplementation) or increasing infectious insults. Probably to some extent all three scenarios occurred. Most likely is that a certain sector of the population, possibly including those with nutritionally deficient mothers, became increasingly vulnerable during the second six months of life to infection and dietary deficiency, one prompting the other. Infection again appears to be a major factor since the pattern of rickets seen amongst these children is similar to that amongst well-nourished children (Salimpour 1975) rather than the serious wasting version seen amongst malnourished groups.

Only some 20% of the original cohort are thus affected. The low percentage of LEH amongst survivors, whilst possibly reflecting differential tooth susceptibility (Skinner and Goodman 1992), does suggest that survivors were less affected, potentially because they were inherently less frail or within a less risky environment.

Therefore between 0 and 1 years children were probably faced with a series of threats. At birth 5–30%(absolute limits) were either low birth weight or premature births, with a greater chance of dying. The remainder were apparently normal but were at risk from endogenous (eg. congenital anomalies) and exogenous risks (eg. neonatal tetanus and other infectious diseases). The period between 3–6 months of age was less stressful for the whole population though a small proportion were obviously at greater risk since they died with active porotic hyperostosis, possibly reflecting nutritionally deficient mothers. This number however is small. In the second six months, however, a pattern is established of synergistic infection and nutritional deficiency. At this stage the effect seems to have been greater amongst non-survivors. The survivors do not show overt signs of stress originating in this age group. Infectious disease, including malaria and gastrointestinal disorders, was possibly one of the main causes of mortality in this group. Tuberculosis can have a significant impact on this young age group (Daniel 1981) but the major impact was probably slightly later. Bejel is not a common cause of death in this age group. Thalassaemia also appears to have begun to extract its toll amongst the small percentage of homozygotes in the population. These conditions combined together create a serious risk for infants, although the stress appears to be

most serious upon an at-risk segment of the population, not the entire group.

1–5 years

While mortality declined after the first year of life, skeletal evidence for morbidity is greater. Of the 62 survivors who entered the second year, 16 had died by 5 years of age. The majority of deaths occurred in the 1 –2 year age group: five in the first six months; four in the second. Growth during the first six months was normal but between 18 months and two years growth had slowed amongst the children who died. Amongst these 1–2 year olds, three quarters had active porotic hyperostosis at the time of death and 4% had some form of periostitis. Again this includes the 8% who were possible thalassaemics. Mortality, however, was much higher than could be accounted for by this disease alone. At the same time, one of the possible cases of childhood tuberculosis belongs to this age group, as does one possible case of bejel. This suggests that during the second year of life, as weaning gradually progressed, increasing infectious episodes with concurrent nutritional stress were seen in the population. The increase in the prevalence of porotic hyperostosis with a decline in mortality suggests a transition from acute episodes with high mortality to chronic stress with high morbidity.

Evidence amongst the survivors supports this interpretation. Between 1.5–2.5 years a growing number of adult survivors experienced their first stressful episode, possibly concurrent with increased reliance on weaning foods. Older subadults who later died, have an earlier mode of onset of LEH, around 1.5–2 years. This possibly reflects the situation observed for the 6–12 months age period where a particular segment of the population was at-risk.

After the second year, mortality declined even further: two died between 2 and 3 years; three between 3 and 4 years; and two between 4 and 5 years. At the same time, the percentage of porotic hyperostosis amongst these was still high, and examination of growth indicates chronic stress. From 2–3 years the velocity of growth slowed, possibly in response to the continuation of stress experienced between 1–2 years of age. After three years there was a slight improvement in growth amongst those who died but it was still slow. Similarly after three years of age there is little evidence of infection in the group and presumed thalassaemia deaths form a greater percentage of the children who died.

Amongst survivors, 2–3.5 years also appears to have been a period of continued episodes of growth disruptions. Between 33–45% of these individuals had at least one hypoplasic line formed during this period. After 3.5 years the frequency declines slowly. By six years of age most of the survivors had experienced on average 2.5 stressful episodes, the greatest number and shortest intervals occurring between 2–3 years. Children dying in later childhood had even more, indicating that enamel hypoplasia in this instance reflects conditions associated with increased mortality.

After six years of age, healed cases of cribra orbitalia and porotic hyperostosis were most common suggesting that the condition occurred during early childhood. This corresponds with the hypothesized development of porotic lesions in young children only (Stuart-Macadam 1985). Assuming that cribra orbitalia and associated lesions occurred during this 0–6 year age period, 15% of adult survivors and 70% of those who died between 1–6 years of age had sufficient anaemia during this time to induce skeletal change. The disparity is evidence of the importance of porotic hyperostosis in reflecting mortality conditions.

Overall amongst the age cohort (1–6 years):

1. 16 died:
   5 without porotic hyperostosis
   11 with porotic hyperostosis
   (1–2 thalassaemic; 9 normal/hetzyg)
2. 46 survived:
   7 with porotic hyperostosis
   39 with no signs
   (overestimated if complete healing occurs).

Amongst the whole cohort during 1–6 years this indicates a minimum prevalence of anaemia (not including thalassaemia homozygotes) of about 25% (16/62). This can only be a minimum considering that not all individuals with iron-deficiency or malarial anaemia develop skeletal signs. On the other hand, the degree of underestimation is partly offset since some of these cases may have been thalassaemia heterozygotes.

As emphasized earlier, anaemia may be due to nutrition or infection. In the latter case it may even be viewed as a protective mechanism: the hypoferrimic response to infection (Stuart-Macadam 1991a). Since many infectious organisms require iron for growth, one of the body's immune responses may be to sequester iron prompting iron-deficiency anaemia and thereby starving the organism. The associated mortality risks of porotic hyperostosis, however, indicate that this should not be accepted as the entire explanation of anaemia in the DS3 population, nor should it be seen in this group as a marker of good health.

The evidence from enamel hypoplasia implies that there were three groups in the 1–6 year age group:

1. Children too sick, with no recovery and early mortality (including thalassaemics) and with a high proportion (c75%) of porotic hyperostosis = c25% of the cohort
2. Children weakened by more frequent and earlier episodes of acute stress and recovery, accompanied by a slightly higher frequency of porotic hyperostosis, and who died during later childhood = c8%.
3. Children with repeated periods of stress and recovery especially during earlier years but who survive with a

lower frequency of porotic hyperostosis (at least 15% of this group) reflecting an anaemic condition = 67%.

The worst age period appears to have been 1–2 years but disease tended to become more widespread during this time with increasing episodes of stress and recovery occurring until c3–3.5 years. Elevated levels of porotic hyperostosis also occur during this second year of life.

It was argued that, during the first year, porotic hyperostosis is indicative of infection induced iron-deficiency. During the period from 1–6 years, however, it is difficult to determine whether this was the case or not. Prolonged breast feeding and weaning are both associated with nutritional stress, particularly anaemia, but at the same time the child is introduced to more environmental pathogens (Wood 1983). The evidence from infectious lesions suggests that tuberculosis and treponarid were both present among the population, and these two diseases, along with malaria and other parasitic diseases, accord well with the distribution of mortality and morbidity observed in this age group.

Tuberculosis was most serious and affected 1–5 year olds with associated high mortality. The skeletal evidence suggests about half of the population had contact with tuberculosis at some period of time. Not all these cases would have occurred in early childhood though the majority may have. Levels of mortality from tuberculosis can range as high as 25% of all cases (Daniel 1981).

Endemic treponematosis, on the other hand, also occurs during early childhood but with much less associated mortality. As children begin playing together the transmission of the spirochaete occurs, causing widespread infection. Acquisition of the disease causes initial infectious mucous patches in the mouth with a generalized non-irritating rash. Later signs of bone destruction include painful osteoperiostitis of the tibia and fibula (Csonka and Pace 1985) but associated mortality is low. The prevalence of skeletal lesions in the population is c 2% implying that about 30% of the population had at some stage had the disease. Two cases indicate that children were affected during the 1–6 year age period but again the absolute frequency is questionable.

Malaria can be a major cause of both mortality and morbidity during the 1–4 year period. As explained earlier, six months of age marks the beginning of malarial attacks, which tend to become increasingly frequent after about one year of age until the development of natural immunity at around 4 years. In hyperendemic areas, such as oasis areas in the Gulf prior to eradication, parasite rates could be as high as 100% at one year of age (Daggy 1959). This rate declines with age. It should also be noted that malaria is one disease clearly associated with anaemia though attack rates tend to be lower in malnourished children (Eddington 1967).

These, however, are only the conditions that can be identified on the skeletal remains. Gastrointestinal disturbances, always a significant cause of mortality and morbidity amongst young children, leave no signs upon the skeleton except non-specific indications such as LEH (evidence of episodic stress and recovery) or porotic hyperostosis when the infection prompts the development of anaemia.

The high frequency of porotic hyperostosis and LEH, plus high mortality during the 1–6 year period, demonstrate that for young children this was a stressful environment. While fewer children died after surviving the first year, the interaction of stressors, nutritional and infectious, created widespread morbidity. As children grew older their natural immunity developed along with their chances of survival, although at this stage deaths from thalassaemia became a possibility.

While the levels of porotic hyperostosis are high in comparison to other skeletal groups, the percentage with infectious skeletal lesions is low. Elevated levels of mortality suggest a situation where vulnerable children died quickly leaving a healthier portion who developed chronic stress symptoms but recovered.

This suggests that infection rather than nutritional disturbance may be responsible for the presence of porotic hyperostosis excess to thalassaemia. In the case of severe nutritional disturbance it may be expected that recovery would not be so complete and that even amongst survivors active porotic hyperostosis would still be seen. In addition, if weaning is hypothesized to have occurred between 1–2 years (most probably 1.5–2 years), then in an area with undernutrition there should be a concurrent increase in mortality, but this does not occur. The worst mortality has already occurred. Therefore the argument is that although this village was situated in an environment containing a range of dangerous pathogens, it was not an environment where the population was chronically undernourished. Comparison with the pattern of mortality in modern populations confirms this (Puffer and Serrano 1973).

