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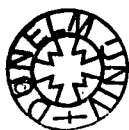
**BIOCULTURAL PERSPECTIVES ON BIRTH DEFECTS  
IN MEDIEVAL URBAN AND RURAL ENGLISH POPULATIONS**

**Judith Fiona Sture  
Department of Archaeology**

Thesis submitted for the Degree of Doctor of Philosophy  
At the University of Durham

**2001**

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**27 JUN 2002**

Thesis  
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**Judith Fiona Sture**

**Biocultural Perspectives On Birth Defects  
In Medieval Urban And Rural English Populations**

The biocultural and epidemiological approaches have been used as investigative methods by which to assess the prevalence of birth defects of the axial skeleton among five English Medieval population samples (Raunds Furnells, Northamptonshire; the hospital/almshouse of St James and St Mary Magdalene, Chichester, West Sussex; St Helen-on-the-Walls, York; Wharram Percy, East Yorkshire, and the Augustinian Friary cemetery, Hull, Humberside).

The author hypothesises that Medieval urban populations produced offspring with higher frequencies of skeletal defects because they were subject to the adverse health-mediating effects of higher population density. These include poor quality, frequently overcrowded living conditions, poor sanitation, increased rates of disease threat and transmission, poorer quality food and drink due to pollution and adulteration, and greater levels of industrial-related air and water pollution. The author proposes that this response was a consequence of the impaired interaction between a population-wide compromised nutritional status and a co-existing weakened immune response. It is proposed that rural populations will express significantly lower frequencies of the same skeletal defects, as they are not subject to the same adverse environmental effects of population density and urban living conditions. The results support this hypothesis among the four populations derived from burial grounds associated with residential areas, whilst the Hull population expresses a rural pattern of defect prevalence, raising questions of possibly limited, exclusive access to burial at that site, available to non-urban dwellers. The author suggests that similar reproductive effects may be found today in populations undergoing demographic transition, for example, those experiencing the process of urbanisation in the developing world, or those migrating to the developed West. The author also shows how the results, when viewed alongside the medical literature, may indicate the presence of soft-tissue anomalies which are invisible to those working with dry bone.

**Keywords:** urban, rural, medieval, birth defects, congenital, urbanisation, population density, biocultural, epidemiology, spine, cleft palate, skeleton, archaeology, palaeopathology.

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## ACKNOWLEDGEMENTS

I would like to thank the following people, without whose help I could not have undertaken, or completed, this PhD thesis.

The Arts and Humanities Research Board (The British Academy), who kindly funded my research for three years, thus enabling us to eat. At the Department of Archaeology at the University of Durham, Dr Charlotte Roberts for supervising my study, offering valuable advice and experience, and for presenting a poster for me at the Annual Meeting of the American Association of Physical Anthropologists in San Antonio, Texas, during April 2000; Dr Andrew Millard for co-supervising my work, and for his advice on statistics; Dr Jenny Price, and especially Sheila Brown, for their advice, encouragement and friendship, which kept me going when I felt like giving up.

For allowing me access to the Wharram Percy and the York material, I must thank Dr Simon Mays of English Heritage, and Dr Elizabeth Hartley of The Yorkshire Museum, York. Simon shared skeletal data with me, and along with Elizabeth, much valuable advice and insight into the materials that they curate, and I am extremely grateful to them. They also made it a pleasure to go to work in some less-than-perfect surroundings! I also thank Dr Anne Grauer, for kindly sharing her skeletal data on the York material; Dr Mary Lewis for her advice and help; Anthea Boylston of the Calvin Wells laboratory, and Dr Carl Heron, the Head of Department, both of the University of Bradford's Department of Archaeological Sciences, who kindly allowed me access to skeletal data relating to the collections held at Bradford.

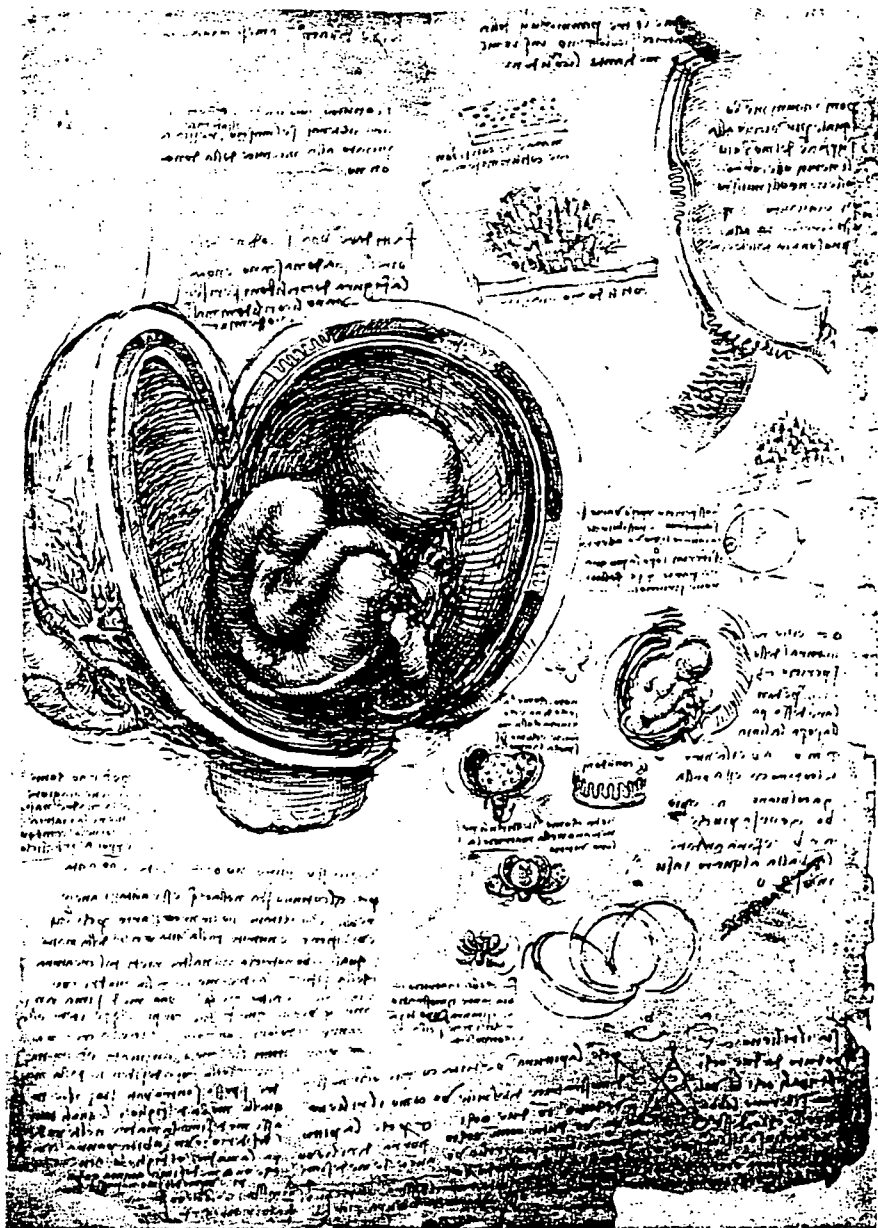
Also at the University of Bradford, I would like to thank Professor John Green of The Graduate School, who has offered his unstinting encouragement, support, advice and generosity. Also, Professor Tony Fell, Dr Wendy Olsen, Dr Terry Allen, Kevin Knifton, Julie Easterbrook and Karen Bailey, all for keeping me sane in the face of imminent mental derangement as I finished my thesis.

Professor Richard Smithells, Professor Malcolm Levene, Dr Michael Smith, and Ms Katie Brewster (osteopath), for their advice, answers and insights.

I especially would like to thank my parents and sister, and my friends Nadia Susdorf, Carol and David O'Keeffe, Maurice and Monica Moody, and Russell and Janine Dever, for putting up with my studies and associated angst for the last few years, and for their constant encouragement and support.

My greatest thanks, however, must go to my children, Jonathan and Hannah. They have supported, encouraged, and fed me, cooked, washed up, cleaned, ironed and generally done more than most children will ever do, while I have worked on my research. Without their support, sense of humour, constant encouragement and understanding, I would not have completed my research successfully. I will never find better research assistants.

"From the ashes of disaster grow the roses of success."



CHILD IN WOMB  
Royal Library, Windsor

## CHAPTER ONE

### Introduction

*"It has long been apparent that congenital malformations in children are, like cancer in adults, the biggest challenge to medical science, and that progress in one field may well yield dividends for the other." ..... "I would like to add to every research protocol within the whole field of teratology, and to every application for a research grant, a compulsory question: 'How do you see this work contributing to the understanding of human malformation?' For the central challenge lies in the hundreds of thousands of children born every year with spina bifida, anencephaly, congenital heart disease, cleft lip and palate; with abnormal eyes, ears, kidneys, bowels; with deformed limbs....we must never lose sight of the malformed multitudes. We must never forget that they are the challenge."*

*Smithells, 1980:85.*

#### 1.1 RECENT STUDIES IN BIOLOGICAL ANTHROPOLOGY

Advances in the field of biological anthropology have been significant over the last thirty years, particularly in the area of palaeopathology. This discipline is defined as the scientific study of diseases whose existence may be demonstrated in the bones and teeth of past human and non-human remains (Roberts & Manchester, 1995:1). Much work during the last twenty years has focused on improving methodology (e.g. Ortner 1991; Bush & Zvelebil, 1991; Buikstra & Ubelaker, 1994), and standard texts addressing ancient disease patterns and processes have appeared for the use of students and other interested professionals (e.g. Iscan & Kennedy, 1989; Roberts & Manchester, 1995; Larsen, 1997; Aufderheide, *et al.*, 1998; Cox & Mays, 2000; Katzenberg & Saunders, 2000). Substantial palaeopathological work has focused on dental disease (e.g. Hillson, 1986, 1996; Kerr, 1994); joint disease (e.g. Jurmain & Kilgore, 1995; Rogers & Waldron, 1995;); specific infectious diseases such as leprosy (e.g. Andersen *et al.*, 1994; Lee & Magilton, 1989), tuberculosis (e.g. Buikstra, 1981; Stirling & Waldron, 1990) and treponemal disease (e.g. Baker & Armelagos, 1988; Dutour, 1994); non-specific infectious disease (e.g. Boocock *et*





*al.*, 1995); metabolic and endocrine disorders (e.g. Ortner & Ericksen, 1997); neoplastic disease (e.g. Waldron, 1996); and trauma (e.g. Lovell, 1997). By comparison, the congenital diseases, whilst offering a fairly lengthy bibliography, have been addressed almost exclusively by case studies until Barnes (1994) and Sture (1997) undertook analytical studies. This thesis aims to develop the initial work of Sture (1997) by building on the advances in the understanding of patterns of congenital defects in archaeologically-derived populations which emerged from that work. The biocultural approach used by Sture (Roberts, 1989; Roberts & Manchester, 1995) involves the consideration of archaeological, documentary and environmental evidence alongside the biological evidence when forming an assessment of the results. By applying this technique to the study of birth defects in past human populations, it may be possible to relate environmental factors to the defect frequencies and patterns observed.

## 1.2 STUDYING THE CONGENITAL DISEASES IN SKELETAL MATERIAL

Defects of the skeleton due to developmental and growth disturbances *in utero* may be genetically- or environmentally-mediated. Neither causal factor has been adequately addressed previously in the archaeological literature. Certain easily recognisable (supposedly) defects such as *spina bifida occulta* have been documented many times, usually in reports which mention them solely as interesting anomalies, with no attempt at analysis to explain why the defects are occurring. Insufficient work has been done on analysis of congenital defects in archaeological samples, either with respect to their prevalence, type or severity, or with regard to the social, economic and health implications for the individual or the community. Brothwell and Powers (1968), Saluja (1988) and Hale (1990) considered *spina bifida* across

time and space, but this type of study has been uncommon. This study aims to address that deficit by taking the biocultural approach to the investigation of links between the environment and rates of congenital anomalies of the skeleton. The medical and epidemiological professions have appreciated the effect of environmental factors on the incidence of certain congenital disorders for some time, but this knowledge has yet to be applied to past populations by biological anthropologists.

The study of birth defects in skeletal populations is unique in that the observed defects are known to have had their origins during a specific time frame, i.e. the thirty-eight week period of gestation. Other signs of disease evident on the skeleton, for example osteoarthritic changes or specific infectious disease processes, cannot, in a living or dead individual, usually be attributed to a specific time of origin. Exceptions include the presence of woven bone as a result of infection indicating active disease at the time of death, but usually it is chronic healed lesions that are observed in the skeleton. Such lesions may be indicative of a variety of disease processes, and identification of the correct cause may be impossible. A defect of skeletal formation, on the other hand, is usually very clearly either present or absent, making assessment relatively straightforward, with fewer possible differential diagnoses necessary. For example, developmental union of vertebrae can be distinguished from a traumatic or infectious event by the nature of the bone formation - there is often no line of union or new bone growth present because the elements were never separated (Barnes, 1994:66). Assuming an abnormality was not directly life-threatening, such as a serious neural tube defect, then an affected individual could feasibly survive into adulthood, allowing us to observe affected

adults as well as children. Issues of infanticide arise when considering some of the more visible and “deforming” defects, and affected individuals possibly had reduced chances of survival and/or of appearing in a “normal” cemetery population due to cultural notions about deformity (Gregg *et al.*, 1981). This suggestion is borne out by the apparent lack of, for example, craniofacial disfigurement observed within skeletal populations (although facial bones are fragile, which may hinder diagnosis), even though many of the factors associated with such defects today were relevant in the past, such as exposure to leather production, and certain viral infections (Bianchi *et al.*, 1997; Garcia & Fletcher, 1998; Hagberg *et al.*, 1998; Knox & Lancashire, 1991; Saxen, 1983). Some studies, however, indicate that “deformed” individuals in the past were accepted by the society in which they lived, and at least some such individuals reached adulthood (e.g. Chadwick-Hawkes & Wells, 1976; Webb & Thorne, 1985). By looking at occult (hidden) defects, many of which the affected individuals would not have been aware of, rather than manifest anomalies, it is likely that a more accurate prevalence will be observed – one which has not been so subject to prejudicial cultural interference. The study of skeletal birth defects may therefore contribute to modern health research by offering a picture of prevalence in and between past pre-industrial populations. It also offers potential insights into past notions of disability by assessing the relative socioeconomic and health status of affected individuals (where recognised), through such data as associated pathology, relative stature, and general health status, all of which may go some way to indicate well-being and/or care or tolerance in that society.

### 1.3 AN ARCHAEOLOGICAL APPROACH TO BIRTH DEFECTS: AIMS OF THIS STUDY

This study arises directly from the perceived lack of analytical work in the field of birth defect research within palaeopathology. The congenital diseases in the past are long overdue for an analytical assessment by biological anthropologists. The case-study approach is not only out-dated and lacking in investigative vigour, but it also highlights the short-sighted perspective of a discipline which should be seeking an active role in contributing to modern epidemiology. The study of human remains and assessment of disease in the past has the potential to offer insights into human health that clinicians working with living populations are unable to access easily. This study combines an assessment of several excavated population samples from urban and rural environments in the Medieval period (9<sup>th</sup> -16<sup>th</sup> centuries AD) with modern clinical data in an attempt to elucidate the following:

- the prevalence of birth defects of the axial skeleton within the populations
- a statistical assessment of the defects observed in terms of environmental context, sex and life-expectancy perspectives
- the socio-economic implications for those affected, their dependants, and society as a whole
- the associated soft-tissue anomalies which are often seen alongside such defects in living populations, which are “invisible” in skeletal material
- suggestions as to the environmental factors which may be acting on the development of the skeleton *in utero*

In effect, this study is an epidemiological investigation into the frequency patterns of certain birth defects, and aims to offer suggestions regarding their environmentally-

mediated aetiology. The author hopes to demonstrate that the biocultural investigation of archaeologically-derived human skeletal material may be a useful epidemiological research tool for the study of the congenital diseases in the future.

#### **1.4 THE WORKING HYPOTHESIS**

The hypothesis investigated by this study states that “Medieval urban populations will express higher rates of skeletal birth defects than their rural counterparts because they are exposed, during their childhood and/or reproductive years, to the effects of relatively higher population density and the urban lifestyle”. These effects include the hazards of crowding such as exposure to larger numbers of people and new infections, associated with raised pathogen loads (Bogin, 1988), inadequate disposal of rubbish and sewage leading to poor communal hygiene (Hall *et al.*, 1988; Roberts & Manchester, 1995), poor quality housing with greater occupancy (Goldberg, 1992), increased exposure to smoke and fumes from industrial processes and domestic fires (Lewis & Roberts, 1997; Reynolds, 1977; Richards, 1993), less light exposure due to the indoor nature of much urban work (Goldberg, 1992, Hodgett, 1972), plus a higher likelihood of food adulteration (Goldberg, 1992), and poor water quality (Hall *et al.*, 1988). In addition, the increased psychological stress levels for those removed from traditional family networks is likely to have caused certain physiological responses which serve to reduce the effectiveness of the immune response, which is, of course, a vital defence against the above named hazards (Brothwell, 1994; Hiramoto *et al.*, 1999; Ursin, 1994). The author suggests that the symbiotic relationship between nutritional and immune status (Chandra, 1978, 1979, 1980; Ferguson, 1993) is impaired by the raised infection rates and pathogen loads

experienced by urban dwellers, and during their reproductive years, affected females expressed this by producing more infants with axial skeletal defects than did their rural sisters. The author suggests that this response is either a result of pregnant females having expended significant physical resources on fighting infection, thereby re-directing nutrients away from the foetus, or of the pathogen load of the mother having a teratogenic effect on the foetus (see Chapter 4). There may be elements of both processes involved in defect aetiology, but quantifying the relative influences is for future work. There may be a paternal role in the aetiology of the observed defects, as spermatogenesis may also be similarly affected by the infection-related hazards outlined above (Cowan, 1997). However, much research shows that nutritional excesses or deficiencies in the mother (before or after conception) have a significant effect on the offspring (Allen, 1986; Bell *et al.*, 1975; Emanuel & Sever, 1973a; Frisancho *et al.*, 1977; Khera, 1984; Laurence *et al.*, 1983; Smithells, 1983, 1992; Smithells *et al.*, 1981), as well as the associations found with certain infectious agents (Knox & Lancashire, 1991; Saxen, 1983; Levene & Tudehope, 1993).

The hypothesis will be investigated by identifying and quantifying differences in the *frequency* of birth defects of the axial skeleton in archaeological populations from English Medieval urban and rural environments. The study also aims to offer suggestions as to why such variations occur. Multifactorial inheritance, due to the interaction of genetic and environmental factors, is the commonest identifiable cause of defects today (20%), and when identifiable genetic factors are added, this accounts for approximately one third of defects. The majority of defects, however, remain idiopathic in origin (60%), and maternal illness and congenital infection account for approximately 5% of defects (Connor & Ferguson-Smith, 1993). The clinical

literature associates certain congenital defects of the skeleton with soft-tissue anomalies that are, of course, invisible to the biological anthropologist. By considering skeletal defects in the observed populations in the context of the clinical literature, and of embryological development, this study aims to provide evidence that:

- a) rates of congenital defects of the skeleton are, to some degree, associated with maternal/paternal environment before conception and/or during gestation;
- b) congenital defects of the skeleton may be used as a key to suggest the presence of certain soft tissue anomalies which do not survive in the archaeological record
- c) will enable measures to be developed which may assist in prevention of such defects in modern populations under epidemiological transition.

### **1.5 CONGENITAL DISEASES AND DISABILITY IN ARCHAEOLOGICAL CONTEXTS**

Disability has been defined as a biologically-imposed phenomenon, with handicap as a sociocultural overlay, dependant on the acceptance of the disability by the general populace (Wright, 1960). Notions of disability and handicap may obviously change across time and space, being influenced by factors such as religious belief, or the economic and political milieu prevalent at any given period. The study of disability in archaeological contexts obviously involves some consideration of birth defects, along with other forms of physical disadvantage. Cultural ideals seem to play a part in the acceptance, tolerance or rejection of certain congenital anomalies. The entertainment value of affected individuals has been a focus in the past. For example, it has been claimed that actors were frequently recruited from amongst cleft

palate defect sufferers in the past, an Egyptian statuette being cited as an example (Fiori, 1983). Similarly, dwarfs have long been associated with entertainment either as actors or as jesters (Johnston, 1963). Cleft defects, however, appear more frequently in the archaeology of some cultures than others, suggesting the possibility of differential acceptance, as well as the possibility of true prevalence differences. Numerous archaeological reports have described “disabled” individuals, including those affected by trauma and disease processes which have rendered them permanently marked in some way (Roberts, 2000). Roberts points out that visible birth defects in skeletal remains usually led to authors inferring the presence of some sort of care system, although this may not always be the case. Much of our knowledge of birth defects and/or apparent disability in the past is dependent on iconographic evidence, particularly with regard to the soft tissue manifestations. Occasionally, investigative research by authors describing interesting archaeological examples has offered insight into likely disabilities (e.g. Arcini & Frolund, 1996; Wells, 1979). This study considers disability/handicap (handicap is not necessarily a disability) in terms of some of the conditions observed in the sample populations and is addressed in the discussion (Chapter 7).

## **1.6 PALAEOPATHOLOGY AS EPIDEMIOLOGY**

The twentieth century saw the recognition of several major environmental hazards to human health, particularly those affecting the human foetus. Interest today is increasing in the identification of environmental hazards which may have an adverse effect on reproductive health and the foetus itself (Sever, 1995). Diseases such as rubella and toxoplasmosis have been identified as the cause of abnormalities



observed in both newborns and older children, as defects become apparent when expected development fails to occur. Similarly, exogenous substances such as chemicals and drugs have become the subject of strict controls once evidence of their toxicity has been established. The thalidomide disaster of the early 1960s showed a new side to medicine - one that incurred costs rather than simply conferring benefits. Infants were born with defects including limb-reduction (phocomelia) which were directly linked to the drug, which had been taken by their mothers as a treatment for morning sickness during the first trimester of pregnancy. The “re-introduction” of thalidomide as a treatment for other disorders is currently causing alarm and outrage amongst the public (Wright, 2000). Chemicals, especially those associated with agriculture, such as herbicides and pesticides, have also been the subject of much public concern and many studies. Agricultural chemical usage has been linked to the occurrence of malformations such as neural tube defects, limb-reduction effects and cleft lip and palate (Sever, 1995: 92).

By their very nature, epidemiological studies may take many years, even decades, to produce evidence of association between certain agents and ill-effects on human health. Even when such associations are documented and accepted, proving a cause-and-effect relationship between Agent A and Illness B is far from straightforward (Traven *et al.*, 1995). Epidemiologists are unable to conduct investigations by experiment - they must rely on observation of “free-living populations”. To reach accurate conclusions from such uncontrolled, and often unreliable, source material is difficult, yet it remains the only option for environmental epidemiologists. Problems associated with epidemiological research include the difficulties in keeping track of

subjects, loss of subjects from the study, biased information from subjects and problems in accurately estimating exposures (Barker & Hall, 1991).

Historical populations, excavated from cemeteries by archaeologists, have considerable potential as ready-made samples of humans from the past which may be studied in an environmental framework. The "Urban-Rural" theme has been investigated in several recent studies by palaeopathologists (e.g. Judd, 1994; Lewis, 1999; Lewis *et al.*, 1995; McMullen, 1998; Orvik-Kondratiev, 1998), and considers comparative health between populations in these two environments in the past. With regard to the limitations of palaeopathological study, Roberts and Manchester (1995:9) noted the problems of interpretation in palaeopathology outlined by Wood *et al.* (1992) including selective mortality, individual variation and the non-stationary nature of populations. They also considered the problems of observing "a sample of a sample of a sample", the slow nature of disease processes acting on the bone, and the difficulties inherent in assessing the cause of death of an individual.

Notwithstanding these limitations, Roberts and Manchester are confident that the constant nature of disease as viewed in the large amount of skeletal material already examined, and the clinical foundations of modern palaeopathology, mean that it is possible to glean much valuable information from excavated dry bone. By employing a biocultural approach, that is, by considering the biological evidence for disease in a cultural context, it is possible to identify the spread and changing nature of diseases associated with, for example, the adoption of agriculture, urbanization and increased travel in the past. From the examination of individuals, data for age-at-death, sex and stature may all be ascertained with varying degrees of accuracy (see Buikstra & Ubelaker, 1994), and these methods are likely to improve in accuracy in

the future. In addition, infectious, traumatic, neoplastic, metabolic, autoimmune, joint, endocrine and congenital diseases may all be recognised in skeletal material (Roberts and Manchester, 1995:14). As mentioned above, the congenital diseases are usually recognisable as either present or absent, thus largely allowing a more straightforward diagnosis.

### **1.7 THE ARCHAEOLOGICAL SAMPLES AND THEIR STUDY**

The cemeteries of Wharram Percy, a deserted medieval village in the Yorkshire Wolds, The Augustinian Friary, Hull, and St. Helen-on-the-Walls, York, represent samples of three late Medieval populations from North Yorkshire, England.

Excavated between 1950 and 1993, they comprise an image of a sample of the living population inhabiting these areas during their period of use (Dawes & Magilton, 1980; Beresford & Hurst, 1990; Boylston *et al.*, forthcoming). These samples were chosen for their close geographical and temporal proximity, whilst being subject in life to varying environmental variables such as levels of population density, standards of accommodation, industrial activity, open areas, amounts and methods of waste disposal and so on, which may be termed rural and urban. In addition, all are pre-industrial sites, offering a picture of the underlying rate of birth defects in humans before the Industrial Revolution exposed the population of England to large-scale industry and much of its related pollution.

Bearing in mind the limitations of cemetery analysis, for example inherent bias due to selection of individuals for burial, preservation issues, excavation strategies, and time scale of the sample (Waldron, 1994), this study seeks to investigate the prevalence of certain congenital defects of the skeleton as observed in the recovered

material from these cemeteries. The study will develop the ideas already explored in a completed analysis of skeletal remains from two other cemeteries (Sture, 1997). These were an early medieval rural site from Raunds Furnells, Northamptonshire (Boddington, 1996), and the later Medieval urban site of the Hospital of St. James and St. Mary Magdalene, Chichester, West Sussex (Lee & Magilton, 1989). Significant differences in defect frequencies and types emerged from that study, and the current project seeks to enlarge the sample for greater statistical significance and to enable comparison between more sites.

### **1.8 LINKING THE BIOCULTURAL, MEDICAL AND EPIDEMIOLOGICAL APPROACHES**

It is now widely recognised that there are many causes of congenital abnormalities. These are accepted to include among others: genetic disposition, maternal nutrition, infectious diseases suffered by pregnant women, and radiation and poisons (Beck *et al.*, 1985; Eskes, 1992; O'Shea, 1995). In essence, the recognition of the *multi-factorial* aetiology of many birth defects is at the same time both a help and a hindrance in their study. A help, in that it may be possible to allay the fears of the public by dismissing many widely-held fears about unavoidable toxic effects associated with powerlines, nuclear power stations and so on. A hindrance, meaning that their multi-factorial nature can almost encourage a delay in research as the origins of many defects are perceived to be extremely obscure or difficult to identify.

This study aims to respond to the entreaty of Smithells (1980: 85), in which he exhorted researchers to focus on contributing to the understanding of human malformations. The purposes of epidemiological investigation include “the provision of data necessary for the planning and evaluating of health care”, and the

“identification of determinants of disease so as to enable prevention” (Barker & Hall, 1991:1). The author believes that an archaeological approach will offer a new perspective on this, the “biggest challenge to medical science” (according to Smithells, 1980:85), and that archaeologically-derived human skeletal material has great potential for improving our understanding of birth defects and their origins.

## **1.9 SUMMARY**

The thesis consists of:

- A review of the medical and archaeological literature on the relevant birth defects
- An outline of Medieval urban and rural lifestyles, with emphasis on the health of women (as the carriers of the developing foetus), to put the archaeological material into a historical and social context
- A review of epidemiological research methods, which may be incorporated into archaeologically-based research into birth defect frequency
- A review of medical issues which are implicated in birth defects, including nutrition, immunity and teratology
- An explanation of the materials and methodology used in the study
- The results
- Discussion and conclusions
- Appendices covering medical terminology and embryology.

## CHAPTER TWO

### Literature review

#### 2.0 BIRTH DEFECTS IN THE MEDICAL LITERATURE

This section principally covers the medical, not the archaeological, literature (see section 2.3). The basics of embryological organ-development are covered in the first section, followed by an explanation of relevant terminology, and a review of the recent literature (1960 onwards) covering the environmental issues in birth defect research and relevant conditions observed in this study.

##### 2.0.i Organogenesis And Critical Periods

Insults to the foetus in the first two weeks of gestation appear to be limited in their effects - that is, they either prevent implantation of the blastocyst in the uterus, or result in spontaneous abortion of the implanted embryo more or less immediately (Moore, 1978:55). It is estimated that a significant proportion of pregnancies end in this manner due to a variety of "natural" causes (Larsen, 1993:22). However, during this earliest phase of gestation, teratogenic agents have the potential to interfere with normal mitotic cell division and produce chromosomal abnormalities which may then lead to birth outcomes which are compatible with life, for example, Down's Syndrome (Moore, 1978). The interval of greatest danger for the foetus (in terms of risk of malformation) is during the period of *organogenesis*, in which the various organs of the body are formed. This lasts from the 15th to the 60th day of gestation, and encompasses several weeks in which the mother may be unaware of the pregnancy. Stillbirths may result from defects initiated during this period, but not all

defects are lethal, and every year thousands of infants are born exhibiting a range from major to minor morphological defects.

## **2.0.ii Defining Birth Defects**

An understanding of past conceptions of disability and cultural agreements on “normalness” is difficult to achieve in the absence of written records which characterises prehistory. The cultural concepts of “defects” and abnormality appear to have been assigned to malformed individuals in the West by at least the first century AD, when Pliny the Elder referred to the transmission of defects between generations (Warkany, 1983:25). By the fifteenth century AD Leonardo da Vinci and others were attempting to understand congenital abnormalities, and by 1651 William Harvey had realised that defective development of the embryo led to the birth of malformed infants. Archaeological representations of malformations date to much earlier periods than this (see section 2.2) but do not, of course, imply any sense of cultural disapproval or rejection.

The World Health Report 1997 (World Health Organisation, 1997:53) defined a birth defect as “any structural, functional or biochemical abnormality present at birth, whether detected at that time or not, and includes the overlapping categories of genetic disorders and birth abnormalities”. The report also noted that birth defects are becoming an increasing cause of death and morbidity in children, as well as of disability, and added: “Despite their significance, however, little progress has been made in determining their cause: the causes of about 70% are still unknown.” (WHO, 1997:54). Maternal diabetes, multiple sclerosis and epilepsy were identified as high-risk defect-producers (WHO, 1997:54). The situation is further confused by

the continuum-like nature of birth defects. When is a condition a defect, and when is it an extreme of normal development? Brothwell and Powers referred to a “no man’s land” from so-called normal variation through minor to major abnormality (Brothwell & Powers, 1968:173). Major defects are defined as those that are “either lethal or significantly affect the individual’s function or appearance” (Levene & Tudehope, 1993:192). A similar definition is of a “structural or functional fault that originates before birth and seriously interferes with the subsequent everyday life of the affected child” (Dryden, 1978:113).

Despite the definitions available in the medical literature, there appears to be confusion in the palaeopathological literature as to the definitions of birth defects. Some authorities refer to “abnormalities”, some to “defects”, and some to “malformations”. This study will refer to “birth defects”, and, following the medical conventions (Levene, 1991; Levene & Tudehope, 1993), will adhere to the following specific definitions where appropriate:

*Malformations* result from some disturbance in the growth of the embryo, i.e. at the earliest stages of foetal life, when the most fundamental formation of the body is taking place. Defects of the neural tube and many anomalies of the vertebral column and skull fall into this category.

*Deformations* result from late changes in previously normally formed structures. Some destructive pathological process or intrauterine force acts on the foetus and produces an abnormality. Defects such as club foot (*talipes*) come into this category,



as the foot itself has all of its elements in place, but lack of space in the uterus forces it into an abnormal position, which disturbs its further growth.

*Disruptions* are essentially the same as deformations, with some authorities preferring to use this term instead.