Saar village should be mentioned here. Amongst individuals from this village not only was mortality lower, but porotic hyperostosis was less frequent. This may of course, reflect a lower frequency of thalassaemia genes; but, combined with the same prevalence of LEH as at DS3 (though with a slightly later onset), it suggests that children from Saar were better buffered from disease during this period. They still experienced repeated episodes of stress and recovery, but these episodes are later in onset. This may reflect later weaning but probably also indicates better living conditions so that disease episodes are more closely tied to weaning than at DS3.

Even so, in both villages a high proportion of children in the 1–6 year age group suffered repeated bouts of serious illness. By three years of age they were through the worst of this, but the costs of such morbidity and mortality would be felt by the whole society.

Later childhood

As mentioned, the period after five years of age in the two populations was marked by decreased mortality and morbidity. Of 46 in the original cohort entering their sixth year, only three to four died before ten years of age. Growth of those dying during this period was maintained slightly below that of other skeletal populations. There is a possible indication of some catch-up growth during this time. Porotic hyperostosis occurred on 50% of these skeletons, including one possible case of thalassaemia, but apart from this one example, the majority of cases had healed by the time of death.

Amongst survivors, LEH declined during the 5–6 year age group which matches the general picture of low mortality during this period.

The situation is even better between 10–15 years of age: even fewer individuals died (c2.5), and growth amongst these appears to be closer to the average of other skeletal populations.

Amongst survivors, there is no means of determining from skeletal indicators what stress, if any, was faced during this age period. Given, however, the low mortality and presence of healed porotic hyperostosis amongst individuals, it suggests that surviving early childhood guaranteed good survivorship except for the small number already severely stressed. This group had slightly elevated levels of porotic hyperostosis compared to adults, even if healed, and an increased frequency of LEH.

Obviously conditions for older children had improved so much from early childhood that only these few individuals are fatally affected. Trauma may also play a larger role in these age groups but the lack of fractures in the entire sample suggests that injury was not a common feature of life in either DS3 or Saar.

15–20 years

By 15 years of age, only 39–40 remained of the original cohort, clearly indicating the risks of childhood. From 15–20 years of age deaths did increase slightly. Of our 40 survivors, it needs to now be assumed that 20 are males, 20 female. This, of course, may not be the case, especially since more females than male had porotic hyperostosis which possibly indicates a more stressful childhood for females than for males. On the other hand, the 50% female representation amongst adult skeletons suggests that any imbalance was minimal.

Of the 20 females, three died before 20 years of age. Two of the 20 males also died during this period.

Two diseases do show upon these skeletons. Amongst the females, a low percentage have evidence of active periostitis including possible treponematosis. Amongst males, 5% have probable tubercular lesions active at the time of death.

The impact of these two diseases is markedly different. While women in this cohort may have died with active treponarid, this is unlikely to have been a direct cause of death. On the other hand, the extensive and serious nature of the tubercular lesions indicates the life-threatening nature of this disease. The secondary impact of tuberculosis upon mortality occurs amongst young adults and this small percentage of individuals with active skeletal lesions probably represents only a small number of the actual deaths from tuberculosis.

Female mortality began to exceed male at this age. This may reflect the beginning of childbearing amongst young females. Maternal mortality is greatest around first birth and then again when the woman has become multiparous (5+ births, Puffer and Serrano 1973). It is probable that some of the female mortality seen here was due to the risks of childbearing, especially considering the high neonatal mortality. The absence of skeletal tubercular lesions does not of course mean that women avoided tuberculosis.

In terms of morbidity it is important to note that at this age signs of occupational stress are visible upon these skeletons. Schmorl's nodes are observed on c30% of skeletons, both male and female, while osteochondritis dessiccans begins to make an appearance. These conditions occur as the result of sudden trauma as well as heavy workloads. Their presence in this young cohort indicates that at an early age everyone was participating in the work of the village.

At DS3, this apparently included equally males and females. At Saar, however, females appear to have been less involved than males. Female mortality at Saar is also less than at DS3 for this age period. In addition there is no disparity between male and female mortality at Saar. This suggests that environmental conditions at Saar may have been better; at Saar females at this early age managed to avoid the additional risks accruing to the female sex. These risks would include the degree of risk associated with childbearing (possibly indicating a later age of marriage).

20–30 years

The possible impact of childbearing is observed more clearly in the 20–30 year age group. Of the 17 women who entered this cohort, 8 died: the highest mortality since the second year of life. Males fared better: of 19, only 5 died.

A small percentage of these women still had active porotic hyperostosis. This may indicate the persistence of iron-deficiency anaemia from childhood. Alternatively it may be thalassaemia heterozygotes who can be liable to chronic marginal anaemia (Huisman and Jonxis 1977). On the other hand, the fact that males are not similarly affected does indicate that at this age women experienced a greater degree

of stress. As always this could be either nutritional or infectious, and the likelihood is that both factors were operating.

In subtropical areas, particularly where malaria is endemic, pregnant women have increased susceptibility to parasitic attack along with increased nutritional demands for iron, which are often not completely filled (Manir and Khaleque 1969). The resulting increase in demand for iron can push marginal iron-deficiency into frank anaemia. Gastrointestinal parasites create a similar effect: not only may there be direct blood loss from organisms such as hookworm; but also part of the body's own nourishment goes towards sustaining the parasites (Memoranda 1972).

These problems increase with parity. The cycle of childbearing and lactating creates a vicious circle in which the mother is not given a chance to recover her nutritional status. This probably explains why female mortality is highest in the DS3 sample during peak reproductive years rather than the 15–20 year period when first pregnancies probably occurred. High maternal mortality is also a correlate of the high neonatal mortality seen in this population.

The relative absence of skeletal lesions, particularly infectious, may not be a reflection of better living conditions in this population compared to others where infection levels are high. The high mortality amongst females is consistent with an environment where infection causing mortality tends to act quickly. The small percentage of individuals with active periostitis possibly marks the resurgence, for some, of a treponemal infection acquired in early childhood. These infections probably caused only inconvenience amongst their sufferers at this age, though the presence of an active infection may result in lowered resistance to other diseases. As described for the Indian village of Khana:

> Frequent sickness of adult women is often responsible for more spontaneous abortions and stillbirths, and also limits conception. The classic example is a population where malaria is endemic. (Wyon and Gordon 1971:68)

Male mortality at DS3 was, in contrast, lower than expected at this age group compared to the Weiss model life tables (Weiss 1973), though still higher than the Coale and Demeney tables (Coale and Demeney 1983). Approximately 4.5% of males who died during this age period had active lesions reminiscent of tuberculosis at the time of death. This is the expected pattern of tuberculosis mortality: the active skeletal cases representing probably a larger cause of death. Whether or not males were more susceptible than females is questionable. The difference may merely be due to better male survivorship—a longer time infected allowing the spread of skeletal lesions.

At Saar during this age female mortality began to exceed male mortality although it was still markedly lower than at DS3. This concurs with the hypothesis that the repeated bearing of children rather than the initial birth is most associated with mortality. Male mortality on the other hand was also slightly less than DS3 and the absence of tubercular lesions suggests that tuberculosis was less of a problem at Saar. Certainly the consistent picture is that, for adults from Saar, living conditions were rather better than at DS3.

30–40 years

Female mortality changed little between 30 and 40 years of age, increasing only slightly. Of the nine women entering this cohort, only four survived. Male mortality similarly increased: 14 males entered the cohort, only eight survived. Obviously there was still an excess of female over male deaths.

No signs of pathology were found on any of these women with the exception of an increasing degree of arthritis. Two percent of men had tubercular lesions but the lower percentage of these lesions and the fact that there are signs of healing indicate the decreased mortality of tuberculosis with age.

At Saar, female mortality is still less than at DS3, but the difference is less apparent. The evidence of infectious lesions suggests that some infections were less prevalent at Saar than at DS3. The decreasing difference between the two mortality distributions at this stage may demonstrate that, as infections such as tuberculosis and treponematosis become less prevalent with age, the difference from DS3 diminished. Saar male mortality is only slightly less than at DS3 and the difference between female and male mortality also declined at this site.

Obviously female mortality was still a major problem, but at the same time skeletal evidence for severe conditions declined and the majority of lesions seen were healed. Amongst women, those who survived to this age are even more susceptible to the maternal depletion syndrome. The declining difference between Saar and DS3 suggests that living conditions and possibly nutrition in these two places are the same but that conditions promoting the spread and virulence of infections, such as tuberculosis, were not as prevalent at Saar.

40–50 years

This age period marks the beginning of obvious and serious degenerative problems amongst the cohort. Female mortality still exceeded male mortality (1.5 of 4 women die, compared to 3.5 of 8 men) but the difference has diminished greatly. During this age the strains of childbearing would ease upon women so that mortality associated with childbearing could be expected to decline.

Males, however, increasingly faced problems from skeletal fluorosis. While only 2.6% of all adults have moderate-severe fluorosis, 40% of those who died in this age group have obvious fluorotic changes. This would result in an increasing degree of incapacitation though less than 2% of

those who died had experienced complete joint ankylosis. Even so there is little obvious impact on mortality: male mortality between 40–50 years is near identical to that between 30–40 years.

At Saar, female mortality was less than male mortality at this age, probably reflecting better female survivorship as childbearing finally finishes. Male mortality exceeded that of DS3, which is intriguing considering the lack of other differences. The disparity however is only small, and may reflect greater risks faced by DS3 males in earlier decades which could have acted as selective forces prior to this age cohort.

The similarity between Saar and DS3 is striking but it is obvious that Saar females always had a better chance of survival than DS3 females. Considering that this ties in with decreased levels of infectious and skeletal lesions at Saar, it should be considered that the environment at the more northern village was more favourable. Obviously this has economic implications. In addition, the increasing degree of joint and bone destruction, especially amongst males, from arthritis and fluorosis must have had an effect on workloads. Again the economic implications of this need to be considered.