### **2.0.iii The Multi-Factorial Nature Of Defects**

Explanations for the majority of birth defects are still lacking, despite the progress made in recent decades by medical researchers. The very diversity of defects makes the quest for a cause even more complex. Defects may be macro- or microscopic, easily visible or hidden away only to emerge in later life, and the actual classification of a phenomenon as a defect is not always straightforward. What some regard as a defect may be simply normal variation to others. Genetically-mediated defects may be passed on to the foetus from the parents' chromosomal makeup, or may be induced as a result of some environmental factor acting on the chemical or structural constitution of genes or chromosomes in the foetus, leading to mutations in the affected or subsequent generations (Moore, 1978: 63). Environmental causes may include any factor that acts on the foetus while it is *in utero*, leading to a departure from normal development and growth. Research has revealed a number of environmental agents as being *teratogenic*, that is, harmful to the foetus (see Chapter 4). The current list of recognised agents includes examples from several source-groups, including drugs, radiation, food additives, pesticides, industrial chemicals, infectious diseases and certain maternal medical conditions. Nishimura (1983), in a review of the problems in teratology, believed that new teratogens will continue to be discovered in the future, although these would be few. In the same review it was

suggested that research would be most usefully directed at specific target groups such as chemical industry workers, rather than at the population in general (Nishimura, 1983:11). Despite the difficulties in teratology however, most authorities are agreed that the majority of birth defects are most likely the result of a *combination* of genetic and environmental factors, interacting in some complex manner which is yet to be identified or understood. This is known as a *multifactorial* causation.

Levene and Tudehope (1993:194) stated that 20% of defects were of genetic origin alone, with environmental causes including maternal infection responsible for 2-3%, drugs and chemicals 2-3% and maternal metabolic disease 1-2% of defects. The remainder of defects are assumed to be multifactorial, that is, due to an unquantified combination of both genetic and environmental causes.

#### **2.0.iv Assessing Birth Defect Rates**

Assessment of the incidence of birth defects in modern populations is not simple.

Figures depend on what constitutes an abnormality. Turkel (1989:110) states:

“resolution of the problem is not yet possible because hard data on variations within both normal and malformed populations are not available for most characteristics”.

It is estimated that about 2% of babies have a major birth defect, but when minor defects are added, the figure rises to 7% (Carter, 1971; Levene & Tudehope, 1993).

The incidence is highest in preterm babies (born before 37 weeks gestation), and in babies that are “small for gestational age” (SGA), including low birth weight (LBW), which is linked to maternal health and nutrition (Levene & Tudehope, 1993).

Accurate data collection is difficult because of the diversity of the defects and some are not detected due to early death; in addition some countries do not record defects

in aborted fetuses. Many countries only record those abnormalities that are recognised at birth, thus “missing” the many conditions which are later diagnosed by approximately 12 months of age.

In developed countries, birth defects are the second largest cause of death in children after perinatal factors, and in children 1- 4 years of age, after accidents (Levene & Tudehope, 1993). Within these parameters, there are geographical variational trends. For example, in the U.K., the commonest overall defects are those affecting the central nervous system (CNS) and the cardio-vascular system (CVS), but there are further regional variations such as the low rates of cleft palate alongside high rates of *spina bifida* in Wales (Levene & Tudehope, 1993:193).

In addition, it must be considered that it is only possible to account for a fraction of all conceived individuals with a defect. It is known that many chromosomal abnormalities result in spontaneous abortion or abnormal development of the foetus, which may or may not survive to birth. It has been estimated that among all conceptions by healthy women, up to 50-60% abort spontaneously before conception has been recognised, and that a further 10-20% abort later in pregnancy as a result of various abnormal developmental events (Larsen, 1993:22). Among spontaneous abortion cases where the foetus has been available for examination, 40-50% have been shown to have chromosomal abnormalities (Larsen, 1993:22). The observable percentage of affected individuals in any population is therefore a small fraction of the true numbers of affected individuals conceived. Of course, many chromosomal abnormalities are compatible with life, for example, the Down's, Turner's, Klinefelter's and Prader-Willi syndromes. Cultural ideals then “take over”, to some

extent, in the long-term survival of affected individuals, even in the medical possibilities for their health welfare. Down's Syndrome individuals are, for example, often denied transplant surgery due to their perceived shorter life-span.

Reports on the trends in birth defect incidence, or annual national reports describing birth defect incidence, tend, understandably, to focus on those birth defects which are the most obvious, and which require medical or surgical intervention to ensure the child's quality of life or survival. Thus, conditions such as the neural tube defects, birth heart disease, club foot, cleft defects and birth hip dislocation appear to be well-documented in terms of annual incidence and journal reports on interesting cases. Unfortunately for the biological anthropologist, those anomalies affecting the skeleton which do not directly lead to medical referral, are usually absent from such reports, or in the case of journal articles, are mentioned solely in passing when found in conjunction with a more serious condition. This makes the biological anthropologist's search for associated soft-tissue conditions somewhat haphazard. Nevertheless, certain skeletal anomalies, such as border shifting in transitional vertebrae, do appear occasionally in the medical literature (e.g. Keim, 1980), and such articles have been of considerable use in this study (for example, see below).

#### **2.0.v Studies In Environmental Agents And Birth Defects**

This study compares and contrasts urban and rural populations, with a view to investigating the possible role of population density and its health effects on the development of skeletal malformations. Infection is suspected to play a significant role in the observed frequencies of defects, by its action on either the mother or the foetus, or both. Maternal infection, mal- or undernutrition, or exposure to

teratogenic agents in everyday life may together, or singly, be responsible for the higher rates of birth defects seen in urban populations. Unfortunately, modern epidemiological studies often produce apparently contradictory results. For example, Saxen (1983) published a twenty-year study of the aetiology of congenital defects in Finland. The results suggested that influenza, or any feverish condition, may be instrumental in the development of birth defects, especially those of the central nervous system (1983:90). In the modern context, this variable is confounded by the associated consumption of medication, but in the past, feverish conditions would have had to run their course uninterrupted, and may thus have had a teratogenic effect on the foetus. Saxen concluded that “infectious diseases” especially viral conditions, were hazardous to the foetus (1983:95). Mothers who had suffered influenza at some point (unspecified, but presumably during organogenesis) in pregnancy had almost twice the risk of the control group. However, Knox and Lancashire (1991) in their study of congenital malformations in Birmingham, England, found no evidence of specific association between cyclical rises in influenza (taken from notified deaths due to influenza in England and Wales during the time of the study) and rates of birth defects. They effectively dismissed the postulated association with influenza (1991:183). Neither were any correlations with specific birth defects found for measles, whooping cough or infective hepatitis (1991:182-3) However, Knox and Lancashire did not address the number of spontaneous abortions in the post-influenza periods or at other periods before mothers were aware of pregnancy. Nor did they record individual cases of pregnant women suffering from influenza and producing infants with defects. They simply looked at the overall population rates for death by influenza and compared them with general defect rates. Whilst spontaneous abortion would be a “natural” way for the mother’s body to eliminate affected embryos or

foetuses, awareness of its frequency following infectious episodes would be a useful indicator of the degree and possible origin of the effect. Thus, Knox and Lancashire were probably premature in their dismissal of influenza as causes of birth defects.

Knox and Lancashire did, however, look for correlations between birth defects and several other specific viral agents. *Hydrocephalus* in offspring is known to be induced by the introduction of a variety of viruses into pregnant animals, and human cases have been reported involving lymphatic chorio-meningitis transmitted from mice and hamsters (Knox & Lancashire, 1991:184). They found a correlation between *hydrocephalus* and Coxsackie-A virus in Birmingham between 1972 and 1984 and even when adjusted for certain inaccuracies, found that there was “probably a specific association” (1991:185). Unfortunately, Knox and Lancashire did not record skeletal anomalies found in association with the *hydrocephalus* cases, so vertebral dysplasias cannot be related to the condition from this study. They did, however, suggest a tentative link between Coxsackie-A9 and atrial septal defect, anophthalmos and biliary atresia. Biliary atresia would have been incompatible with life in the past, as would atrial septal defect if severe enough; some individuals could have survived possibly into early adulthood if cared for, however. Syndactyly was found to be related possibly to Coxsackie-B virus, but a significant correlation was noted between Coxsackie-B and cleft palate. The authors noted that “the correspondences [between the virus isolation periods and the prevalence peaks of cleft palate] are sufficiently striking to suggest that the majority of cleft palates [observed here] are probably determined by Coxsackie-B4, and that the characteristic consequence of an embryonic Coxsackie-B4 infection is a cleft palate” (1991:186-7).

Knox and Lancashire also noted that seasonal and other temporal variations in neural tube defects (these were in decline in Birmingham since 1973) could not be adequately explained. They suggested that an infectious process may be involved, as there was a visual association in their results between neural tube defect rates and isolations of Echovirus-9, another Coxsackie enterovirus. The major decline in NTD cases between 1973 and 1984 was “associated with a similar decline in the number of virus isolations” and the cyclical variations of virus isolations and following variations in NTD cases during this period corresponded well (1991:188). Hepatitis B and Hepatitis C are also “probable” teratogenic agents, and mumps and flu “possibles” (Levene & Tudehope, 1993:199).

Neonatal development is known to be seriously affected by a variety of viral organisms (Moreland *et al.*, 1979). Defects related to the influenza virus have been recognised since 1947, largely as a result of the epidemics in Europe in the early twentieth century. The influenza virus enters by the respiratory tract and *hydrocephalus*, nervous system abnormalities and wasting of the foetus were noted originally (Moreland *et al.*, 1979:53,61). The mumps virus was noted in 1966 to be associated with raised foetal mortality if exposure occurred in the first trimester, and was also associated with *hydrocephalus*. As this virus passes across the placenta, the foetal tissues may be invaded by the organism, thus explaining the rise in foetal mortality (Moreland *et al.*, 1979:63). The Western Equine Encephalitis virus has been associated with *hydrocephalus* also, even though the adult infection is often sub-clinical (the disease is present but shows no signs or symptoms).

Maternal fever has been associated with an increased rate of neural tube, cleft and chromosomal defects. In a U.S. study, febrile illnesses in the first trimester produced increased *spina bifida*, Down's Syndrome, cleft palate and anencephaly (Layde *et al.*, 1980). They found that the incidence of congenital malformations is known to rise following influenza epidemics, and the central nervous system is apparently the most vulnerable (Layde *et al.*, 1980:106). The authors considered maternal fever to be "a fairly typical teratogen", suggesting that if it is genuinely a neural tube teratogen, it is likely to act at or around the time of closure of the neuropores at 23-25 days gestation (Layde *et al.*, 1980:106).

Kalter and Warkany (1983) suggested that maternal illness, including diabetes, phenylketonuria, vaginal bleeding and hyperthermia, is frequently associated with congenital malformations. Khera later found that maternal toxicity was associated in mice with exencephaly, hemi-vertebrae, fused arches or centra of lumbar and/or thoracic vertebrae, and fused, missing or supernumerary ribs (Khera, 1984).

Increased maternal toxicity resulted in increased incidence and severity of the defects. Khera thus suggested that maternal toxicity is an aetiological factor for foetal defects. Maternal conditions in pregnancy including diabetes mellitus, rubella, cytomegaly, toxoplasmosis, hypothermia, herpes simplex, varicella (chicken pox), parvo virus (a canine disease) and treponemal disease are also associated with some common vertebral and limb malformations such as mild leg shortening, caudal regression, sacral agenesis, as well as more serious effects.

Diabetic mothers commonly produce over-large babies if the pregnancy is not closely monitored. The risk of congenital malformations, stillbirths and spontaneous abortions among diabetic women is also much higher than that in non-



diabetic mothers. The risks of congenital malformations have been assessed as three to four times greater in some studies, with incidences of up to 80% in diabetics with longstanding disease (Sadler, 1980). These high-risk defects include cardiac, skeletal and central nervous system anomalies (unspecified). The teratogenic mechanisms of diabetic serum (high blood glucose content in untreated diabetic mothers) are still not well understood. Earlier work has suggested that hyperglycaemia may be teratogenic in mice and rats (Sadler, 1980:345).

#### **2.0.vi Environmental Factors And Birth Defects In The Past**

In light of the above considerations, it is easy to appreciate the risks of maternal and foetal exposure to pathogens in the past, when public health was not a recognised issue and effective medical treatment was not available. Golden outlined environmental factors associated with poverty today that could have been an accurate description of Medieval living standards. These included: illiteracy, inadequate sanitation, poor personal hygiene, insufficient access to medical services, poor agricultural practices, overpopulation and inefficient and inappropriate use of resources (1993:446). In addition, adolescent pregnancy, maternal infection, pre-eclamptic toxemia and so on (Golden,1993:446) must all have taken their toll on the reproductive health of Medieval populations, leading to low birthweight in those who survived to birth, and probably to an increased risk of birth defects. Children with birth defects are already identified as being at high risk of malnutrition in modern populations (Golden, 1993: 447), leading one to suspect that the same discrimination may have also occurred in the past, thus further disadvantaging those affected.

### **2.0.vii Maternal Nutritional/ Immunocompetent Status And Defect Rates**

The association between adequate nutrition and effective immune response is well-recognised. Healthy nutrition preserves a complementary relationship with the immune system by maintaining a balance between the absorption and use of required nutrients and the effective elimination of pathogenic threats to the body (Reeves & Todd, 1996). Chandra (1978, 1979, 1980) states that nutritional deficiencies increase the frequency and severity of infections, and that “nutrition appears to be a critical determinant of susceptibility to infection” (1980:76-77). In addition, certain nutritional imbalances alone are associated with birth defect occurrence. For example, Vitamin A toxicity has been linked to spontaneous abortion, cleft palate, severe ear malformations, and defects of the heart and brain. The association between folate deficiency and neural tube defects has already been mentioned.

Alcohol taken to excess during pregnancy is associated with birth defects. Offspring of an alcohol-abusing mother, an adult brother and sister observed by Lowry both had cleft palate, short, webbed necks, and mild conductive hearing loss. Mental retardation, short stature, and significant vertebral anomalies (including Klippel-Feil syndrome) were also present. Lowry commented that it is “possible that skeletal defects, particularly those of the vertebrae, are more frequent [in children of alcohol abusers] than previously recognised” (Lowry, 1977:56). Yasuda and Poland (1974) reported on a seventeen-year-old mother with a history of alcohol abuse who spontaneously aborted a sixteen-week foetus. Defects predominantly affected the left side of the foetus, and included left foot adduction, left thumb absent, left great toe duplicated, aortic abnormalities, a small ventricular septal defect, only one lobe in the left lung, and kidney and ovary anomalies. Klippel-Feil syndrome was present, with

other cervical vertebral anomalies, and there were fused ribs also. Although this was a severe case incompatible with life, it is feasible that an individual with a smaller number of these defects could survive.

Wood has recently addressed the debate on “the impact of maternal nutritional status on reproduction” (1994:522), commenting that different conceptual and methodological approaches adopted by physiologists and demographers produce differing opinions on the importance of maternal nutrition status in reproductive ability. However, Wood’s work continued to focus on the role of maternal nutrition as a factor in regulating *fertility*, rather than considering the possibilities of impaired *reproductive outcome*. Neither was any comment made on the possible role of nutritional status on spermatogenesis. Hogue mentioned dietary deficiencies or excesses as “common exposures” during pregnancy and suggested that pregnancy-related metabolic interactions with drugs and environmental chemical exposures could also result in birth defects (1984:47). Reduced stature was considered, for a while during the 1970s and early 80s, as an *adaptive* reaction. Children who suffered extreme undernutrition in childhood grew to be shorter. The suggestion was that smaller individuals could survive better under times of stress because they needed fewer calories (Wood, 1994:528). Studies in the 1980s then showed that nutritional stunting is in fact associated with increased mortality in later life (Kielmann & McCord 1978; Chen *et al.*, 1979; Heywood, 1982) and is therefore not considered a successful adaptation, at least in terms of longevity.

Frisancho *et al.* (1977) investigated urban Peruvian mothers and infants for any relationship between maternal nutritional status and infant health. They found that an

increase in maternal calorie reserves (fat) led to an increase in infant fatness. An increase in maternal protein reserves (muscle) enhanced both birth weight and prenatal linear growth. As low birthweight is associated with an increased frequency of birth defects (Allen, 1986; Levene & Tudehope, 1993), it is possible that there may be some unquantified association here. The authors noted that in populations suffering chronic malnutrition this mechanism may not apply. In such cases they suggest that an increase in calories alone may result in significant prenatal growth (1977:273). Whether this applies to chronic undernutrition due to infection, malabsorption and inadequate nutrient uptake as is often the case in poor urban environments, is unknown. This may be applicable to the urban Medieval populations examined in this study. Any statistically significant decrease in urban stature compared to rural stature may perhaps be explained by this.

Maternal size is known to have a constraining effect on the size of the foetus (Barker, 1994:122). It is to be expected, therefore, that the majority of smaller women will tend to produce smaller offspring than their larger sisters. Previous work has also shown that the birth weight of mothers is related to their offspring's birth weight, and to their grandchildren's birth weight (Barker, 1994:122). Dutch women who were *in utero* (first and second trimesters) during the Netherlands famine of 1944-5 were found to have normal birth weight themselves, but their offspring had reduced mean birth weight (Barker, 1994:123). This intergenerational aspect of development supports the hypothesis suggested by Emanuel and Sever, who stated that a combination of genetic, immediate and intergenerational factors affected birth outcome (Emanuel and Sever, 1974b:329). This effect seems to be passed on through the maternal line, as babies related only through the father do not exhibit this birth

weight similarity (Barker, 1994:122). This “maternal constraint” effect is believed to illustrate the limitations of the mother in passing nutrients on to the foetus (Barker, 1994:123). It seems fair, therefore, to expect such nutritional limitations to also be reflected in other adverse effects on foetal growth and development, such as birth defects. We know that the principal adaptive mechanism of the foetus to undernutrition is to slow its growth rate (Barker, 1994). In order to accomplish this slowing down of growth the foetus lowers its metabolic rate by reducing the use of substrates (substances which are acted upon by enzymes) (Barker, 1994:123).

Reduced maternal nutrient intake results in a reduction of amino acids and glucose being transferred to the foetus (Barker, 1994:133). This slowing of growth in late gestation is known to affect kidney development; what the exact effects are of such stress in the earlier stages of gestation is not well understood, but the author would suggest that it may be expected to involve a form of teratogenesis in some individuals. We do know that undernourishment in the early stages results in proportionately small babies at time of birth (Barker, 1994:131), so perhaps there is a case for expecting more morphological anomalies in such cases. What is clear is that the size (and indirectly, the health) of a foetus depends not only on the environment in its mother’s uterus, but on the foetal environment experienced by the mother herself and that of the grandmother as well (Barker, 1994:124). Barker suggested that this effect may be an adaptation to protect a population against periods of famine (1994:124). In the poor conditions of Medieval urban environments this may help to explain the higher urban rates of defects over long periods than are seen in rural environments. If urban mothers were themselves subject to intrauterine nutritional stresses, then their own reproductive ability may be compromised. When this factor

is added to the environmental insults of increased infection and further undernutrition, the rates of morphological anomalies may be expected to stay high.

Studies in which pregnant women were given dietary supplements have had mixed results. Trials which involved additional calories as high protein density resulted in a drop in mean birth weights, but those in which protein was a low percentage of the extra calories resulted in an increase in mean birth weight (Barker, 1994:127). As well as birth weight, other foetal measurements are recognised as being associated with the health of individuals. Proportionate size of body parts and organs has been associated with specific health problems in later life (Barker, 1994:128). Barker noted that restricted blood supply to the foetus *in utero* is associated with reduced body length, but that *the head and the brain are spared* (1994:128). This suggests that the head and brain are spared at the expense of other parts of the body. The reduction of amino acids and glucose being transferred to the foetus (the result of reduced maternal nutrition) appears to concentrate the foetus's growth on the head and the brain. Could it be that some malformations are the result of this sparing of the head and brain? Are morphological malformations such as vertebral segmentation anomalies induced in some way by the loss of amino acids and/or glucose at critical periods? Perhaps it is simply the lesser of two evils to have a vertebral anomaly than a compromised brain, and this is the "price" that the body is prepared to pay in order to maintain adequate brain and head development. There is, of course, the issue of cranio-facial anomalies, although it may be that some of these are governed more by genetic than environmental factors. Alternatively, a facial anomaly still spares the brain, and does not necessarily have a reducing effect on *the size* of the head, therefore still allowing space for normal development of the brain.

Placental insufficiency or enlargement may of course be a cause of poor foetal nutrition. Enlargement of the placenta is an adaptation to reduced nutrient supply from the mother, and is associated in later life with cardiovascular disease and hypertension amongst other diseases (Barker, 1994:129). Any woman suffering from these conditions may, of course, herself be placing extra stress on her offspring *in utero*, and may be abetting the development of anomalies.

### **2.0.viii Immunogenetics**

Environment is commonly held to be among the pre-eminent factors in susceptibility or resistance to infection. However, it has been shown that a genetic component is also at work in the immune response. Hurley (1969) and Hurley and Bell (1974) suggested that the interaction between human nutrition and genetics may be a source of birth defects. Studies showed that the dietary level of specific nutrients could affect the expression of certain genes, thus affecting the incidence of particular defects (1974:205). Hill (1998) reviewed the work to date on immunogenetics, that is, the relationships which are apparent between certain infectious diseases and allele variability (alleles are variations of a particular gene) among certain genes. He states that: "...host genetic factors are major determinants of susceptibility to infectious diseases in humans..." (1998:593). The Human Leucocyte Antigen (HLA) system is a series of four gene-families that code for the polymorphic proteins which are expressed on the surface of most nucleated cells (Hill, 1998; Reeves & Todd, 1996). Variations in the genetic make-up the HLA system have been associated with both susceptibility and resistance to several diseases including malaria, tuberculosis, AIDS, leprosy and the hepatitis virus (Hill, 1998:593-4). In addition, the active form

of Vitamin D is also known to have an immunomodulatory effect. Alleles of the Vitamin D receptor have been associated with “differential susceptibility to several infectious diseases” (Hill 1998:593). Genetic effects in regard to infectious disease have been illustrated by studies of twins and of adopted children in northern European populations (Hill, 1998). Adoptees with a biological parent who died from infectious disease before the age of fifty were found to have an increase in relative risk of 5.8 of dying from infectious disease themselves. The death of an adoptive parent from infectious disease had no significant effect on the child’s life outcome (Hill, 1998:595). A strong genetic effect on the risk of death from cardiovascular disease was also reported, although an environmental relationship was stronger in the case of cancer deaths (Hill, 1998:595). Twins were found to have different susceptibility to tuberculosis and leprosy depending if they were monozygotic (identical) or dizygotic (non-identical). The former had a greater disease concordance than the latter (Hill, 1998:595). The active form of Vitamin D has been shown to impair the growth of *Mycobacterium tuberculosis* in macrophages (white blood cells) (Hill, 1998:602-3). In a study in The Gambia, polymorphism (variation) in Vitamin D receptor genes was associated with pulmonary tuberculosis and hepatitis B infections. Those with the diseases had less receptor polymorphism. In an Indian study, the tt genotype was associated with tuberculous leprosy and the TT genotype with lepromatous leprosy (Hill, 1998:602-3).

Hill states that “...It is likely that susceptibility to most micro-organisms is determined by a large number of polymorphic genes...” (1998:593), and concludes that certain genes are associated with resistance or susceptibility to certain diseases



(1998:609-10). Thus, genetic makeup in a population may also predispose towards differential susceptibility to infection.

### **2.0.ix Occupational And Everyday Exposures**

A study of women in the Netherlands between 1980-1992 (Blatter *et al.*, 1996) showed that women involved in agricultural occupations were more than three times as likely to produce a *spina bifida* baby than those in non-agricultural occupations. No differences could be detected between the cases and the controls in the occurrence of exposure to specific chemicals or physical agents. The relationship between agricultural occupations and the occurrence of *spina bifida* could not be explained, as few of these women used pesticides and no other exposure was noted that could be a causal factor. The conductors of the study did not follow up for interview the mothers with *spina bifida* children whose occupations did not involve chemical or physical exposure. This effectively ruled out those with jobs such as nursing, teaching or secretarial/office work. Unfortunately, by concentrating on exposures to chemicals, the authors missed an opportunity to consider the possible role of infection in the aetiology of *spina bifida*. Teachers, for example, come into close contact with infections via their pupils on a daily basis, which could have a bearing on their reproductive outcomes. Neither did Blatter *et al.* consider the possibility of the condition being caused by exposure to animals and manure, which probably constitute an equal exposure to chemicals. Animal exposure would be strongly associated with Medieval rural life, especially in longhouses where animals and humans shared the same building.

Knoblauch *et al.* (1996) reviewed evidence for the ill-effects of manure on human health in Switzerland. Aside from the increased risk of human injury associated with the presence of large animals, they found that noxious gases produced by the fermentation of manure were hazardous to health. Several toxic gases including hydrogen sulphide and methane were associated with reports of farmers collapsing into unconsciousness. The authors found that the action of agitating or moving of manure increased the release of the gases and their inhalation by the worker. Inhaled in high concentrations, such gases lead to unconsciousness and a resulting toxic pulmonary oedema (secretion of excessive fluid in the air spaces of the lungs). This research has implications for Medieval populations, particularly rural groups who had regular very close contact with domesticated animals. The practice of living in the longhouse for example, even though the animals were kept at one end, was widespread in many parts of England, including at Wharram Percy. The presence of manure would therefore be a permanent feature of the domestic environment, and the enclosed nature of houses would increase the concentration of fumes and risk of toxicity. This must have been exacerbated further at night when many people slept in the house along with the animals, and with the doors closed. "Mucking out" was, in essence, a domestic chore in the context of the longhouse, and as such, may have been carried out by women, but the author is not aware of any direct evidence for this. In winter, manure levels were probably allowed to build up more in an attempt to keep the house warm. Even in rural areas with separate houses for animals and humans, the close contact between them would have involved substantial exposure to manure. In addition, manure may well have been useful as a fuel, and emitted gases from the domestic hearth.

In the absence of public health measures in the past, controlling pollution in the workplace, exposure to toxic substances (fumes in metal working, chemicals in tanning, bacterial and viral infections in butchery and so on) would not only have affected the workers but also the surrounding population in Medieval settlements. This is supported by the study of a Finnish population by Saxen (see above) which showed a significant association between maternal employment in industry and the frequency of CNS (central nervous system) defects such as *spina bifida*, *hydrocephalus* and anencephaly as well as oral clefts. Organic solvents were suggested as a possible causative factor (Saxen, 1983:93).

Lauwers *et al.* (1986) showed that blood lead levels affect the neurological and haemopoietic (blood formation) systems adversely. This in turn compromises organ and gastro-intestinal function, and appears to also adversely affect immunodefence and hormonal secretion. In their Belgian study they found that children living in close proximity to lead smelters had a higher morbidity rate than those in other areas, and suggested that children would ingest more lead by playing in dirt. Coupled with the ability of children to store lead more readily than adults, this could have serious implications for their adult health. They also found no correlation between socio-economic class and blood lead levels, and male and female levels were the same.

Aswathi *et al.* (1996) looked at the significantly raised blood lead concentrations of pregnant women in Indian inner-city slums. They found that there was no association with piped water or paint (both of which may contain lead), and that the association with the Asian eye cosmetic *surma*, which is lead-based, did not show the expected relationship. Some samples of *surma* have been found to contain 86% lead (Kolev *et*

*al.*, 1996:842). When use of *surma* was stopped, blood lead levels rose, a phenomenon which the authors could not explain. They concluded that the factors of poor nutrition, infectious diseases and the other disadvantages of poverty, when added to exposure to excessive lead, threatened the normal development of their children.

Kolev *et al.* (1996) looked at blood lead levels and ethnicity in the UK. They measured the blood lead levels of 779 children between 1980-1994. They found that 26.5% of the Asian children had PbB (blood serum) levels above the acceptable level of 200ug/l, compared to 0.8% of European-origin children. More worryingly, 10.5% of Asian children showed PbB above 500ug/l compared to 0.8% of European-origin children. The authors concluded that there was a significant relationship between the blood lead levels of children and their ethnic origin. These were thought to be influenced by cultural habits and traditions including traditional remedies, cosmetics (*surma*), diet and socio-economic status. In addition, it must be borne in mind that most Asian communities tend to live in inner-city areas which suffer high levels of petrol fumes, although this must now be reduced with the increasing use of lead-free fuel. Following the reduction in the lead content of petrol it will be interesting to note any reductions in blood lead levels among the Asian community. The issue of lead levels may therefore be related to ethnic differences in the aetiology of certain birth defects. High blood lead is associated with anaemia because lead compromises haemoglobin production (Kolev *et al.*, 1996: 842). This in turn, may lead to the problems outlined elsewhere regarding anaemia.

Palmer and Coggan (1997) looked at the effects of metal fumes and by-products on the health of workers in the metal industry. Research has shown that levels of iron present in the body influence bacterial virulence and the element has a role in host defence against infection. They found that manufacturing occupations that involved exposure to metal dust and/or metal fumes were likely to lead to raised levels of iron in the body tissues. Metalworkers such as polishers and fettlers were included in this as well as those involved in the production of ferrous objects such as smiths. Workers involved in the extraction of iron ore were also likely to be affected, due to their exposure during the processing of the raw material involving blasting, crushing and smelting. As well as ferric oxides, sodium and calcium fluorides, sodium and potassium carbonates and magnesium oxides are also associated with metal fumes and dust, which the authors considered possibly dangerous. Considering that free iron in body fluids promotes bacterial growth, the authors concluded that exposed individuals carry an increased risk of infection for at least as long as the raised blood levels persist. They also suggested that there may be a generalized hazard from metal fumes, with exposure to non-ferrous metals and their fumes or dust resulting in compromised macrophage function. Obviously any impairment of macrophage function would impact on the immune response of an affected individual, and possibly lead to reduced immunocompetence. Metal workers in the past, without the benefits of safety knowledge, must have been exposed to significant quantities of toxic substances that may have affected their health. Windblown substances in the surrounding areas may also have affected others not directly involved. Farrow *et al.* (1998) found that low birthweight was associated with mothers working in the metal forming, welding and textile trades, and that their infants had a 148g mean weight difference (lower) to professional mothers' infants in the same study.

Garcia and Fletcher (1998) and Bianchi *et al.* (1997) noted a significant relationship between maternal occupation in the pelt/leather industry and oral clefts in their infants. Anomalies included central nervous system defects, *epispadia* or *hypospadia* (malformations of the urethral opening in the penis) and musculo-skeletal disorders (unspecified) in the offspring of mothers working in close contact with leather. Whether this was related to rawhide and associated infections, or to some aspect of the tanning process (modern solvents may be implicated) is unclear. However, given the nature of rawhide, containing skin, fat, muscle, hair and dirt, all in a state of decay following the death of the animal, a high bacterial, fungal and/or viral presence may be assumed. Medieval tanning practices may thus have posed a significant health risk to those involved in them.

### **2.0.x Warfare-Related Exposures**

The rate of birth defects rose in Vietnam following the use of Agent Orange (a dioxin-based herbicide) by the United States during the Vietnam War of the 1960s and 1970s (Constable & Hatch, 1985). Individuals who were directly affected by Agent Orange during the war went on to produce infants with genetic mutations and environmentally-mediated birth defects, which first raised the question of toxicity. Some induced abnormality of spermatogenesis in males was also reported at an early stage (Constable & Hatch, 1985:231). Vietnamese studies reviewed couples living in areas that had been sprayed with herbicides and those in which the husband had been involved in fighting but the wife had been unexposed. Four groups of outcomes showing increases were identified: miscarriage, stillbirth, hydatidiform mole (a “pregnancy” without an embryo), and congenital defects (Constable & Hatch,

1985:232). Although Constable and Hatch found that the Vietnamese data were often confused and inconsistent between studies, they agreed with the Vietnamese researchers that there was a strong association between exposure to herbicide both before and during pregnancy, and increases in birth defects, miscarriages, stillbirths and hydatidiform moles (1985:239).

Iraqi populations were exposed to depleted uranium and the effects of exploded chemical and biological plants during the Gulf War, and Iraqi doctors have since claimed at least a three-fold rise in congenital defects since (Guardian, 21/12/98). Interestingly, the depleted uranium appears to be suspected as the most likely cause rather than the biological agents freed into the environment by Western bombing. Gulf War Veterans from Western countries are still engaged in conflict with their respective governments over their claims that children conceived since their return from the Gulf have a higher than normal rate of birth defects.

In an unusual case, residents of a Dorset village have successfully lobbied for research into “germ-warfare” experiments undertaken by the British Ministry of Defence in their area during the 1960s and 1970s. These experiments involved the off-shore release of quantities of bacterial organisms into the air, and the monitoring of their spread inland. Often linked with viruses, these bacteria are claimed to have been absorbed into the DNA of residents in the area, producing genetic mutations which resulted in those affected giving birth in later life to children with birth defects (Sunday Times, 9/11/97).