50+ years

The final cohort is of indefinite length. Of our original 100 births only some 2.5 women and 4.5 men entered this age group. Males have extensive lesions due to fluorosis (20% have severe changes including joint ankylosis). As described earlier these lesions, while not causing complete paraplegia, may have limited their sufferers to lighter work. Arthritis, too, would have had a slowing effect on this segment of the population.

Differences between Saar and DS3 are minimal, though at Saar probably more adults, especially females, survived to enter this cohort. Fluorotic and arthritic changes are also prevalent throughout the population though male/female differences are more obvious.

Overall there are no active skeletal lesions at this age. All those individuals who experienced skeletal infection earlier in life have healed by this time, indicating that despite the prevalence of degenerative changes, life was probably reasonably secure by this age.

Conclusion

This discussion has presented an age by age analysis of morbidity and mortality at DS3 village, and at Saar by comparison. The major points to appear are:

1. The vulnerability of the neonatal period and prevalence of low birth weight or premature babies;
2. The early onset of infection and associated high mortality at six months, probably related to infectious diseases;
3. High morbidity experienced by children until c3 years of age, inlcuding the possible early effects of tuberculosis and thalassaemia on a segment of the population;
4. Better than average survivorship once this period is passed;
5. Female vulnerability during childbearing years, possibly related to infections such as malaria, and maternal depletion syndrome, and more serious at DS3 than at Saar;
6. The higher prevalence of infection at DS3, especially tuberculosis, amongst young adults;
7. With the survival of adults into middle age there was an improvement of conditions as infection became less of a problem; and
8. The major impact of degeneration and fluorosis had on the oldest age groups.

It demonstrates that three age periods would have been particularly difficult in terms of associated mortality and morbidity: early childhood (especially at DS3); early adulthood for males (at DS3); and early-middle adulthood for females at both sites, but especially DS3. Old age would have been less difficult in societal terms since these individuals represent a much lower proportion of the entire population.

To look at the social implications of the above analysis, we need to turn back to the composition of the living population as calculated in the analysis of palaeodemography. Using this, it may be possible, with data from modern traditional agricultural groups, to look and the social and economic organisation of the two villages.

The organization of village life

The composition of the living population from both DS3 and Saar was described in Chapter 6. As pointed out, this population carried a heavy load of dependents. Pathological lesions also indicate a high level of morbidity amongst the very young, and possibly among young adults.

At DS3 approximately 18% of the population would have been less than five years of age. A further 4–6% were over 50 years of age. This later group comprised mainly men, the majority of whom may have been unable to work at the peak of their physical capacity, although there is no evidence for total incapacitation.

In the majority of agricultural societies the household represents the primary unit of production (Strickland 1990). Household composition, however, may change over time in response to demographic and economic circumstances. At the same time during cycles of agriculture co-operative

networks may need to be formed, for example during harvest, for fishing, and for maintaining irrigation.

In a situation where adult mortality, especially amongst females, is high and where fertility has to remain high in order to maintain the population, the work contribution of children would be of considerable importance. The productivity of children in agricultural societies, particularly those with high fertility, has been the focus of numerous demographic studies. In rural Bangladesh, it has been calculated that male children are net producers by 12 years of age, by 15 years they have begun repaying accumulated consumption, and by 22 years have also covered for one sister (Payne 1985). In Egypt, children begin contributing to productivity by five years of age. Early tasks include shepherding, and minding younger siblings. With age these responsibilities increase (Mueller 1976).

Payne has modelled for an average Bangladesh family the ratio of labour to productivity during a household cycle (Payne 1985). The same procedure can be followed for DS3 (Plate 9). In the case of DS3 several unknowns need to be introduced. Firstly it is assumed that the children of lowest and highest parity will be most susceptible to mortality. The reconstruction is also based on a nuclear household without remarriage.

The artificiality of this is obvious, particularly since it is based on an 'average' household in a village that may only have contained 15 such units. It does, however, highlight periods of risk within the household cycle. These occur during early childhood, especially when the second child is still too young to assist with the third and later siblings.

The situation is even more difficult when it is considered that during this same period morbidity in the household is at its highest level. Apart from the presence of young children suffering from repeated infectious episodes, the mortality of young women is near its highest. On top of that up to 50% of the population may have had contact with tuberculosis, which on a household basis would be most serious around this period. All this occurs when household demands are highest and productivity at its lowest.

As pointed out in Chapter 7, in this situation it is advantageous that mortality is greatest in the first year of life before accumulated investment in rearing a child has outweighed the potential benefit of their labour. The pattern has been seen in numerous countries where a decrease in mortality is accompanied by an increase in morbidity amongst survivors (Gruenberg 1977). In DS3 an alleviation in mortality without a corresponding decline in fertility could easily have had disastrous consequences. Cultivatable land in Bahrain is a limited resource due to the constrained water supply so expansion would not easily be accommodated. With no real scope for extending agriculture and a quickly growing population, the village system could easily slip into a cycle of food shortage and increased infection where population growth was regulated by a cycle of crises.

It is, of course, possible that this did occur during the period under study. Certainly later in Bahrain's history and the history of the surrounding oases, settlements were highly susceptible to disease epidemics (Daggy 1959; Lorrimer 1908; Philby 1986). The continuity of cemetery use, however, argues against this specifically happening during the Tylos period. During the period analysed here it appears that, instead, a delicate balance was maintained. In addition there was always on Bahrain one escape from acute agricultural shortages: an increased reliance upon fishing.

So far this discussion has addressed only the economic implications of mortality and morbidity for DS3 village. The impact of these two forces must also have been felt socially. It has been suggested in the analysis of demography that demographically the best chance for this society, particularly considering the number of orphaned children, would be to consist of extended households or communal units larger than the nuclear household. Given that during a household cycle there may have been particular periods of risk and that some cohorts of the population were more at risk than others, on a village basis there was probably a degree of economic inequality. In periods of smaller harvests some households would be better able to survive. For instance it can be demonstrated that a household where both parents survived was likely to be more productive than one with only one parent. Such inequalities tend to breed greater inequalities as mortality and morbidity impact more upon one segment of society as opposed to another. Nevertheless most of the pathogens appear to be the result of external environment so that, in any case, the whole village experienced a high level of risk. This would tend to have a levelling effect and may explain the small social distinctions evinced in burial practices: 20% single burials, 80% multiple.

The one social implication that has already been alluded to is the need to maintain outside links. Given high female mortality, shortage of females may have been a real problem. There was probably a very real need, apart from strengthening social and economic bonds, to bring brides into the community. External ties would also assist in tiding over periods of real shortage, when male adults might well have needed to work outside the village. Trade and fishing (for both fish and pearls) are two areas of work on the island that could have absorbed extra labour.

It must be pointed out that the demographic profile of the village shows no real imbalance attributable to labour emigration. In addition, labour demands of the village itself were high which would mean that, like many agricultural societies in that area today (Wyon and Gordon 1971), emigration may only have been short-term. The basic similarity of burial practices on the island reflects in any case, the maintenance of cultural links.

Finally, and more speculatively, there is the effect of workload on society. Activity may be divided into two spheres: economic and discretionary (Pollitt and Amante 1984). Discretionary activities includes the effort put into

cultural and social activities. Work amongst malnourished children has demonstrated that in periods of shortage children will expend less time upon playing, a discretionary activity. This has been interpreted as an adaptation to low energy supplies (Spurr 1984). The same phenomena has been observed amongst Sudanese agricultural workers with schistosomiasis (Collins et al. 1984), and hypothesized more generally for numerous societies suffering nutritional shortages (see Pollitt and Amante 1984).

Given the demands of physical work in DS3 village, the same effect may have occurred. In addition it may be that the burial of multiple individuals in a single grave is not merely a reflection of purely economic status but also of 'energy' status (compare with Tainter 1978). In this society the labour involved in building a grave would be even more of a scarce commodity than the building material involved. No doubt the link may not have been perceived by individuals building the grave, but this does not the negate the fact that in DS3 the opportunity for surplus was only small, and that the amount of surplus was closely tied to factors of demography and health.

The difference between DS3 and Saar becomes even more important when the extent of the implications of mortality and morbidity are realised. It has been pointed out throughout that, even with allowances made for the relative completeness of the samples, living conditions at Saar were probably better than at DS3. For instance, the diagram of labour versus productivity at Saar looks rather better than at DS3 (Plate 10). The periods of risk are less acute, primarily because of better female mortality and possibly better child survival. The difference is not within relative energy demands since at Saar they are higher but in the supply of labour and the decreased morbidity. There is still uncertainty about the early childhood period since it may be suspected that the trade off for less mortality is greater morbidity at Saar.

Obviously many of the environmental pathogens in both villages were similar. Infection, however, generally appears less at Saar suggesting that the village may have been less crowded. Nutritionally as well, the village may have been better off. Sources of spring water at Saar are greater than at DS3 allowing more extensive agriculture. In addition the greater supply of labour appears to have allowed clearer sexual division of occupations and possibly more male activity in work other than pure agriculture. This may explain some the differences in patterns of arthritis between the two groups. More participation in non-agricultural occupations, especially fishing, would also improve diet composition and availability. The greater economic surplus at Saar that would result may be reflected in the differences seen in burial practices: only 50% multiple burials and fewer per grave. The perceived differences in burial practices combined with evidence of disease and death all support the hypothesis that northern villages were better off, economically, environmentally and socially, than those further to the south. Larger village size too would be more of a buffer against shortage since there tend to be better economic opportunities with a larger population size.

On Bahrain not only were there intra-village divisions but across the island conditions changed. This could well begin to explain the differences perceived by Salles and initially interpreted as different ethnic or religious groups (Salles 1984). Overall however it must be emphasised that there is the underlying similarity of remains and the obvious continuation of conditions from one period to the next.