## 2.1 BIRTH DEFECTS OF THE AXIAL SKELETON IN THE MEDICAL LITERATURE

### 2.1.i. Spondylolysis and spondylolisthesis

Spondylolysis is a defect of the spine that involves the separation of most, or all, of the neural arch from the vertebral body. It can affect one or more vertebrae, typically L4-5 (Ortner & Putschar, 1981:357), but may also affect other spinal segments. The body of the vertebra is separated from the inferior facets, usually at the *pars interarticularis* (see fig. 2.1), the area between the superior and inferior facets, and the vertebral body. Spondylolisthesis involves the forward slippage of a vertebra affected by spondylolysis, over the vertebra below it, as a result of the spondylolysis. Being separated from the vertebral body, the inferior facets of the affected vertebra can no longer counteract the forward thrust of body weight from above, and the vertebral body is pushed forward. The lumbar region is the most commonly affected, particularly by spondylolisthesis, as it is the main weight-bearing area of the spine. Pain is likely to have been a major factor in the lives of sufferers in the past, as it is often the only symptom observed clinically today (Grieve, 1981).

Spondylolysis and spondylolisthesis in the lumbar region are seen in approximately 4%-7% of the world population (Burkus, 1990; Ferriter *et al.*, 1984). Lowe *et al.* state that it occurs in 2%-10% of "active young individuals" (1987:582). The defect may occur at higher levels of the spine but this is rare (Ferriter *et al.*, 1984), although some disagree (Downey *et al.*, 1986). The majority of lesions are bilateral, with unilateral spondylolysis affecting less than 1% of the population (Burkus, 1990:555; Porter & Park, 1982:346). In the medical literature, spondylolysis is diagnosed from the presence of a crack or visible line (by radiograph) in the *pars interarticularis*, as well as from the presence of an actual break (Edelson & Nathan, 1986). Clinical



opinion now holds that unilateral spondylolysis is not always due to fractures: Porter and Park (1982:347) found that the condition was often associated with localised underdevelopment or hypoplasia of adjacent structures. They also noted that in their experience, asymmetry of the neural arch was demonstrated by the spinous process deviating away from the side of the lesion, and they considered this to be a form of spondylolysis (1982:347). They believed that the clinical implications of *pars interarticularis* defects “must remain speculative”, and they raised the possibility of torsional damage to the adjacent discs and traction damage to the nerve root (1982:347). Burkus found (1990:558) that impingement of the nerve root is usually associated with bilateral lesions and, of these, 16-17% present with radicular symptoms (inflammation of the nerve root and accompanying pain). The condition is therefore heavily implicated in low back pain syndrome. Porter and Park considered *spina bifida* a significant factor in *pars interarticularis* defects as, alongside spondylolysis, the condition produces a floating fragment (the neural arch) which adds to the hypermobility of the segment (Porter & Park, 1982:347). *Spina bifida* occurred in 11% of patients with spondylolysis in one study (Burkus, 1990:555). It is thought that midline instability caused by the presence of *spina bifida* may weaken the *pars interarticularis* and cause a break (Burkus, 1990:555).

The condition is strongly associated with lower back pain in modern populations, particularly among heavy-manual workers, obese individuals and pregnant women (Jayson, 1992). It is generally believed that there is a genetic basis to spondylolysis, and in addition that physical stress and increasing age play a part in its aetiology on top of this (Newman, 1963; Wiltse 1964; Wiltse *et al.*, 1976; Smith & Micheli, 1995). Most cases present when the subjects are in middle age or later, when

physical work decreases and pain becomes apparent. However, Kornberg (1988:712) says that spondylolisthesis secondary to defects of the arch may also be seen towards the end of the first decade of life. Muscle tone probably keeps the loose element in place as long as physical fitness and activity are maintained, and pain may well begin as the muscular sleeve relaxes. Ericksen (1976, 1978a, 1978b) found that morphological changes occur in the lumbar vertebrae with increasing age. These involve a widening of the vertebral bodies and endplates, associated sometimes with posterior wedging of the body (sloping towards the posterior) which appears twice as commonly in females than in males (Ericksen, 1978a). The L5 vertebra not only broadens with age but also is often affected by the growth of reinforcing bone between osteophytes on the inferior endplate and the pedicle bases (Ericksen, 1978b:249). This may be a factor in nerve root compression (radiculopathy) which is associated with lumbar pain and mobility problems (see below). The changing shape of the lower lumbar vertebrae may have a role in the development of spondylolysis.

Wiltse *et al.* (1976), following on from the work of Newman (1963), classified the two conditions as a result of clinical research. Their classifications of spondylolysis include the following groups: dysplastic (an abnormality in growth), isthmic (weakness sited on the isthmus of the *pars interarticularis*), degenerative, traumatic, and pathological. The modern incidence of the condition is around 3% in Caucasian populations (Merbs, 1989) although racial variation is apparent, with Eskimo populations suffering possibly up to a 50% incidence (Roberts & Manchester, 1995). Grieve (1981) considers most modern clinical cases to be degenerative, with only a minority suffering from the dysplastic and isthmic forms. The dysplastic form is a birth dysplasia of the upper sacrum or the neural arch of the 5th lumbar vertebra. An

affected *pars interarticularis* may not be sufficiently damaged to cause major slippage in adjacent vertebrae. A spondylolisthesis of up to 25% is not usually implicated in any degree of paralysis, as pressure on the *cauda equina* would require a greater slip in order to have a significant effect.

#### **2.1.i.a** *Spondylolysis and spondylolisthesis in the medical literature*

Many reports of spondylolysis in the medical literature feature cases where the lesion affects an unusual site such as the cervical segment, or mention the spondylolysis as incidental to or co-occurring with, some other medical condition. For example, Libson *et al.* (1984) looked at scoliosis risk in young males with or without symptomatic spondylolysis. A selection of more recent reports follows, focussing on reports which show associated medical conditions, associated restrictions in movement, experience of pain, and in some cases, the surgical correction required. These illustrate the variation of symptoms and lifestyle restrictions that may be associated with the condition, many of which are not obvious from skeletal material.

Lowe *et al.* (1987) reported on a retrospective series of 32 patients in Jerusalem over four years, which included patients who had presented with spondylolysis in the upper parts of the lumbar segment. Twenty of these had bilateral defects and of those with unilateral defects, 7 had structural changes or anomalies in the opposite posterior arch. These included *pars* hypertrophy, sclerosis, deformities, and healed *pars* defects (1987:582).

Kornberg (1988) reported on a case of spondylolysis with spondylolisthesis in a thirty-two year old female. A spondylolisthesis was present at L5-S1, with a left-sided L5 *pars* defect (spondylolysis) and a right-sided L5-S1 facet joint with hypertrophy and degeneration. The right L5 *pars* was intact, and the patient also had a *spina bifida* defect, although the author did not state at which level. Surgery corrected the spondylolisthesis.

Edelson and Nathan (1986) reported on a series of 34 patients with isthmic (probably congenital in origin) spondylolysis. They found that 32% had stenosis of the intervertebral foramen (a narrowing of the neural canal containing the spinal cord), and suggested that this was an important factor in the aetiology of nerve root pain associated with the condition (1986:596).

Burkus (1990) reported nerve root compression in three patients with unilateral spondylolysis, associated with *spina bifida occulta*. All three presented with nerve root entrapment at the L5 pedicle defect, causing severe pain.

Hazlett (1984) reviewed 134 lumbar spondylolysis patients with back pain and leg symptoms lasting for at least ten years. He found that backache was due to disc degeneration as well as the “stresses associated with the defect” (1984:298).

Garber and Wright (1986) reported a case of spondylolysis with accompanying spondylolisthesis in a patient with a fractured contralateral pedicle. The patient was a 26 year - old male who presented with severe low back pain. Radiography revealed a “sclerotic” lesion of the L4 right pedicle, and a left-sided spondylolysis of the same

vertebra. The sclerotic lesion was shown on computerised axial tomography scan to be a fracture of the pedicle.

Downey *et al.* (1986) reported on congenitally absent posterior elements of the thoraco-lumbar spine, principally the inferior facets, typically unilateral. They concluded that this was a “normal variant” in the spinal column. In the literature they found less than twenty reported cases in the lumbar and sacral regions, and concluded that the condition was slightly more common in the cervical spine. Ferriter *et al.* (1984), however, pointed out that from their review of the literature, cervical spondylolysis is rare (see below).

Mizutani *et al.* (1989) reported on a ten-year old boy with a congenitally absent lumbar pedicle and transverse process (L1). He presented with low-back pain and was found to also have hypoplasia of the affected vertebra, hypoplasia of the pedicles of T12, L2, L3, and L5, hypoplasia of the inferior articular processes of T12, L1, and L4, and a butterfly vertebra at L5 (1989: 890).

Ferriter *et al.* (1984) reported a case of cervical spondylolisthesis in which the patient suffered upper motor neurone deficits and sensory loss. The subject was 57 year old accountant who had a history of cervical spine problems, including a congenital fusion of the posterior elements of C2 and C3. Following exercise involving prolonged extension of the neck the patient suffered a loss of sensation up to the elbows and a diminution of sensation up to the shoulders. He also exhibited some spasticity (stiffening of the muscles due to impaired innervation) of the right leg and was unable to write or do tasks such as fastening buttons. Surgical intervention

revealed a synovial pseudo-joint between the arch of the atlas and the laminae of C2. There was a bilateral pedicle defect at C2 ( a spondylolysis). This resulted in a spondylolisthesis of C2 over C3. The inferior surface of C1 was compressing the spinal cord causing the symptoms, presumably as a result of the hyper-extension of the neck exacerbating the spondylolysis and spondylolisthesis. Surgical treatment resulted in full recovery. They found only 26 other cases of defects (spondylolysis) of the *pars interarticularis* or pedicles reported, and of these only twenty had corresponding spondylolisthesis. The majority of these affected C6 on C7, with only two previous cases reported at C2/C3.

Hirota *et al.* (1988) reported on a case of cervical spondylolysis resulting in tetraplegia (loss of use of all four limbs). The subject was successfully treated by surgery. They concluded, as had Ferriter *et al* (1984), that the condition was rare.

Hanson *et al.* (1990) reported an infant male with congenital pedicle defects and spondylolisthesis of the axis (C2). Conservative treatment with a collar relieved the symptoms of the spondylolisthesis while the lesion healed as the child grew. His “overall development” was delayed (1990:238).

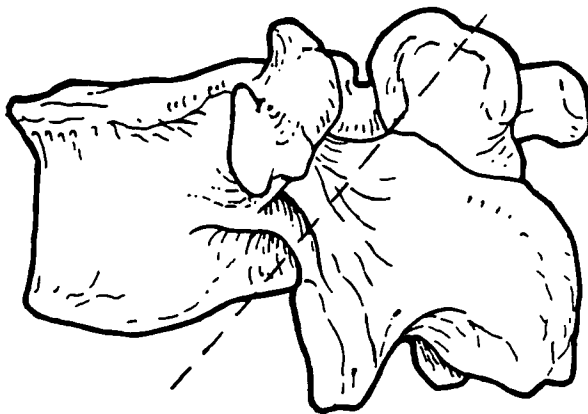
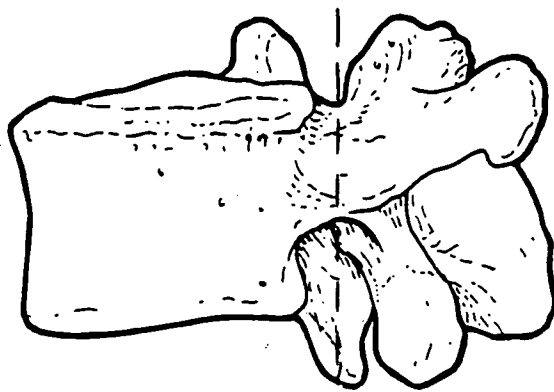


Figure 2.1: Lumbar pedicle defects

Summary of spondylolysis/spondylolisthesis review

- both unilateral and bilateral lesions are associated with symptoms of pain and/or impaired innervation of the back and/or lower extremities
- co-occurring *spina bifida occulta* appears to be associated with increased problems leading to referral to a doctor
- narrowing of the neural canal may be a problem for a significant number of patients, associated with increased pain and probably also reduced movement because of this
- radiculopathy (pain due to nerve root compression) is a frequent symptom in affected individuals which may significantly reduce mobility and lead to adjustments in lifestyle (and possibly lead to depression) as it affects the leg(s) and lower back
- the absence or hypoplasia (under-development) of the inferior vertebral facets and/or pedicles may well be considered a normal variant in the spine but may still be associated with lower back pain
- cervical spondylolysis is probably rare, but is associated with impairment of the nerves supplying the arm(s).
- spondylolysis/spondylolisthesis have the potential to cause problems requiring lifestyle adjustments and some degree of physical impairment



### 2.1.ii. Spina Bifida

This condition involves non-union of the neural arch of at least one vertebra, and often affects two or more vertebrae. The condition is one of the neural tube defects (NTD). As the purpose of the vertebral arch is to form a protective canal for the spinal cord, any impairment in bone development can lead to damage of the cord. Where there is an opening in the neural arch, the spinal cord and/or meninges may herniate out of the canal. This is a potentially life-threatening disorder, and cannot be adequately treated today in the majority of cases (Laurence & Tew, 1971; Avery & Tausch, 1984). There are two suggestions as to the cause. One is that some insult occurs to the embryo that prevents neural closure of the spinal cord and its protection (the meninges), or of the vertebral arches; the other is that the neural tube is ruptured after normal closure because of some agent causing the pressure to rise within it (Gardner, 1965; Avery & Tausch, 1984). Whatever the cause, however, *spina bifida* is always associated with open neural arches, often with double ribs, and occasionally with skull-base defects, especially the posterior arch of the atlas (Avery & Tausch, 1984). As seen in the previous section, it also has some association with spondylolysis.

There are two forms of this birth defect: *spina bifida cystica* (the most severe form) and *spina bifida occulta* (see fig. 2.2). *Spina bifida cystica* can occur with varying degrees of severity, which in antiquity would usually have been incompatible with survival. Barnes (1994:50) describes two possible cases from the New World, although both are subadults, one being six years old and the other dying in the mid-teens. *Spina bifida occulta*, giving greater protection to the spinal cord, is not life-threatening, although there is clinically often some degree of motor function

impairment where there is associated neural involvement. Individuals with this form of the condition were therefore able to survive into adulthood and, in the absence of neural involvement, probably led a normal life. Many individuals would not have been aware of their condition, as is the case today. *Spina bifida occulta* is usually found at the level of the fifth lumbar vertebra and the first sacral element (L5/S1). It is also common at the base of the sacrum, where it is usually asymptomatic (Avery & Tausch, 1984). *Spina bifida cystica* can occur at higher levels and involve two or more lumbar vertebrae, some of the sacral elements, and occasionally, the lower thoracic vertebrae (Avery & Tausch, 1984; Barnes, 1994).

The modern incidence varies geographically. In Japan the incidence of *spina bifida cystica* is 0.2/1000 live births, whereas in Ireland, it is 4.2/1000 live births (Laurence & Tew, 1971). The world average incidence for the *cystica* form is 1:1000 live births (Levene & Tudehope, 1993). There has been no satisfactory explanation for the geographical variation, although genetics and environment are both known to play a part (Smithells, 1980, 1983, 1992). Its modern incidence is higher in towns than in the countryside, it shows seasonal variation (generally highest in late winter/early spring, and lowest in July/August and October/November) and it is clearly related to

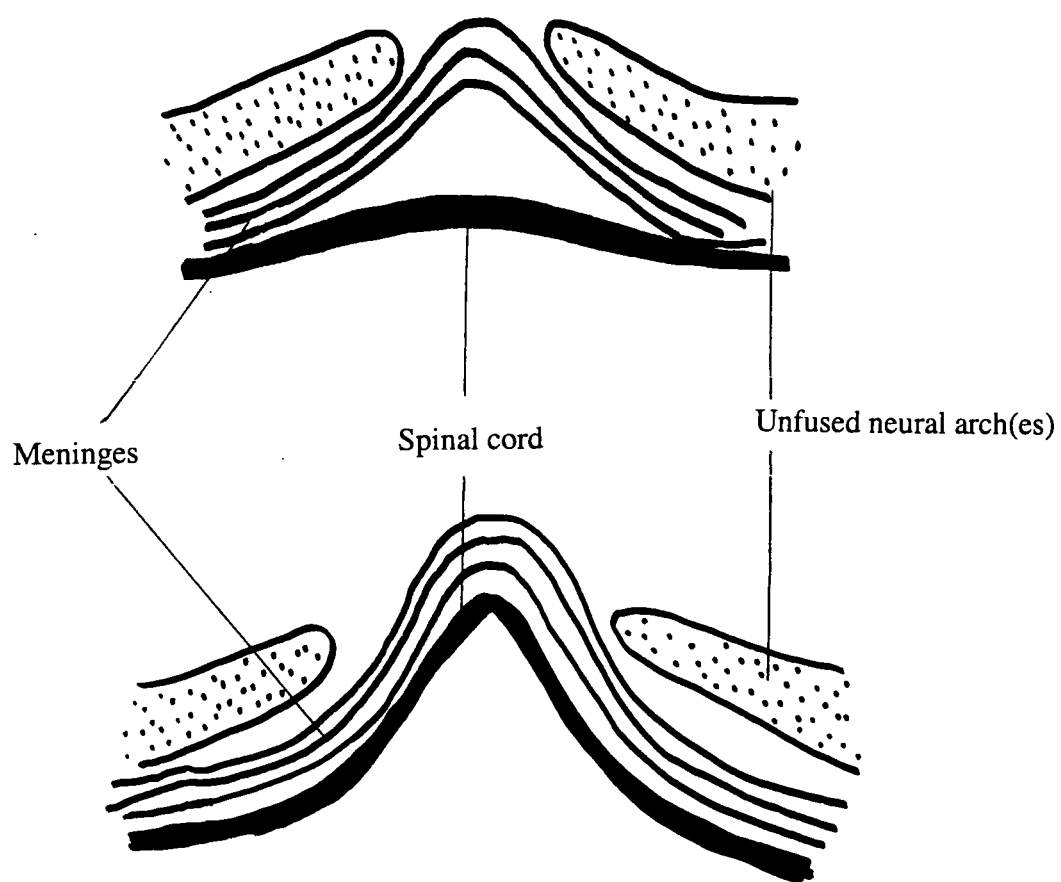


Figure 2.2: Meningocele and myelomeningocele

poor social conditions (Knox & Lancashire, 1991:187). Laurence's major study of central nervous system anomalies in the Welsh valleys (Laurence, 1966; Laurence *et al.*, 1968) showed a similar high rate of *spina bifida cystica* to those recorded in three major cities at the time. Laurence was also able to show that the *occulta* form is not simply a minimal manifestation of the mutant gene that causes the *cystica* form (1967). Laurence also noted a Japanese study that showed that miscarried/aborted foetuses had a higher rate of neural arch anomalies than among neonates, suggesting that a large number spontaneously abort before birth (1967:645). Jorde *et al.* (1987) used the Utah Genealogical Database to study neural tube defect rates in the US. They found that more females than males are affected with NTDs and estimated that there was a 70% risk of heritability.