The vulnerability of these groups, especially those in more marginal areas, also explains the close link seen between settlement and water levels. This could be seen as support for Larsen's model of water and land use, but, while it is an explanation for the mechanism of fluctuating settlement, it also introduces several other factors into the equation which negate the idea of a mechanistic relationship between water levels and settlement. Firstly, as water levels drop the range of responses possible for a village is limited in part by their organisation, including factors such as fertility and mortality. These parameters determine not only how many mouths there will be to feed from one generation to the next but also how many people are available to feed them. As can be seen from the above data, even within one village some groups will weather these changes better than others. In addition, as the comparison between DS3 and Saar demonstrates, significant differences can exist between villages: the population at Saar was more buffered against crises. Demographically these villages had a potential to be extremely unstable. The high rates of mortality and fertility mean that gross environmental change would not be necessary to provoke a major catastrophe–even slight improvement in mortality or decline in fertility could lead within a short space of time to a burgeoning or rapidly contracting group of people. The upshot of this is, that unlike previous views of Bahrain's history which have seen it as a cohesive unit, the reality is probably a very spotty history indeed: regional continuity may well have been based upon a pattern of local extinction and growth which has only a loose connection with external factors.

Conclusion

The population structure observed for these two Bahrain populations may be seen as typical for intensive agriculturalists in malarial areas. This is evidenced by its similarity to the Bronze Age population from Lerna, Greece (Angel 1971), and to modern agricultural groups in a similar ecological zone (eg. Keneba and Manduar; McGregor and Billewicz 1981). Given a different economic base or variation in agricultural practices, the population structure could be very different, not only because the causes of death would alter but also because the decisions made by individuals regarding fertility, migration, and access to resources would also change.

Throughout the Arabian Gulf there were, historically, different populations co-existing: at times co-operatively, probably at times actively competing. These groups included trading enclaves, military bases, pastoralists and fishermen, along with agriculturalists. Each of these experienced different living conditions and different opportunities for change. In order to fully interpret the history of this area, the living conditions and biosocial constraints that operate for these groups need to be fully explored.

On Bahrain itself there is a unique opportunity to begin to answer some of the questions arising from this thesis: the nature of history at a village as opposed to regional level, the relationship between subsistence and society, the tie between population structure and social stratification, and the link between population density and access to resources. What the current analysis has demonstrated is that the Island's population cannot be treated in archaeological and historical reconstructions as an unchanging, invariable entity upon which external or environmental factors operate at will. Individual communities operated within their own constraints and the interests of one group were not necessarily the interest of all. While long-term continuity is evident within the Island's history this is probably based upon localized disruption. The emphasis of research on the Island needs to take account of internal forces and conditions rather than adopting mechanistic interpretations of population or completely ignoring local conditions.

Chapter 11

Conclusion

This work was conceived with the aim of developing a model of analysis which allows for the interpretation of the results of skeletal analyses in terms of a living population. The analysis of the DS3 and Saar populations from Bahrain has demonstrated that this can be achieved while at the same time acknowledging and accommodating the limitations of the data. The results are explicable and accessible to not only physical anthropologists but archaeologists, social anthropologists and demographers. At the same time the ability to compare these results with modern populations gives another dimension to reconstructions of the past.

In the process of this analysis several other issues have been raised. Most obvious is the assumption of a 'prehistoric' demography which is either dramatically worse, dramatically better, or just different from modern populations. In this examination of an agricultural population it is evident that there are major similarities between agricultural societies in the past and peasant societies today. The difference in adult mortality between skeletal and modern national populations has blinded researchers to the structural similarities between small populations in the past and small populations today. While hunger and disease in the past did not generally operate on the global scale observed today, on a local level no doubt these conditions did at times apply. This should not be read as advising researchers to use any modern small population as an analogy to the past, but rather to examine the results of skeletal research in the knowledge of what conditions occur in small traditional populations.

The analysis has also focused upon the possibility of localized extinctions and demographic instability. While it is tempting in archaeological reconstructions of diachronic change to see history as broad sweeps of unchanging, or very slowly evolving conditions, this superficial picture is based upon the conglomeration of experiences of small population groups. As the difference between DS3 and Saar, or between Keneba and Manduar indicates, even within 10 kms and within the same environment, different conditions may occur. If the role of archaeology is to research change in the past, then this research should be concerned not just with describing change but also with examining the nature and causality of change. This can only be fully done by looking at particular histories and then the interaction between a particular history and history on a regional level.

This book advocates the realisation that the comparison of frequencies of pathological lesions between populations is not necessarily an end within itself and that these numbers and percentages are indicative of human experience. Population characteristics and disease reconstruction need not be treated as end results but as part of the revelation of an adaptive biosocial cycle.

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Appendix 1
Construction of a life table

D_x: Age distribution of deaths.

d_x: Proportion of deaths/age interval (calculated to 100) $d_x = D_x / D_x \times 100$

l_x: Number of survivors of each age interval based on a radix of 100. $l_o = 100$; $l_x = l_{x-1} - d_{x-1}$

q_x: Mortality rate; the proportion of individuals of age x who will die before reaching age x+n.
$q_x = d_x / l_x$

L_x: Number of years lived in each age class by all those entering age class x. $L_x = n(l_x + l_{(x+n)}) /2$ [This is based on the assumption of an even distribution of deaths per year in that age category. Weiss suggests the use of approximations used by Coale and Demeney to compensate for the uneven distribution of deaths at ages 0,1,5 and 10 (Weiss 1973):
$Lo = .35(100) + .65 (l_1)$
$L_1 = 1.361(l_1) + 2.639 (l_5)$
$L_5 = 2.5(l_5) + l_{10}$
$L_{10} = 2.5(l_{10}) + l_{15}$
Before using these approximations however, it is important to demonstrate that the shape of deaths during childhood is similar to that of the Coale and Demeney tables. To accommodate the final age interval two maximum ages will be calculated: 70 and 80 years.]

T_x: Total number of years to be lived by those reaching age x.
$T_o = L_x$
$T_x = T_{(x-n)} - L_{(x-n)}$

e_x: Life expectancy or the average number of years left to those entering age x.
$e_x = T_x / l_x$

C_x: The proportion of the living population/age class.
$C_x = L_x / e_o$

Following the methodology suggested by Palkovich (1978), sample variance was also calculated:

Sq^2_x: sample variance for the probability of death for each age interval. This is calculated in the basis of q_x values since this value is not subject to cumulative error.
$Sq^2_x = q^2_x (1 - q_x)/D_x$

The above formulae are for use with a stationary population. Given a growing population, the number of survivors in each age group is proportional to the number of deaths adjusted by r. To calculate this it is necessary to convert the age distribution of a burial population into the age distribution of deaths of a cohort born during the same interval. The two are not identical since each cohort in a growing population is successively larger, although the actual shape of age distribution does not change over time [Preston & Coale 1982]).

In order to calculate population structure based on a growth rate, the methodology of Asch (1976) was followed. Thus:

$e^{rx}D_x$: The number of deaths at age x in a hypothetical cohort, where e is the base of natural logarithms, r is the rate of growth, and $x = [x + (x+n)]/2$

Survivorship and mortality are both calculated in the same manner as for the stationary population. Age structure of the living population, however, has to take account of the fact that older age groups come from smaller initial cohorts in a growing population than a stationary case where all cohorts are of the same initial size. Therefore:

C_x : Age distribution of the living growing population.
$Cx = e^{-rx}{}_n L_a / e^{-rx}{}_n L_x$

Life expectancy is no longer identical to the mean age of death.

Other elements of the life table

Final elements of the life table are:

Crude Birth Rate:
The number of births per individual per year.
$b = d = 1/e_o = 1/ (L_x / l_o)$ Stationary case: r=0
$b = d + r = 1/ e^{-rx} l_x / l_o$

Dependency Ratio:
The proportion of the population which is either juvenile or elderly compared to the productive proportion (both reproductively and economically).
$DR = C_{(0,15)} + C_{(50,w)} / C_{(15,50)}$
[60 years is frequently considered the beginning of elderly status but given the truncation of age classes to age 50 in skeletal populations 50 years is chosen here following the convention of Weiss (1975:40)].

Appendix 2
Number of skeletal elements

Subadults—DS3

	Age Group (yrs)					
Element*	0 - 1	1 - 3	3 - 6	6 - 10	10-15	N
Occipital	35	22	11	5	6	163(79)
Parietal	78	43	23	8	18	300(170)
Orbit*	46	20	13	7	5	671(91)
Frontal	31	17	9	6	7	111(70)
Face	18	13	12	3	3	108(49)
Basicranium	27	9	11	2	2	51(41)
Endocranium	17	11	7	3	3	187(41)
Temporal*	64	30	19	7	5	504(125)
Mandible	30	14	13	4	2	229(63)
Vertebrae	20	10	5	4	3	112(42)
Pelvis*	58	22	21	12	15	522(128
Ribs	29	10	8	3	1	154(51)
Sternum	1	1	5	0	0	7(7)
Clavicles*	32	15	13	9	4	383(73)
Scapula*	42	18	15	6	4	406(85)
Humerus*	64	32	15	14	18	521(143
Radius*	50	22	11	12	12	400(107)
Ulna*	56	25	11	9	7	522(108)
Hand	2	2	2	2	1	8 (8)
Femur*	87	29	20	14	13	573(163
Patella*	5	4	0	0	4	13(3)
Tibia*	49	22	15	7	9	462(102
Fibula*	26	10	12	9	5	264(62)
Foot*	2	6	4	2	7	191(21)