The average incidence for *spina bifida occulta* is 1:10 worldwide, with degrees of severity varying. The majority of these cases are probably asymptomatic, and the condition is only diagnosed when the individual is examined for some other complaint (Levene & Tudehope, 1993). The commonest challenge in *spina bifida occulta* occurs if the *cauda equina* is tethered to the base of the vertebral column, resulting in neurological dysfunction including bladder and lower limb paralysis by the time the subject reaches the age of about ten years (pers. comm. M. Levene). Without surgical intervention, death follows eventually. This may explain some of the cases of juveniles in archaeological contexts around this age who are observed to have cleft sacrum or less severe *spina bifida occulta* with neural tube involvement (Sture, 1997).

### 2.1.ii.a *Spina bifida cystica*

This form is always characterised by a herniation of the spinal cord and/or its protective layers, the meninges, out of the neural canal, through the open vertebral arch (Gardner, 1965; Laurence & Tew, 1971; Avery & Tausch, 1984). Avery and Tausch consider that of all *spina bifida cystica* cases born among modern populations, 75% are suffering from myelomeningocele (a herniation of the meninges and part of the spinal cord), and 25% from meningocele (a herniation of the meninges only). If populations in the past suffered similarly, then only the 25% with meningocele would have stood a chance of survival into adulthood and, of those, a reduced number may have survived childhood, as the nerve-supply problems associated with the condition, such as the tethered cord, or others less devastating, would not have allowed a good quality, or continuation, of life.

### 2.1.ii.b *Spina bifida occulta*

This form is either so marginal (for instance at the base of the sacrum) that it is asymptomatic, or it occurs when a meningocele (see fig. 2.2) is well covered by a layer of skin and fat (lipoma), affording greater protection for the spinal cord. It is, however, often associated with other spinal defects. The defect may only appear as a dimple in the overlying soft tissue. There can be some motor function deficit, although this may only become apparent after trauma. Even in mild forms, restriction of the cord space due to pressure from fibrolipomatous tissue can result in back, hip and leg pain, so it is not true to say that the condition is asymptomatic in all cases. *Spina bifida occulta*, without meningocele, is, however, usually asymptomatic, and today occurs up to 25% more commonly than the form with meningocele (Barnes, 1994). Clinically, the clefting of vertebrae without neural

involvement is usually seen at the borders between different segments of vertebrae, and often only involves two vertebrae. This form can also produce a cleft sacrum, and originates in the paraxial mesoderm developmental field (Barnes, 1994:49). It is assumed that true neural tube defects are present in cases where the spinal canal is widened, and the bony edges pushed outwards (Barnes, 1994:49, 120). It may be best to adopt the terminology used by Barnes and refer to “cleft neural arch” instead of *spina bifida*, as this does not automatically imply any neural involvement.

Individuals in the past who were affected by true *spina bifida occulta* ( a NTD rather than a neural arch defect) may have been expected to suffer some degree of back pain, and may also have suffered complications of posture, gait, and secondary osteoarthritis resulting from this. Where the *filum terminale* (the end portion of the spinal cord, extending from L1 to S3) is pressured, problems with walking, leg and foot muscle-related problems, and bladder complications, are known clinically. In cases where the *filum terminale* is tethered, problems with nerve supply arise only as the child grows into adolescence and pressure is put on the spinal cord (Grieve, 1981). The condition therefore has potentially quite severe implications for the individual.

### **2.1.ii.c** *Causes of spina bifida*

In 1981 an important connection was made between the occurrence of *spina bifida* and maternal nutrition (Smithells *et al.*, 1981). By assessing maternal nutrition, it was found that folic acid deficiency in mothers was associated with neural tube defects in infants, and that when supplements of folates and other vitamins and minerals were taken by the mother prior to becoming pregnant, the incidence of *spina bifida* fell significantly. Since this discovery, much work has been done to further

understand the links between maternal health and infant *spina bifida* (Smithells, 1983, 1992; Laurence *et al.*, 1983; Eskes, 1992; Poggel & Gunzel, 1992). Recent figures show that the reduction in NTD incidence observed during the 1980s has not continued in the 1990s (Abramsky *et al.*, 1999) despite the widespread publicity about the need to take folic acid if planning a pregnancy. However, a plateau such as this is usually seen in epidemiological studies following advances in preventive care (pers. comm. Michael Smith). Eskes (1992) noted that the condition was associated with genetic predisposition, maternal nutrition, infections, radiation, poisons, and maternal illness during pregnancy. Of these, one of the easiest to address was maternal nutrition. Smithells *et al.* (1981) found that folic acid was the only consistently deficient nutrient amongst women in the developed world, and to give it as a supplement was easy and economical. However, taking adequate amounts of folates is not always the answer, as malabsorption may result from other causes of maternal ill-health. Diarrhoea, for instance (which may have been a permanent feature of life in the past as it often is in developing countries today), and inadequate iron intake, are just two reasons why folate deficiency can occur in women of child-bearing age.

Kurent and Sever (1974), using data collected from pregnancies and live births during 1959-1966 in the United States, considered the relationship between perinatal intrauterine infections and the occurrence of *spina bifida* and anencephaly. They concluded that there was no evidence of perinatal infection playing a “prominent” role in the aetiology of these conditions, although they did not venture a suggestion as to what “non-prominent” could amount to. They did, however, note that previous suggestions of a *single* common environmental factor lying behind *both* conditions

could no longer be sustained unmodified, as racial differences in the frequency of *spina bifida* were similar, but very different for anencephaly, with whites having a rate six times greater than that of blacks (Kurent and Sever, 1974: 360-61). Racial differences in NTD rates are marked; for example Sikh populations have a very low incidence of *spina bifida* (pers. comm. RW. Smithells).

Emanuel and Sever, as well as considering the relationship between neural-tube defects and potatoes (see Chapter 4), suggested that zinc deficiency may be a factor in the aetiology of neural-tube defects and that calcium deficiency may also be involved as a cause of growth disturbances in the mother's earlier life (1973a:117; 1973b:329). Emanuel and Sever were among the first to consider the intergenerational effects of birth defect aetiology. It was their suggestion that an interference with the growth and developmental processes of the mothers of affected children could be a significant aetiological factor in central nervous system defects in the infants (1973b:329).

McDonald *et al.* (1974) suggested that there may be a link between nutritional factors, viral and/or bacterial organisms and the aetiology of neural tube defects. They suggested that latent organisms offered an explanation for the epidemiological behaviour of neural-tube defects, as the already-noted association between Herpes virus type 2 and cancer of the cervix had important features in common with the epidemiology of neural-tube defects. Both conditions show a significant inverse relationship with socioeconomic status, and a low rate amongst Jewish communities. They concluded that an impaired immune response to Herpes virus infection or latency of the virus may therefore be associated with neural-tube defects.



Cases of *spina bifida occulta* are not represented *per se* in the medical literature due to their largely asymptomatic nature. Most details of associated soft-tissue anomalies come from personal communication with physicians or from cases where the condition is mentioned incidentally as part of a larger study (e.g. Castellvi *et al.*, 1984).

Summary of *spina bifida* review

- *spina bifida occulta*, involving the meninges protruding through the bony arch(es) of the vertebrae, is a neural tube defect which originates in the blastemal stage in the neural tube developmental field
- *spina bifida occulta* is compatible with a normal lifestyle, although children with tethered cord syndrome would have had a raised likelihood of early death in the past
- there are marked racial differences in the frequencies of NTD
- there is a significant socioeconomic association with frequency of NTD
- there may be several nutritional components in the frequency of NTD
- there may be a link with maternal infectious processes in the frequency of NTD
- *spina bifida occulta* should not be confused with cleft neural arch, which originates in the paraxial mesoderm developmental field, rather than the neural tube developmental field.

### 2.1.iii Numerical variation of vertebrae

It is not uncommon to find archaeologically-derived skeletons with extra or absent vertebrae (even in skeletons where it is known that the entire vertebral column is present). Supernumerary elements are usually found at the segmental borders, and involve the transitional vertebrae, particularly at the thoraco-lumbar or the lumbo-sacral borders. Cervico-thoracic supernumerary vertebrae are less common.

Variation in the number of vertebrae in modern populations has been shown to be associated with ethnicity and is discussed below. The development of the regional characteristics of the vertebrae (eg. cervical, thoracic, etc.) depends on the successful differentiation of the segments at the blastemal stage (see section on embryology).

Normal segmental-junction vertebrae (C7, T1; T12, L1; L5,S1) take on the characteristics of the adjoining segment, therefore the inferior aspect of C7 looks similar to a thoracic vertebra and the superior aspect of T1 looks more like a cervical vertebra, and so on.

Anomalies in separation and differentiation at the blastemal stage produce confused vertebrae, both in number and in type. Cervical and lumbar ribs can occur (cervical ribs are more common than lumbar ribs), as a result of confusion in the division and new-pairing of the cranial and caudal sclerotome cells. Extra or congenitally absent vertebrae can also result occasionally from an unusual number of somites (Barnes, 1994:78). Extra vertebrae are commonest at the thoraco-lumbar and lumbo-sacral borders. The former usually take on the characteristics of thoracic vertebrae and the latter, those of the sacrum, as the transverse processes are widened into ala-like projections. Sacralised lumbar vertebrae can be united completely to the sacrum, or

in part, in varying degrees. Lumbarisation of sacral vertebrae (S1, usually) is less common.

A study undertaken by De Beer Kaufman (1977) amongst groups of African descent found a significant association between ethnic origin and variation in the number of precoccygeal vertebrae. High rates of extra vertebrae were observed among Southern African and San samples (up to 31 elements), with a slightly lower rate among American negroes (sic.). Caucasoid and Mongoloid samples had a much lower frequency of extra vertebrae (De Beer Kaufman, 1977:411). In the Southern African and American Negroid samples there was a small percentage with absent vertebrae, down to as few as 28 elements. The lower frequency of extra vertebrae among American negroes was explained by the approximately 20% Caucasoid admixture present in this group (1977:413). It was suggested that this pattern of vertebral count variation reflected the descent of the Southern African and San groups from a common proto-negriform stock. In addition, there was sexual dimorphism, with females much more likely to present with absent vertebrae than males (de Beer Kaufman, 1977:411). Weighted male and female means showed that males had a 30.3% rate of extra vertebrae, with females having a 15.6% rate, whereas females had a rate of 3.0% absent vertebrae against males with 1.3% (1977:414). The frequency differences covering counts of between 28 and 30 vertebrae are statistically highly significant.

Numerical variations do not seem to feature in the medical literature as case studies, presumably because they are considered incidental to other anomalies, assuming that there is no significant association with soft-tissue disease.

Summary of numerical variations review

- numerical variations originate in the blastemal stage in the paraxial mesoderm developmental field and are derived from segmentation /differentiation anomalies of the somites
- numerical anomalies appear to be genetically-mediated within racial groups
- there may be an environmental component affecting this predisposition over time
- statistically significant variations have been observed between males and females in the frequency of absent/extra vertebrae
- extra vertebrae may be partially or completely fused to adjacent elements (for example, sacralisation)
- frequency of the condition within a genetically homogenous group may be an indicator of changes in genetic or environmental influence over time.

**2.1.iv Border shifting ( transitional vertebrae)**

This phenomenon is most commonly found at the lumbo-sacral border, although it does occur at higher borders. The anomaly is possibly due to a delay in the formation of the blastemal developmental unit that includes the vertebral segments and interjoined disc space, at the segmental borders. Barnes (1994:79-80) believes that the severity of the anomaly may reflect the amount of delay in the response to some critical threshold event that leads to border demarcation. At the lower end of the vertebral column it involves either *sacralisation*, a partial or complete attachment of the lowest lumbar vertebra to S1, or *lumbarisation*, a partial or complete separation of S1 from S2 (see fig. 2.3), as the upper sacral element undergoes an inappropriate segmentation (Barnes, 1994). Border shifting is described as cranial- or caudal-

shifting in direction. This does not refer to the individual vertebrae, but to the segment in which the numerical variation is incorporated. Thus, the sacralisation of L5 indicates a cranial-shift of the lumbo-sacral border because the sacrum's superior border has moved in a cranial direction, *and shortened the lumbar segment*.

Lumbarisation of S1 indicates a caudal-shift of the border as the sacrum's superior border has moved caudally, *and shortened the sacral segment*.

Transitional vertebrae are associated in the medical literature with herniated discs in the lumbar spine (Castellvi *et al.*, 1984) and as such, may be also related to the appearance of Schmorl's nodes in archaeologically derived materials.