*Left and right combined
() Numbers of identifiable individuals

Subadults—Saar

Element	0 - 1	1 - 3	3 - 6	6 - 10	10-15	N
Occipital	2	2	1	1	2	8
Parietal*	1	4	7	3	8	23
Orbit*	8	4	5	2	5	24
Frontal	2	2	4	2	4	14
Face	1	2	2	1	1	7
Basicranium	1	1	1	1	3	7
Endocranium	3	0	1	0	0	4
Temporal*	3	3	5	2	6	19
Mandible	1	2	2	0	1	6
Vertebrae	1	2	2	1	3	9
Pelvis*	2	4	5	2	6	19
Ribs	3	2	2	1	2	10
Sternum	0	0	0	0	2	2
Clavicle*	4	0	1	2	5	12
Scapula*	5	3	3	2	6	19
Humerus*	6	4	5	2	8	25
Radius*	0	4	3	2	6	15
Ulna*	4	4	5	2	7	22
Hand	0	1	-	-	1	2
Femur*	5	4	4	2	8	23
Patella*	0	0	0	0	2	2
Tibia*	3	2	3	2	6	16
Fibula*	3	2	3	2	2	12
Foot*	0	2	3	0	3	8

Adults—DS3

Element	Male						Female						Total					
	6	7	8	9	10	N	6	7	8	9	10	N	6	7	8	9	10	N
Occipital	1	8	14	7	6	**67**	4	19	14	6	6	**57**	5	27	28	1	36	**125**
Parietal*	3	18	36	18	14	**150**	11	46	30	20	1	**156**	16	55	66	38	16	**312**
Orbit*	4	12	19	12	17	**105**	8	39	22	14	1	**116**	12	51	41	26	18	**122**
Frontal	2	6	15	9	8	**63**	3	17	14	10	1	**63**	5	23	29	19	9	**126**
Face	2	2	3	1	5	**24**	2	7	4	3	0	**20**	4	9	7	4	5	**44**
Basicran.	1	4	3	2	3	**26**	3	9	6	4	1	**29**	4	13	9	6	4	**55**
Endocran.	1	4	5	2	4	**29**	2	14	10	5	1	**44**	3	18	15	7	5	**73**
Temporal*	2	18	24	12	14	**115**	9	31	26	16	4	**126**	6	49	50	28	16	**251**
Mandible	1	5	5	2	6	**29**	4	7	10	8	1	**34**	5	12	15	10	7	**61**
C. Vert.	2	9	18	7	12	**60**	4	18	13	11	1	**57**	6	28	31	18	13	**119**
T. Vert.	2	10	19	8	13	**67**	4	19	15	9	1	**63**	6	30	34	17	14	**132**
L. Vert.	2	11	20	8	13	**68**	4	18	16	9	1	**60**	6	30	36	17	14	**130**
Sacrum	3	9	11	7	12	**48**	4	10	10	8	1	**38**	7	20	21	15	13	**87**
Pelvis*	3	19	30	18	29	**127**	9	30	38	14	4	**109**	12	55	76	32	33	**236**
Ribs	2	7	8	7	10	**47**	2	14	10	8	1	**42**	5	21	18	15	11	**90**
Sternum	2	4	5	5	6	**28**	1	9	4	5	1	**24**	3	13	9	10	7	**55**
Clavicle*	6	23	28	9	19	**111**	7	30	29	20	3	**119**	15	61	57	29	22	**235**
Scapula*	4	15	18	8	20	**79**	7	20	14	10	3	**66**	11	34	32	18	23	**145**
Humerus*	5	20	30	14	25	**134**	12	34	24	12	3	**111**	17	54	54	26	28	**146**
Radius*	3	19	18	12	21	**123**	12	28	24	12	3	**104**	16	48	60	24	24	**229**
Ulna*	6	8	28	14	22	**111**	*	25	20	10	2	**94**	15	41	48	24	24	**205**
Hand	3	4	2	5	4	**25**	2	6	7	2	0	**22**	5	10	9	7	4	**47**
Femur*	5	22	39	14	24	**139**	9	34	24	12	3	**116**	14	56	63	26	27	**255**
Patella*	1	15	21	13	7	**75**	6	26	9	7	1	**61**	8	41	30	20	8	**69**
Tibia*	3	13	29	9	17	**104**	12	27	21	8	2	**89**	15	40	30	17	19	**193**
Fibula*	5	9	23	11	15	**85**	6	28	20	9	3	**79**	11	38	43	20	18	**165**
Foot*	4	7	22	10	20	**74**	6	27	13	8	2	**65**	11	34	35	18	22	**142**

Adults—Saar

Element	Male						Female						Total					
	6	7	8	9	10	N	6	7	8	9	10	N	6	7	8	9	10	N
Occipital	0	2	2	2	3	**9**	0	3	4	6	2	**15**	2	4	6	7	4	**30**
Parietal*	2	3	7	6	4	**22**	2	13	13	6	2	**44**	6	18	20	18	8	**87**
Orbit*	1	2	8	8	5	**24**	2	8	10	12	1	**33**	3	10	18	18	8	**73**
Frontal	1	1	3	2	3	**10**	1	6	6	10	1	**19**	2	7	9	7	4	**36**
Face	0	1	3	3	1	**8**	1	2	1	5	1	**11**	1	4	4	9	2	**22**
Basicran	0	2	1	3	1	**7**	1	2	1	6	1	**9**	1	4	2	7	2	**17**
Endocran	1	2	1	2	1	**7**	1	3	3	4	1	**11**	2	5	4	5	2	**19**
Temporal*	1	3	9	7	5	**25**	2	12	13	3	7	**45**	5	15	22	18	12	**87**
Mandible	1	2	3	3	2	**11**	1	4	4	11	0	**14**	2	6	7	8	2	**28**
C. Vert.	1	2	3	5	4	**17**	1	4	9	5	2	**26**	3	7	12	12	6	**45**
T. Vert.	1	2	4	7	4	**20**	1	7	10	7	2	**31**	2	10	14	14	6	**53**
L. Vert.	1	2	4	7	4	**21**	1	8	10	7	3	**32**	2	10	14	14	7	**54**
Sacrum	1	2	5	4	2	**15**	1	3	5	7	2	**21**	2	5	10	11	4	**36**
Pelvis*	1	5	10	8	6	**32**	0	13	12	14	6	**49**	2	18	22	22	12	**81**
Ribs	0	2	3	5	2	**13**	0	4	5	5	2	**19**	1	7	8	10	4	**35**
Sternum	1	2	2	2	2	**11**	0	1	3	4	2	**12**	1	3	5	6	4	**23**
Clavicle*	1	8	10	10	6	**28**	2	17	15	8	15	**57**	2	19	21	19	13	**88**
Scapula*	2	5	8	6	5	**37**	1	8	11	13	2	**37**	4	13	19	19	7	**65**
Humerus*	2	5	7	11	6	**39**	1	13	21	12	4	**60**	4	18	28	23	10	**99**
Radius*	2	2	8	9	4	**30**	1	10	14	13	3	**49**	3	12	22	22	7	**79**
Ulna*	0	1	7	11	6	**29**	0	12	15	12	2	**46**	0	15	22	22	8	**77**
Hand	0	1	3	2	2	**9**	0	3	4	6	2	**18**	0	5	7	8	4	**28**
Femur*	0	6	8	10	6	**36**	2	14	12	12	4	**52**	2	20	20	22	10	**88**
Patella*	0	4	5	8	4	**23**	0	9	10	7	3	**33**	0	14	15	14	7	**57**
Tibia*	1	5	2	10	1	**24**	0	10	13	5	3	**35**	1	15	15	15	4	**59**
Fibula*	1	2	3	5	2	**14**	1	6	6	4	1	**22**	2	8	9	9	3	**36**
Foot*	0	1	1	2	2	**6**	0	6	4	5	1	**16**	0	8	5	7	3	**23**

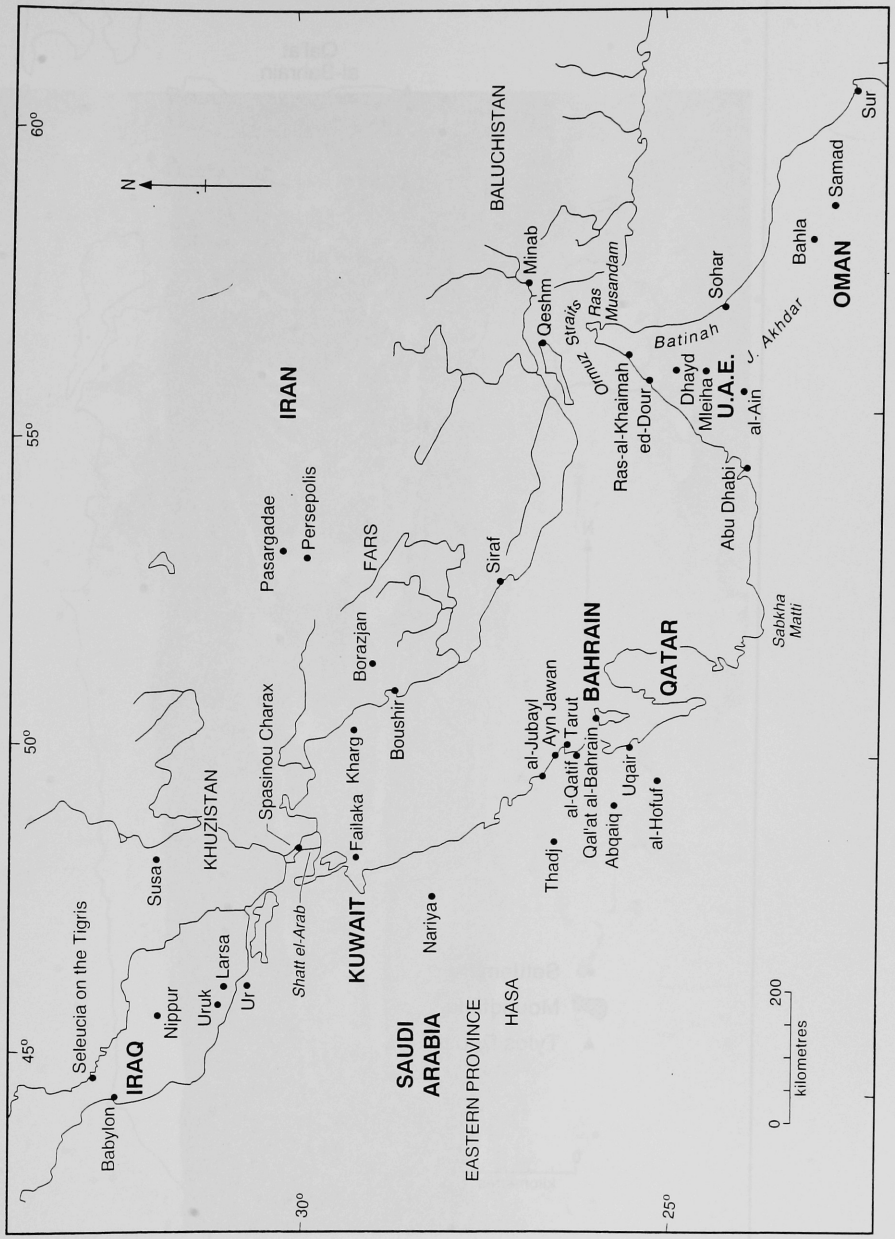

Plate 1. Location of Bahrain in the Arabian Gulf.

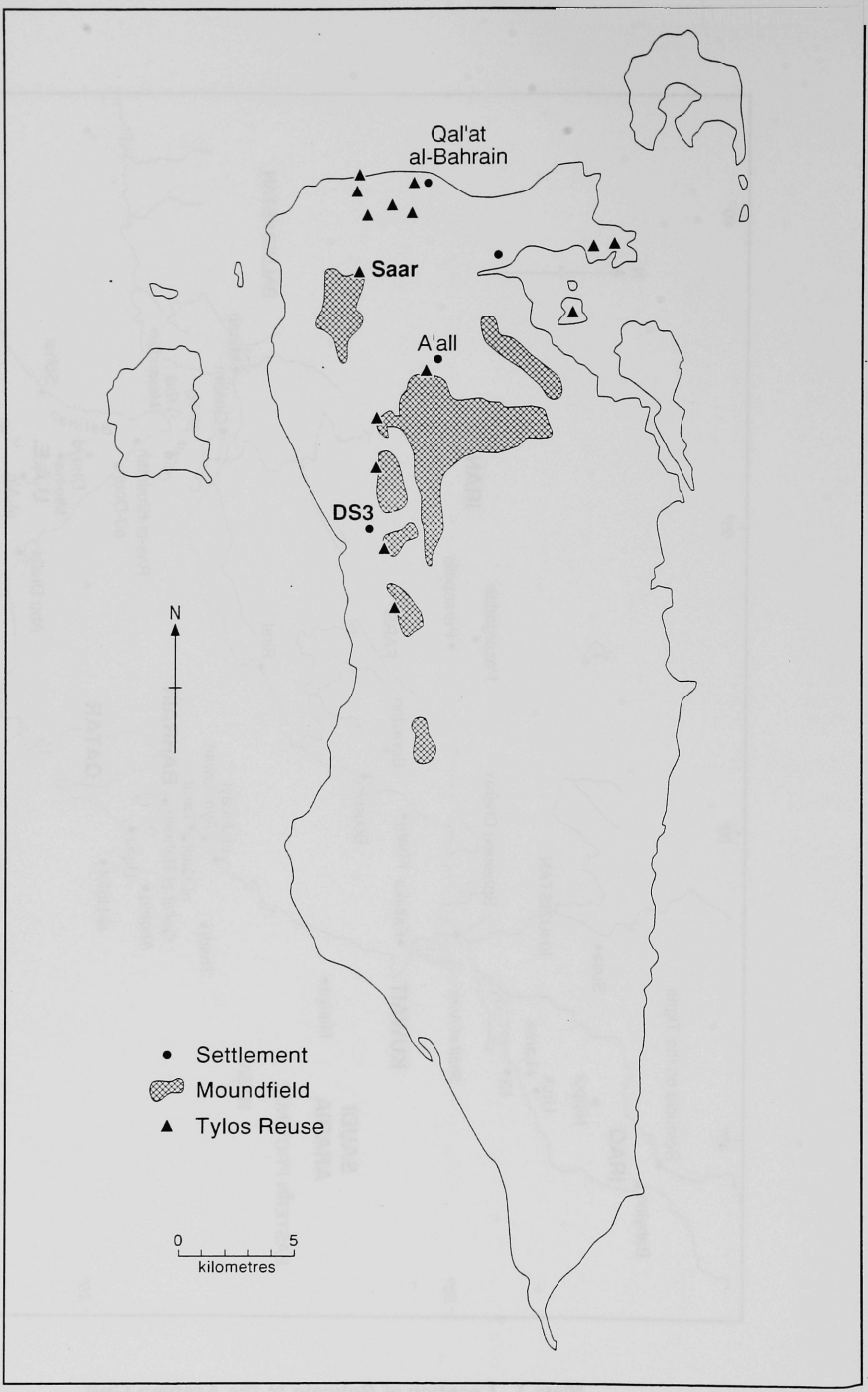

Plate 2. Tylos period cemeteries on the main island of Bahrain

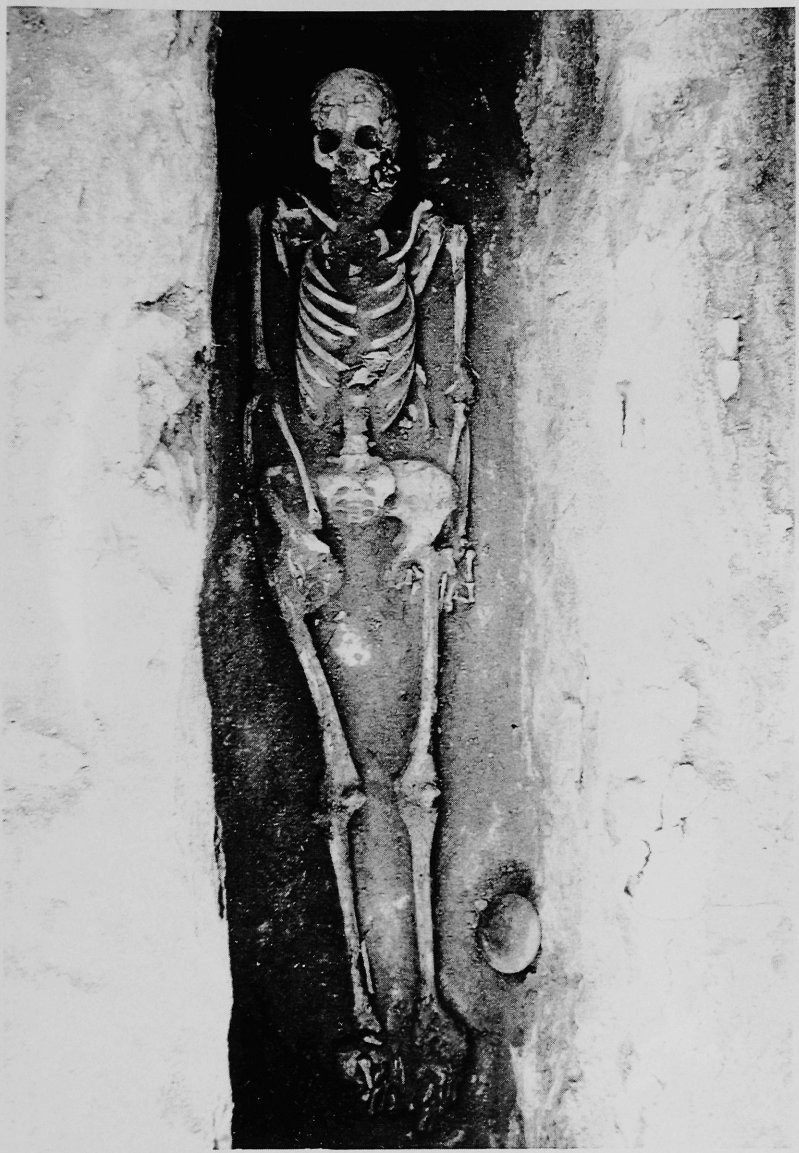

Plate 3. Tylos period grave.

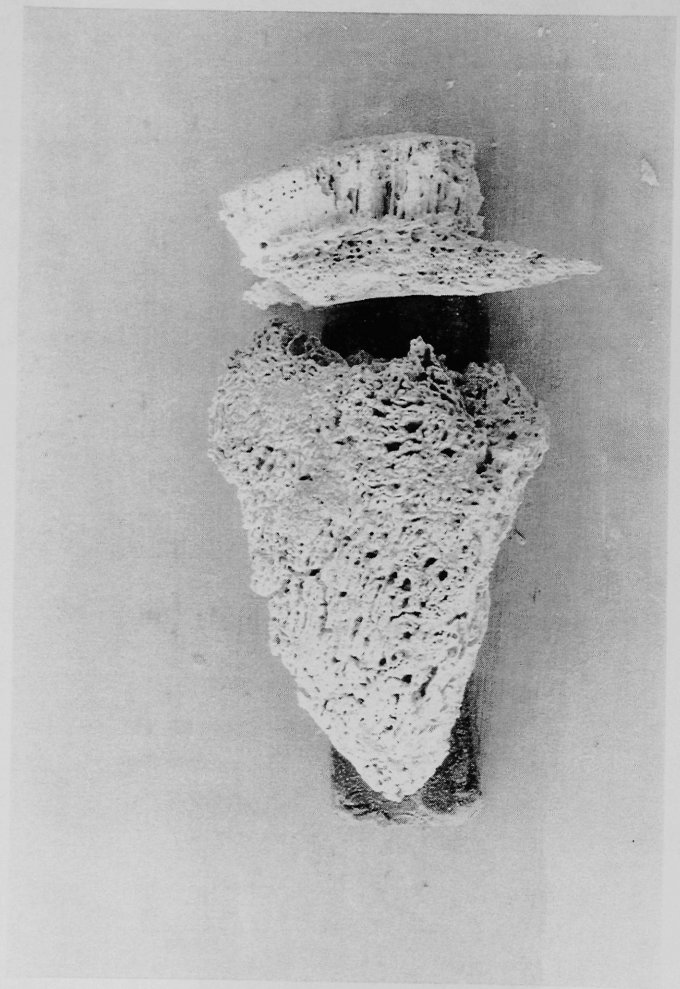

Plate 4. Porotic hyperostosis on child's calvarium.