#### **2.1.iv.a** *Transitional vertebrae in the medical literature* (see figs. 2.3)

Castellvi *et al.* (1984) investigated the relationship between herniated lumbar disc and defects of lumbar transverse processes. They referred to "transitional" vertebrae, which by word and illustration (fig. 2.3) were shown to be the same as the partial and complete lumbarisation/sacralisation observed in this study. They outlined the history of the debate on the clinical significance of lumbo-sacral transitional vertebrae since 1917 when Bertolotti first described "assimilation of the fifth lumbar vertebra into the sacrum associated with low-back pain" (cited in Castellvi *et al.*, 1984:493). They reviewed the cases of two hundred subjects with herniated lumbar discs and found that sixty (30%) had related lumbo-sacral anomalies (lumbarisation/sacralisation). They presented a "new classification of lumbo-sacral transitional vertebrae" (1984:493-4) divided into four classes, all of which may be identified in archaeologically-derived bones. These diagnoses were made by lumbo-sacral joint radiography and checked with chest radiographs to count the number of

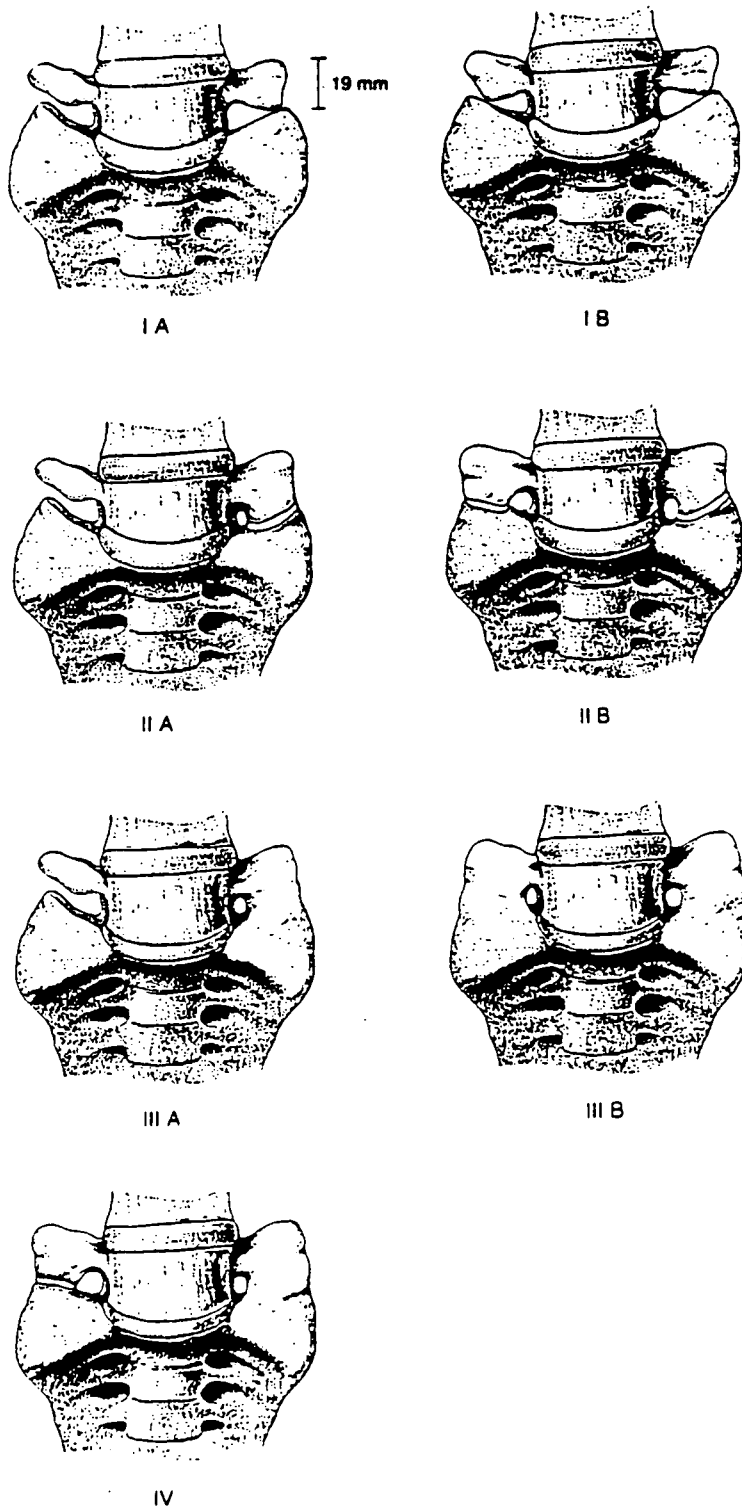


Figure 2.3: Lumbo-sacral anomalies (from Castellvi *et al.*, 1984)

lumbar elements present (1984:493). The sixty patients who were found to have lumbar transverse process anomalies made up 30% of the sample, with males having a 71.5% frequency. Twenty nine patients had a Type I defect or six lumbar vertebrae. Four patients (2%) showed six lumbar vertebrae with twelve thoracic vertebrae, and all of the four showed complete lumbarisation of S1.

Jonsson *et al.* (1989) reviewed a series of patients with “anomalous lumbo-sacral articulations”, and related these to low back pain. They found that of the eleven patients, with a mean age of thirty-nine years, back pain histories were all of less than ten years (1989:834). It was suggested that secondary arthritic changes or a disturbance of bio-mechanical forces at the articulation lead to the onset of pain. They also noted that they had never treated any patients with bilateral defects - all of their patients had unilateral lesions (1989:834). These ranged from articulation of the elongated/altered lumbar transverse process with the sacrum at the *ala*, or with the sacrum and the iliac crest of the pelvis (1989:831-2). Treatment involved surgical resection of the transverse processes and was successful at reducing or eliminating the low-back pain. The L5 nerve root was frequently found to cross just below the transverse processes at the point of resection, involving it in the morphological anomaly. Cailliet found that the L3-L4 nerve roots were irritated by their passage over the pseudoarthrosis between the elongated L5 transverse processes and the iliac crest in her patients with sciatic radiculopathy (1995:361-2). Jonsson *et al.* noted that their patients reported post-operative increased mobility in their backs, probably due to the increased movement at L5-S1. This accords with the findings of Elster (1989:1376) who concluded that there was probably “very limited” movement between L5-S1 in cases of unresected transitional vertebrae.

Summary of border shifting review

- the condition originates in the blastemal stage and is probably due to segmentation/differentiation anomalies, either genetic/environmental/both in origin
- it is associated with generalised back pain and with herniated intervertebral discs (which can lead to severe back and leg pain, and occasionally, nerve impairment)
- secondary osteoarthritis and/or disturbed biomechanical forces also contribute to back pain
- in studies, nerve root compression associated with elongated lumbar transverse processes caused sufficient pain to indicate surgical intervention and these patients all had unilateral defects
- mobility in the lumbo-sacral region was inhibited prior to surgical repair and slightly increased afterwards
- border shifting, particularly unilateral defects involving elongated transverse processes at the lumbo-sacral junction, may be associated with moderate to severe pain due to mechanical forces acting on the nerves leaving the spinal cord, and also with restricted mobility, either due to fusion of the elements, or as an adaptation to pain.



### 2.1.v Block vertebrae and Klippel-Feil Syndrome (see Fig 2.4)

Fused vertebrae develop as a result of segmentation failure during the blastemal stage in the paraxial mesoderm developmental field. The anomaly does not represent *fusion* of two or more vertebrae, but a *developmental union*, as the individual segments never developed separately. The defect originates with the sclerotome pairs, as in other vertebral numerical errors, and is the result of a failure in the development of the sclerotomic fissure which produces the intervertebral disc, thereby separating the adjacent vertebral elements after recombination of the sclerotome halves (see Chapter 3). Cervical and lumbar vertebrae are affected more often than thoracic vertebrae. One of the commonest levels of fusion is at C2-C3. The majority of patients present as young adults and may experience varying degrees of myelopathy (muscle weakness) and spasticity (Epstein *et al.*, 1983: 346). When the thoracic segment of the spine is involved the defect usually shows in the mid-thoracic region. (Barnes,1994). Block vertebrae below the cervical level are usually asymptomatic (Smith & Micheli, 1995).

Congenital fusion of any vertebrae is often referred to as Klippel-Feil syndrome, but the term actually describes the union of two or more *cervical* vertebrae. This type of union can be very different from block vertebrae elsewhere in the spine. An individual who is asymptomatic with two or more cervical vertebrae united is the exception rather than the rule (Pizzutillo, 1983; Smith & Micheli, 1995). When combined with a cervical rib(s), neurological symptoms may be present. The lowest part of the brachial plexus (a group of nerves supplying parts of the shoulder, the arm, forearm and hand) runs across the “rib” causing these neurological anomalies.

Occasionally a fibrous or bony bar splits the spinal cord in the neural canal - a condition known as diastematomyelia (Dandy & Edwards, 1997:433).

Klippel-Feil syndrome is divided into three classes of severity:

*Type I:* a severe defect involving several cervical and thoracic elements united in one grossly abnormal block, usually associated with other major abnormalities.

*Type II:* involvement of only two or three cervical elements, commonly C2 and C3, followed by C5 and C6. There is a known genetic predisposition for this type. This is also applicable to thoracic and lumbar block vertebrae. It is the most common type of Klippel-Feil syndrome.

*Type III:* involves cervical block vertebrae and associated segmentation errors elsewhere in the spine, usually thoracic and lumbar (Pizzutillo, 1983; Barnes, 1994).

Union of vertebrae can also occur as a result of other developmental defects.

Hypoplasia causes the affected centrum to be wedged either dorsally or ventrally, due to delayed development of the chondrification centre during the embryonic stage.

Aplasia also results in a dorsal or ventral malformation, but without any growth of the affected half at all. The absent bone space is filled with intervertebral disc material. The condition is obviously more severe than hypoplasia. Both forms of developmental delay can cause kyphosis (ventro-dorsal curvature of the spine). The adjacent vertebrae develop normally (unless affected themselves), and the spine develops a kyphosis as a result of the wedge-shaped element. This could easily be confused with infectious or degenerative disease, but this can be ruled out by the appearance of the block vertebra (other signs of infection or degeneration would also be seen on the skeleton). The lines of union in a birth anomaly may be visible and, if so, they are smooth and well-coalesced. The bodies are usually a single, complete

unit all round, and the arches may be totally or partially united, with or without visible fusion lines. The key is the absence of signs of inflammatory or reactionary bone growth which is characteristic of disease processes due to infection or trauma (Barnes, 1994).

Elster (1989) suggested that the hypermobility often observed by clinicians in the interspace *above* fused sections of the vertebral column may render those areas more liable to degeneration and disc problems (Elster, 1989:1376). The area immediately above a block vertebrae anomaly may therefore be more prone to these changes and the associated modifications of stenosed (narrowed) neural canal and foramina (Elster, 1989:1376). This could lead to ensuing neurological deficits in areas innervated by the nerves leaving the spinal column at that interspace.

#### **2.1.v.a**     *Klippel-Feil Syndrome and block vertebrae in the medical literature*

Numerous accounts of Klippel-Feil syndrome are reported in the literature, too many to comprehensively review. A selection of these cases and studies is included below.

Hirsch (1982) reported a case of failure of fusion of the left lateral mass of the axis associated with Klippel-Feil syndrome. The patient presented with a neck injury following an accident and radiography revealed the anomaly. The spinous process of the axis was also underdeveloped and there was birth fusion of C2 and C3.

Conservative treatment resulted in a full recovery and no neurological deficit. Hirsch suggested that the problem originated with faulty fusion at the lateral ossification centre.

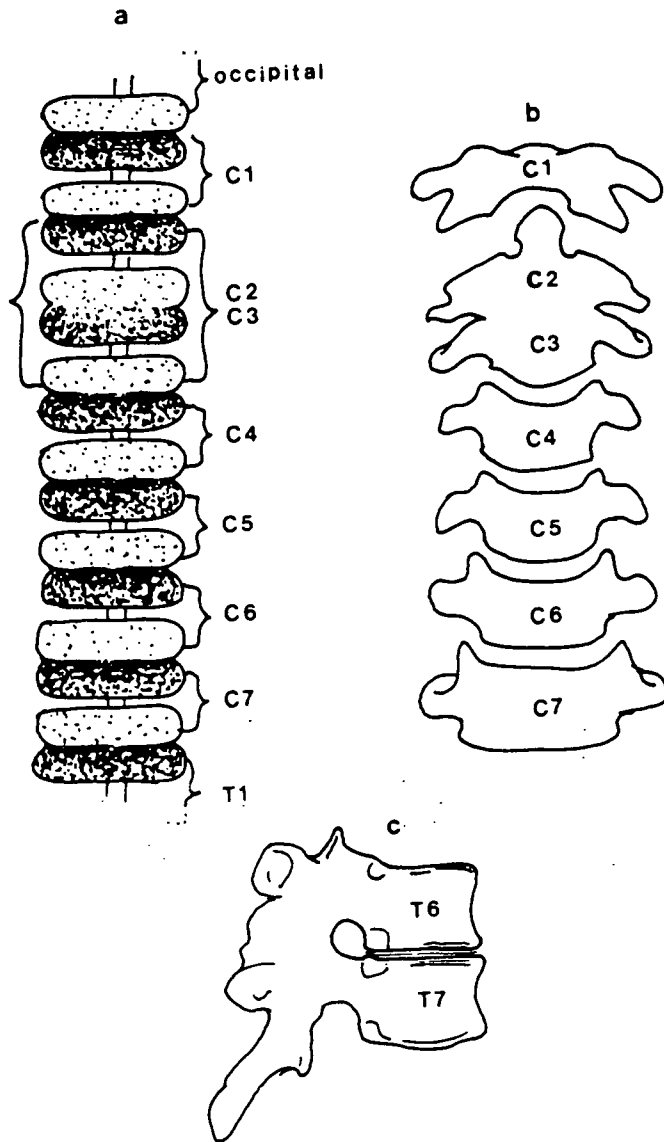


Figure 2.4: Fused or block vertebrae (from Barnes, 1994:68).

Epstein *et al.* (1983) reported a case of traumatic myelopathy and cervical spinal stenosis in association with Klippel-Feil fusion. A seventeen-year-old male suffered an injury resulting in immediate quadriplegia. On examination he was seen to exhibit central cord syndrome (trauma to the cord due to injury) although there were no fractures or dislocations. Radiography revealed fusion of C2 and C3 and cervical stenosis. Conservative treatment resulted in complete recovery. The patient recalled numbness in his arms for several years when playing sports.

Pizzutillo *et al.* (1994) looked at risk factors of Klippel-Feil Syndrome. They reviewed 111 patients with two or more fused vertebrae. This included 44 males and 67 females with ages ranging from 6 years to 53 years. Not all of the cases were due to birth fusion, but those that were showed the same range and type of symptoms as those with traumatic or degenerative fusion. Eleven of the patients with congenital fusion had associated cardiovascular disease, particularly ventricular septal defects. Thirty had reduced hearing; many had hypoplastic, absent or extra thumbs, thenar hypoplasia, carpal fusion, metacarpal fusion, short phalanges, absent radius, congenitally-dislocated radial head, and radio-ulnar synostosis (1994:2112). Rib anomalies, cleft palate, odontoid hypoplasia, and seizure disorders were also noted. The commonest cosmetic problem was a shortening of the neck with webbing of the skin, which affected 38 patients (1994:2113). Limitation of neck movements was a significant finding. Lateral bending and rotation was limited but flexion and extension (forward and backward motion) was normal (1994:85). Hypermobility of the segment below the fusion resulted in degenerative disc disorders (1994:2114). The authors concluded that neurological problems were associated with fusion in the upper cervical segment, particularly if there was hypermobility in flexion and

extension. Patients with abnormal mobility of the upper and lower cervical segments were at risk of neurological and degenerative consequences (1994:2114).

Winter *et al.* (1983) conducted a retrospective review of 1215 patients with birth scoliosis and kyphosis. Twenty five percent showed segmentation defects (block vertebrae) in the cervical spine. Of these, eighty-two patients had single-level defects, of which fifty two were at C2-C3. Seventeen patients had a single-level defect at C6-C7. Thirty nine patients had defects at two levels, of which sixteen had defects at C2-C4, and twenty three patients at C5-C7. A small number of patients showed a three-level defect covering either C2-C5 or C4-C7. Overall, 235 patients had scoliosis, 36 had kyphoscoliosis, 10 had lordoscoliosis and 17 had kyphosis alone. The birth nature of the scoliosis and kyphosis eliminates the suggestion that they were secondary to the segmentation defects.

Lowry (1977) considered Klippel-Feil Syndrome and its relationship to Foetal Alcohol Syndrome. The suggestion was made that maternal alcohol abuse may have an effect on cervical segmentation. Yasuda and Poland (1974) also reported a case of vertebral fusion in the neck in association with other defects in the aborted foetus of a seventeen-year-old mother with a history of alcohol abuse.

Fused cervical vertebrae are clearly associated with a variety of neurological deficits and birth defects. Further study is therefore warranted on the examples of Klippel-Feil syndrome reported in the archaeological literature (for example, Barnes, 1994; Sture, 1997; Wade, 1981).

Summary of fused vertebrae/Klippel-Feil syndrome review

- the condition originates in the blastemal stage in the paraxial mesoderm developmental field and is a developmental union rather than a fusion of elements - therefore it is an error of segmentation
- it can be associated with stenosis (narrowing) of the neural canal
- it is associated with hypermobility of adjacent vertebrae which can lead to nerve impairment to the shoulders, arms and hands
- it is associated with excess maternal alcohol consumption before and during pregnancy
- it is associated with a number of soft-tissue anomalies including cardiovascular disease (particularly ventricular septal defect), reduced hearing, severe malformations of the arms and hands, anomalies of the ribs, cleft palate, webbing of the neck skin, limited rotation and lateral bending of the neck, degenerative disc problems in the neck, and associated nerve impairment
- the condition clearly has the potential to adversely affect health significantly, and may also be associated in some individuals with visible birth defects which may result in social exclusion or negative discrimination.

**2.1.vi Supernumerary ribs**

This condition is believed to be atavistic, as the general evolutionary trend is reported to be tending towards an overall reduction in the number of ribs (Black & Scheuer, 1997). Cervical ribs appear to be more common than lumbar ribs, and tend to be bilateral. They are usually associated with C7 and are more common in females.