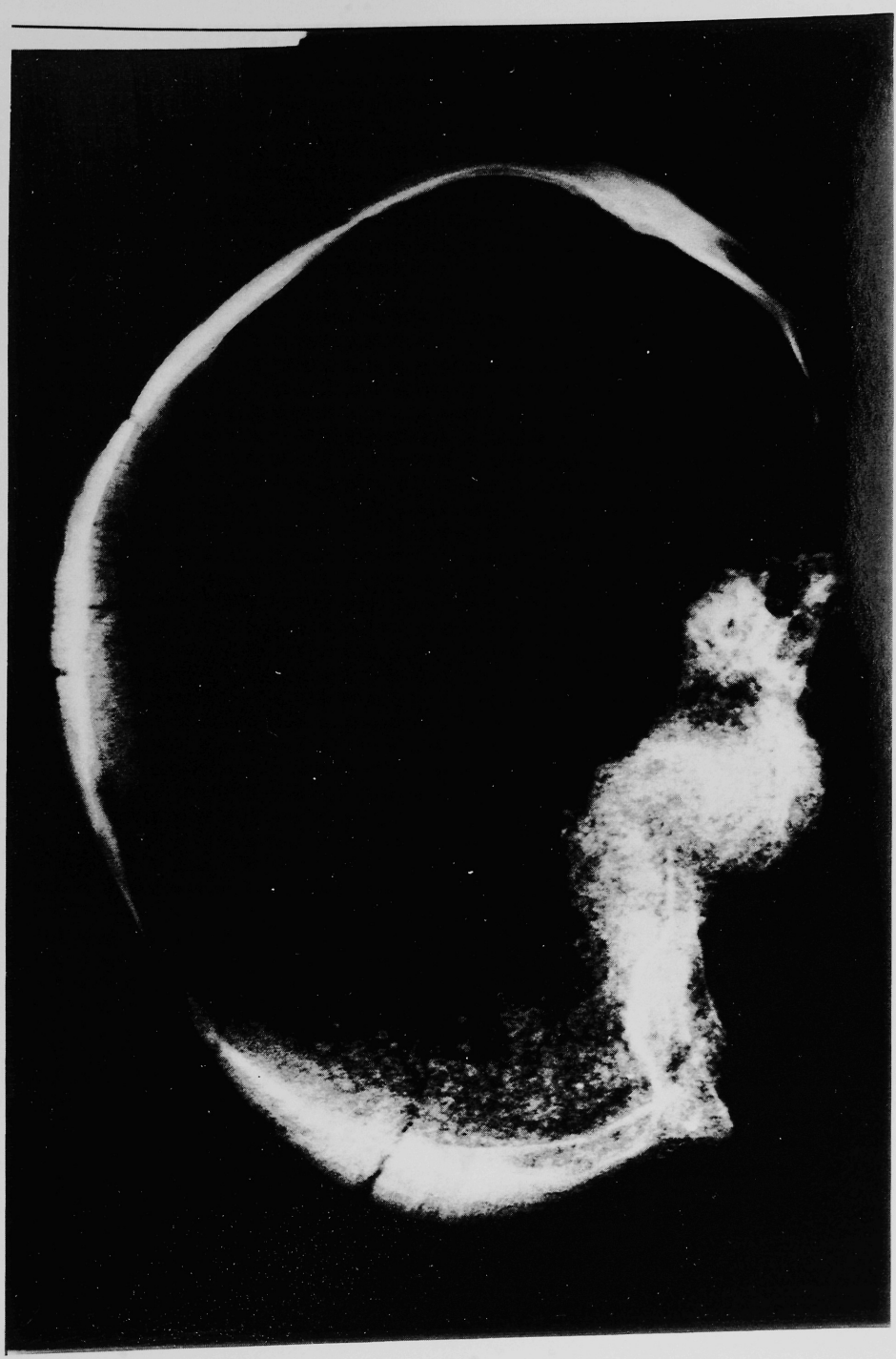

Plate 5. Hair-on-end appearance in child's cranium.

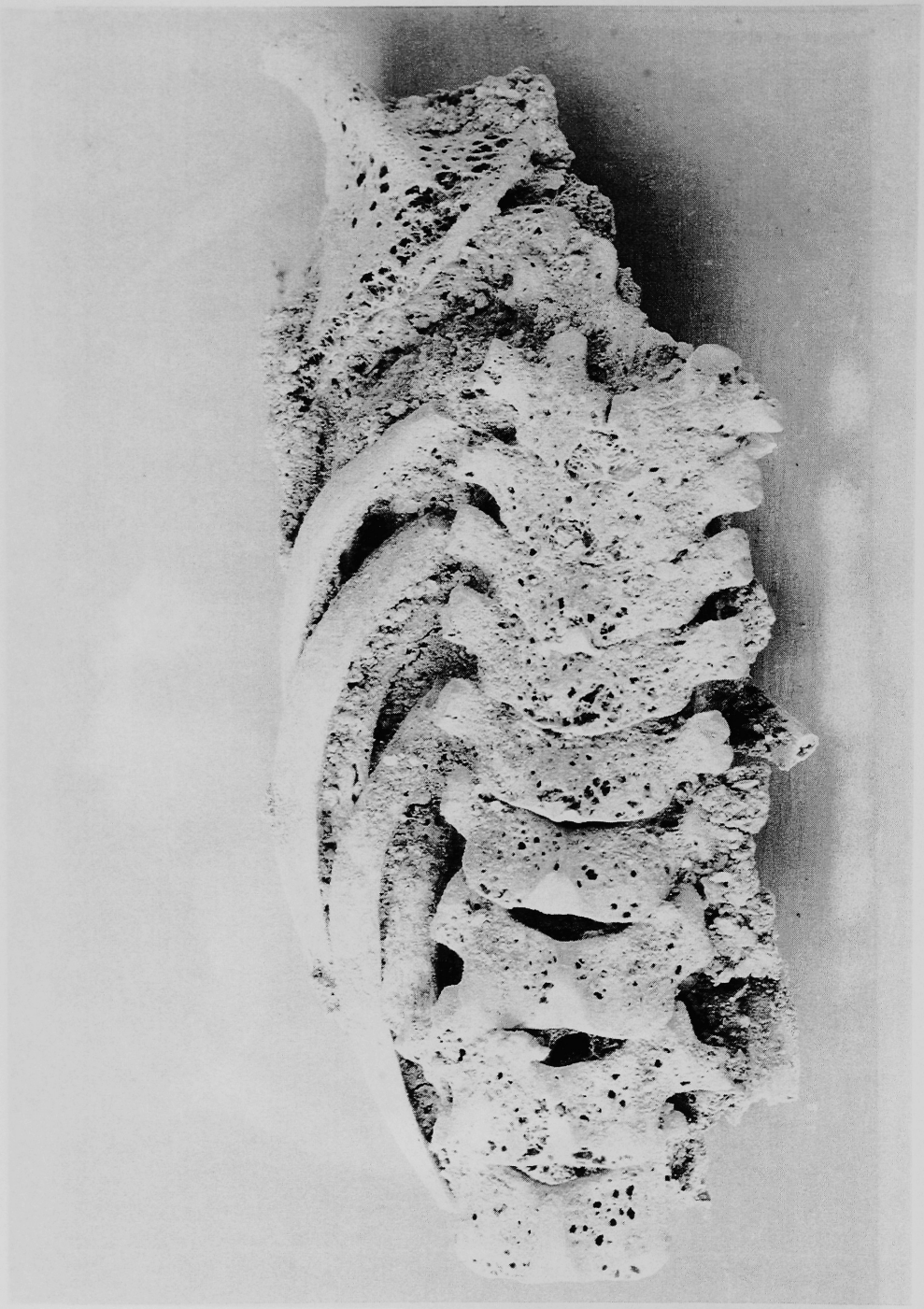

Plate 6. Post-cranial changes due to porotic hyperostosis.

Plate 7. Endocranial lesions in infant.

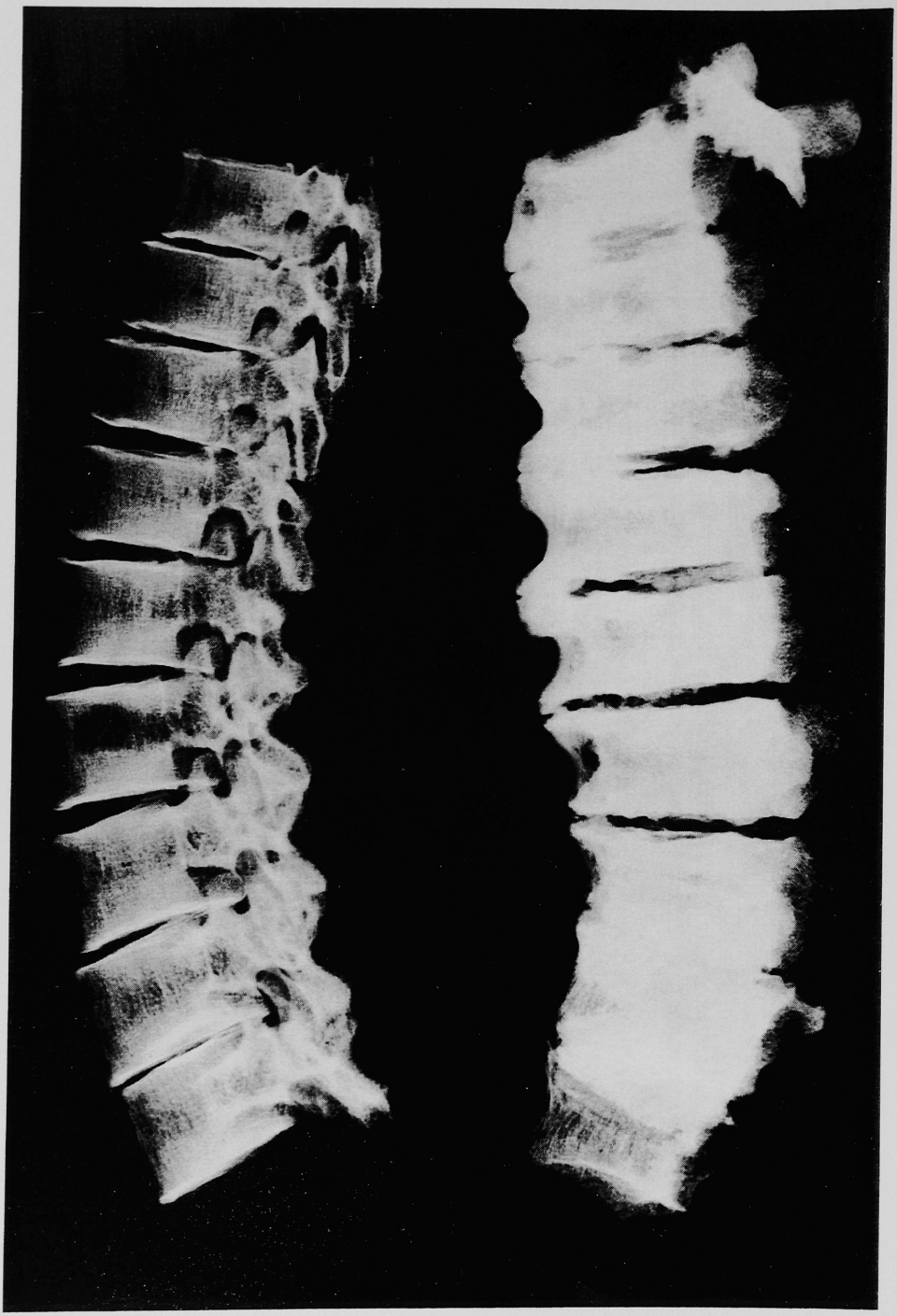

Plate 8. Radiograph of fluorosed spine (right) compared to normal (left).

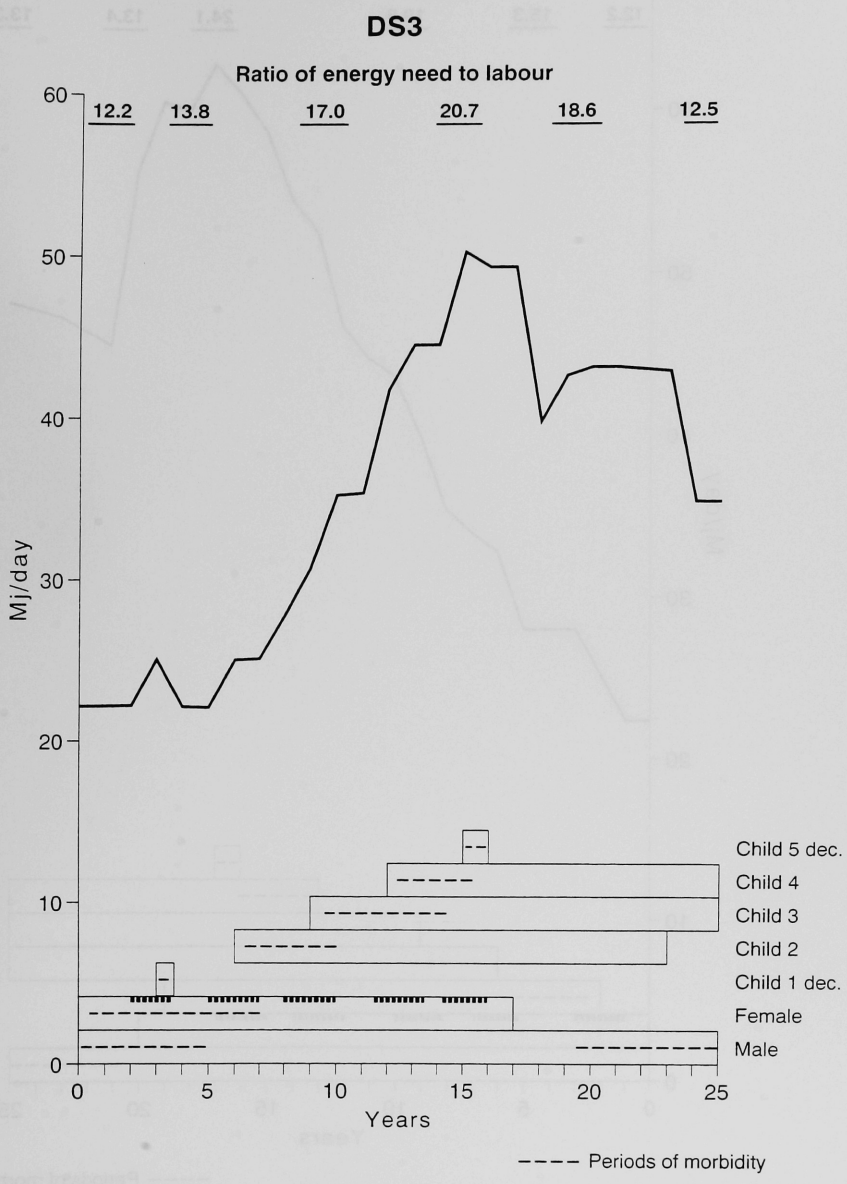

Plate 9. Model family cycle at DS3 adapted from Payne (1985).

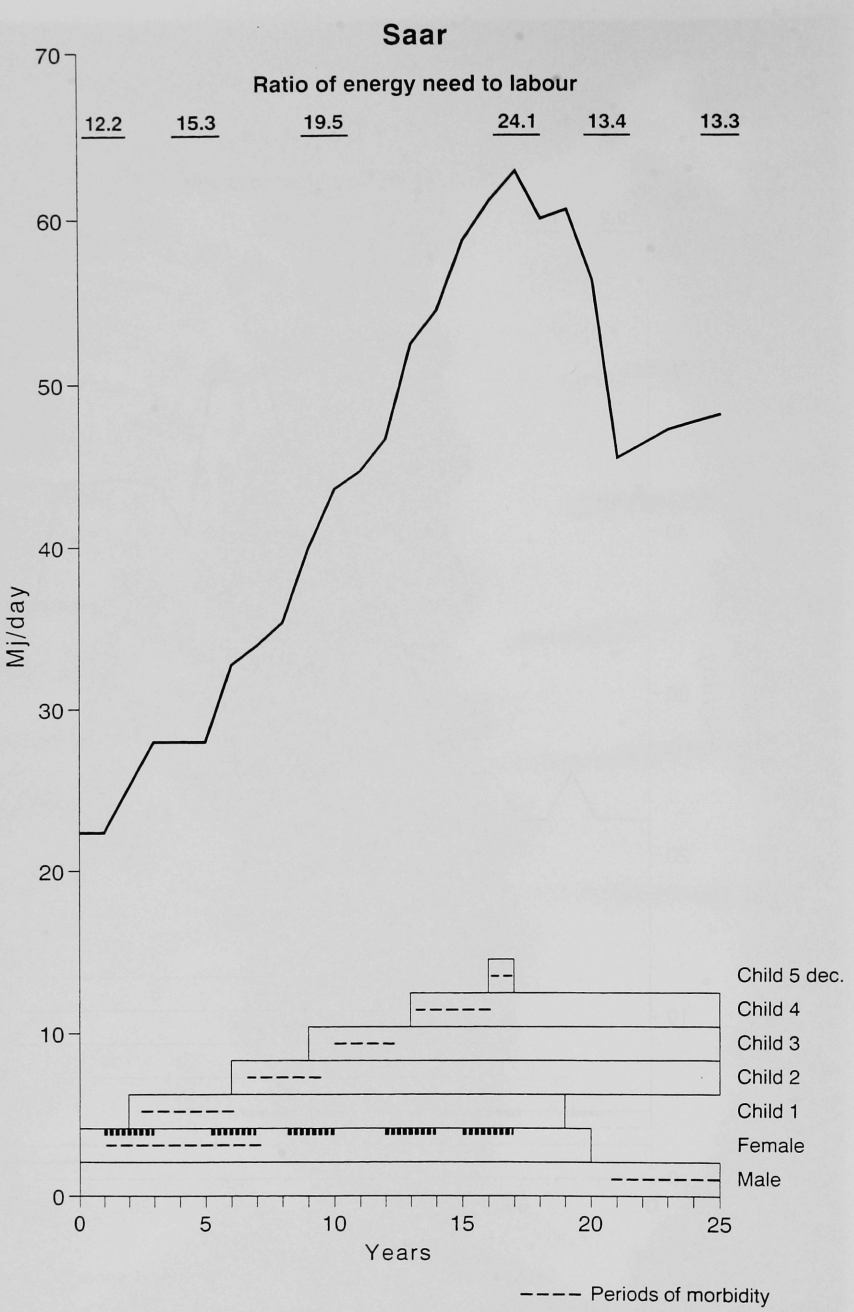

Plate 10. Model family cycle at Saar adapted from Payne (1985).