There is believed to be a familial tendency and therefore a genetic effect. Clinically, only about one third of sufferers present with symptoms, and the remainder are discovered incidentally. Lumbar ribs seem to be invariably asymptomatic (pers. comm. RW. Smithells). The rib(s) may continue to grow until the age of about 25 years, so symptoms from cervical elements may present only in early adulthood (Black & Scheuer, 1997). Symptoms are typically neurological. The spinal nerve roots leave the spinal cord through the transverse processes of the cervical vertebrae. When a cervical rib is present, the nerve trunk may be compressed, causing either sensory or motor deficits, or both, in the arms and shoulders. Conversely, the growth of the rib is itself restricted by the pressure from the nerve trunk, otherwise it could potentially grow a lot longer. As a result, most cervical ribs merge with the transverse process of the parent vertebra (Black & Scheuer, 1997). Clinically it has been noted that the hand is more frequently affected than the arm (Finnegan, 1976:9). Clearly there is potential for further study of the neurological deficits possibly associated with reported cervical ribs from archaeological contexts (for example, Wells, 1979, see section 2.3.i.).

#### **2.1.vii Cleft palate/lip** (see Fig. 2.5a and b)

This condition is a malformation of the maxilla, or upper jaw and originates in the Branchial Arch I developmental field. The soft and/or the hard palates may be involved, with a cleft, or opening, remaining in the roof of the mouth at the time of birth. The defect may affect the bones of the palatine process, which form the roof of the mouth, or the soft-tissue forming the soft palate at the back of the mouth, often involving a cleft uvula. Cleft mandible occurs very rarely (Warkany, 1971, was



unable to give a modern incidence), and presents as two bony halves joined by connective tissue bands in life. The cleft mandible usually contains the normal complement of teeth and a cleft tongue may be involved (Warkany, 1971:626).

Cleft defects of the primary and secondary palate are the most frequent birth malformation of the head and neck (Hagberg *et al.*, 1998: 40). There are two forms of cleft palate. The commonest, known as midline cleft palate, affects the back of the mouth, either unilaterally or bilaterally. Modern incidence of cleft palate is 1:400 (a world average, with variations between countries), with males affected twice as often as females (Levene & Tudehope, 1993). Barnes, however, states that females are affected more commonly than males (1994:172). The second form may occur in association with cleft lip (hare lip), in which case only the front of the mouth (the pre-maxilla) is affected. This form is usually unilateral. Cleft lip may occur on its own without any palatal defect (Levene, 1991; Barnes, 1994) and in this condition also, males are affected twice as often as females (Avery & Taeusch, 1984:819). The incidence of cleft palate and/or cleft lip is 1:500 (Avery & Taeusch, 1984; Levene, 1991), with the male:female ratio the same as in midline cleft palate, i.e. 2:1 (Levene & Tudehope, 1993). A developmental delay in the formation of the roof of the mouth leads to the non-union of the palate. Clefts affecting the back of the mouth may be relatively insignificant, and covered in oral mucosa (a "sub-mucosal" defect), so that the individual may not have a serious problem with eating, drinking, or breathing. This type of defect is a "dorsal notch", affecting the posterior margin of the palate (Barnes, 1994:171-4). Dorsal notches are the result of short delays in development, and clinically are often only discovered when individuals suffer from recurring middle ear infections, chest infections and hearing loss (Avery & Taeusch,

1984; Levene, 1991; Barnes, 1994). In antiquity, these may have been the only symptoms of the condition in affected individuals, with nothing ever seen in the mouth. Clefts in the pre-maxilla (the part of the upper jaw between the two canine teeth) may be small notches in the alveolar bone, or may extend up to the nasal margin. Severe defects like this can also extend back along the maxilla into the mouth, leaving the mouth and the nose as one cavity instead of two. This can be surgically corrected today, but in the past it may have been incompatible with survival in some babies, as eating, drinking and breathing would be difficult for the infant (Avery & Tausch, 1984; Levene, 1991). Corrective surgery in the past has been reported: Anderson (1994) described the surgical repair of a cleft palate (although this must surely have been a cleft lip) during the English Anglo-Saxon period and a repair carried out in China c.390 BC has also been described (Anderson, 1994; Fiori, 1983).

The most severe form is aplasia of the palatine processes. This is a wide cleft leaving only a rim of palatal bone at the alveolar edge of the maxilla. Hypoplasia results in some palate formation, usually symmetrical if the cleft is bilateral, varying in size depending on the degree of delay in formation. Bilateral clefting does not, however, allow correct fusion of the roof of the mouth with the vomer in the nasal cavity (Barnes 1994). The commonest form of midline cleft palate is a unilateral defect, varying in size and reflecting developmental delay time. This form allows fusion with the vomer on one side of the palate, so that some tissue can form as a separation between the nasal and oral cavities.

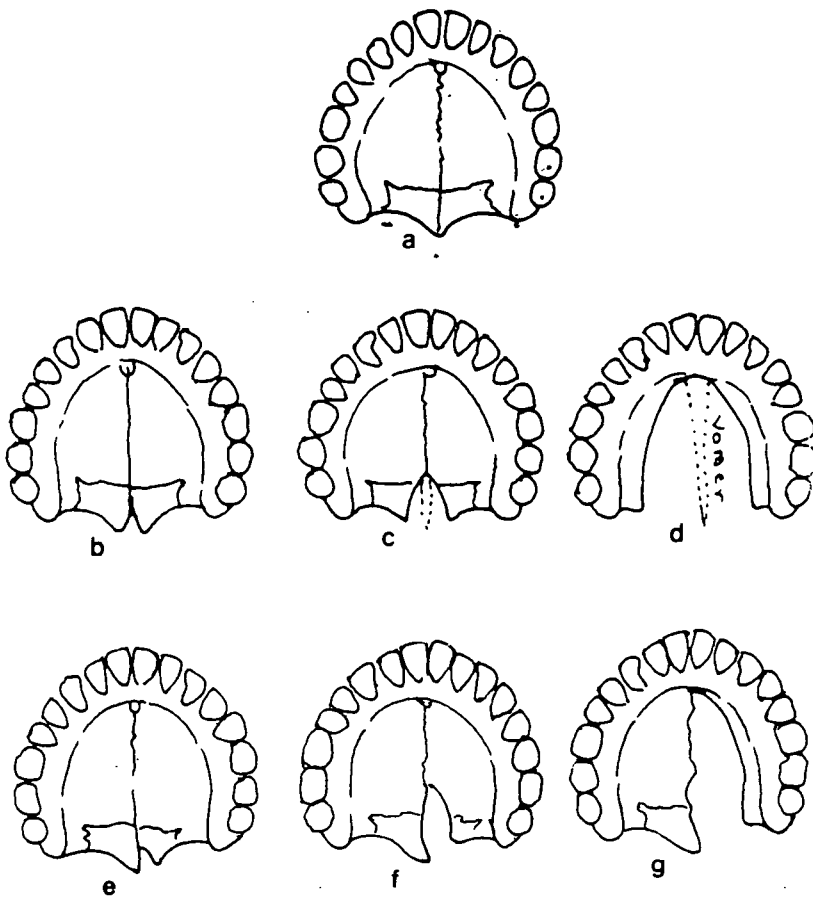


Figure 2.5a: Cleft defects of the maxilla (from Barnes, 1994:173).

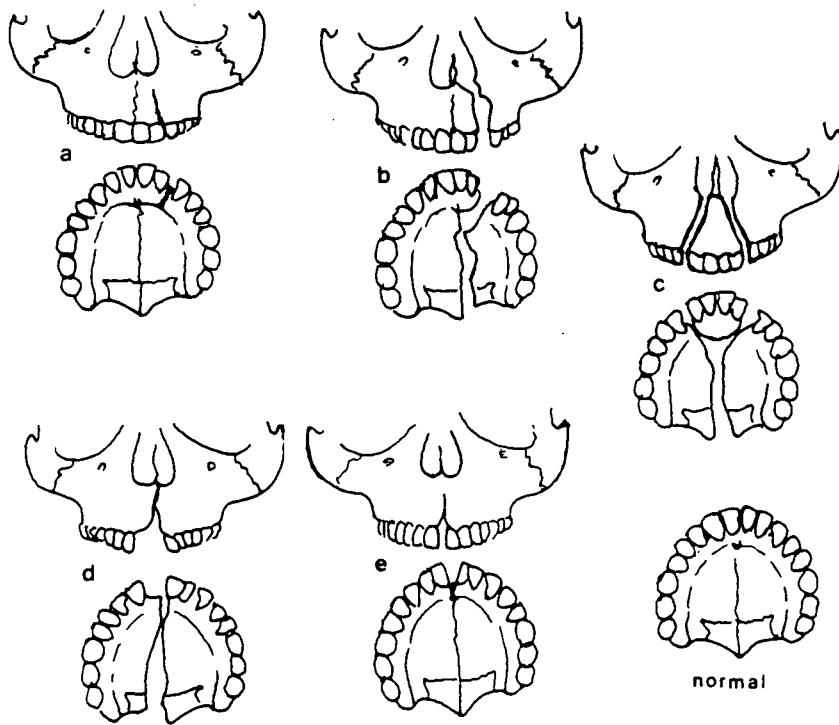


Figure 2.5b: Examples of cleft lip (from Barnes, 1994:186).

The implications in the past for babies born with an open cleft palate were potentially serious. Open clefts could prevent normal feeding, with the milk being expelled through the nose as the infant suckled (Avery & Taeusch, 1984; Levene, 1991; Levene & Tudehope, 1993). Obviously, babies so affected would probably have had reduced chances of survival beyond the neonatal period, although there are examples of adult skeletons in American collections who did survive with such defects (Barnes 1994:176). The assumption that most babies born with serious oral clefts would have died relatively quickly due to feeding problems is probably erroneous - the complications are possibly over emphasised, with many infants and small children being able to survive quite well with adequate care and attention (pers. comm. M. Levene). The apparent lack of such children in the historical period archaeological record could therefore suggest the practice of infanticide before baptism, thus removing the infants from the Christian burial place. The environmental conditions associated with variation in cleft defect frequency arguably were as common in the past as they are today, for example close contact with leather and pelts, such as in the tanning and curing industries (Garcia & Fletcher, 1998; Bianchi *et al.*, 1997), so it is reasonable to assume that more cases of cleft defects occurred than are visible in the record. Preservation of the maxilla is, however, an issue in trying to reconstruct past rates of cleft palate, as it is a particularly fragile part of the skeleton; this, of course, could be a major hindrance in assessing past rates. Of those individuals who survived into childhood and beyond, many would have suffered problems with ear infections, impaired hearing and upper respiratory tract infections (Avery & Taeusch, 1984; Levene, 1991). Speech may also have been affected, as a result of deafness due to infection (impairing the ability to learn). Broen *et al.* (1996), in a study of 57 children (28 with cleft palate and 29 without) found that the affected children almost

universally suffered from *otitis media* with effusion (OME), a middle ear infection with associated production of fluid. OME is related to eustachian tube dysfunction, and children whose tubes are blocked or otherwise impaired, for example, with cleft palate, do not have normal middle ear function. This results in their hearing becoming impaired. Whilst this is surgically treatable today, in the past affected individuals would have suffered the consequences of OME - increasing hearing loss, possible language problems, and cognitive delays (Broen *et al.*, 1996:127-133)

There is distinct geographical variation in the incidence of both forms of cleft palate today (Levene & Tudehope, 1993). For example, in Britain, cleft palate has a very low incidence in Wales compared to the rest of the country, a fact that remains unexplained (Levene & Tudehope, 1993). Cleft lip and palate rates are known to be high among Asian populations (possibly related to consanguinous marriage practice), intermediate in white populations and low in blacks/African populations (Murray *et al.*, 1997). However, from an archaeological point of view, there may be sufficient numbers of individuals from around the world in excavated skeletal collections to demonstrate a meaningful prevalence in the past (or at least a minimum observed rate) if the condition is correctly identified. Explanations for today's geographical variation have yet to be agreed upon, but different recording processes may not help. For example, some authorities do not record sub-mucous cleft palate because it is not always apparent at birth, and many do not record defects in still-births or aborted foetuses (Levene & Tudehope, 1993). Metabolic disorders are thought to play a part in the formation of cleft defects. For example, the inability to metabolize folic acid, certain nutritional deficiencies, starvation and Vitamin A excess, have all been investigated as possible factors acting on individuals with a genetic susceptibility to

produce cleft defects (Avery & Tausch, 1984; Barnes, 1994:187; Levene, 1991; Levene & Tudehope, 1993). Seasonal peaks in incidence (high in December and January, low in May and June) suggest some environmental link (Barnes, 1994:187), possibly related to dietary factors such as fresh vegetable availability.

Humans are not the only populations affected by cleft defects. A reported case of spontaneous cleft palate in a gorilla (Siebert *et al.*, 1997), showed identical morphology to the defect in humans. The parents' reproductive history (genetic input) was reported to be "non-contributory". The authors recognised the role of environmental factors in the origins of cleft palate, and documented similar defects in other non-human primate species. In view of the apparent frequency of oral-facial clefts in non-human primates cited by the authors, further research into such cases as they occur may provide useful evidence as to the possible influence of environment on the incidence of the defect in primates in general.

#### **2.1.vii.a** *Cleft defects in the medical literature*

There are too many reported cases of cleft defects in the literature to review. These are published principally in the national oro-facial cleft journals and may be viewed by interested parties. A considerable amount of work has been published on research into cleft defects, much of it in the teratological literature, and various studies have been reported which examine ethnic, genetic and environmental issues in relation to cleft defects. A selection of those illustrating these points follows.

Moss (1956) described malformations of the skull base in association with cleft palate, and Oldfield (1959) made suggestions about the causes of cleft defects based

on clinical work, including the possible roles of infection, viruses and malnutrition in early pregnancy on cleft defect rates. Miller found that nutritional disturbances resulted in cleft palate development in mice without the addition of any teratological agent (1973). An American study showed that cleft defects seem to behave differently, epidemiologically speaking, depending on whether they are isolated defects, or found in association with other anomalies (Emanuel *et al.*, 1973). The usual excess of males with cleft palate with or without cleft lip was found only for “pure clefts” and those associated with other minor malformations, often hypospadias ( a urethral opening under the penis) and inguinal hernia. Excess female clefts, on the other hand, were associated with other major malformations (unspecified). For pure cleft palate alone, the “usual” female excess was observed. Low birth weight was particularly noted in cases of cleft with associated defects. Maternal age, birth order, season and time-space clustering effects were absent, suggesting non-genetic aetiological factors “are probably less important in the pure clefts than they are in certain malformations such as anencephaly and spina bifida” (Emanuel *et al.*, 1973:279).

Ikeda (1973) noted that cleft lip and/or palate may often be associated with disorders of the urinary and gastro-intestinal tracts, and with hydrocephaly. Lowry (1977) noted cleft palate in association with Klippel-Feil Syndrome. Lowry and Trimble (1977) compared the rates of cleft defects between ethnic groups in British Columbia. They concluded that only a small number of clefts were due to teratogenic action, the remainder being of multifactorial inheritance. However, teratogenic agents may play a part in this. Murray *et al.* (1997) looked at the incidence of cleft defects in the Philippines and found higher risk rates for occurrence



in siblings than are seen in Britain. Hagberg *et al.* (1998) also looked at the association of additional malformations with cleft palate in a Swedish study. They noted discrepancies in rates between ethnic groups and that rates have risen in recent years in Scandinavia, although this may be due to immigration from developing countries.

Summary of cleft palate/lip review

- the condition(s) originate from the first branchial arch developmental field, due to a developmental delay in the formation of the roof of the mouth, resulting in non-union of the two halves of the maxilla and/or the formation of the pre-maxilla
- clefts may involve hypoplasia of one half of the palate (a unilateral defect) or a bilateral defect which is usually symmetrical in shape
- the condition appears to have genetic and environmental origins, as there are marked racial, geographical and occupational differences reported in the literature
- males are affected more than twice as often as females (though some authors disagree)
- mild expressions of cleft defects may affect only the pre-maxilla, incisors, canines and/or the shape of the nasal aperture, or, may involve a dorsal notch (a sub-mucosal defect) at the back of the mouth, which may be associated with frequent upper respiratory tract infections and some hearing loss
- nutritional deficiencies/variations are implicated in cleft defect rates
- there is an association between maternal occupation in the leather/pelt industry and a raised rate of cleft defects

- there are differences in male:female rates depending on the type of cleft defect, with variations in associated malformations elsewhere in the body
- these include malformations of the genito-urinary tract, the gastro-intestinal tract, Klippel-Feil syndrome, and hydrocephaly.
- The conditions clearly have serious adverse health effects in some individuals, apart from the cosmetic issues, which may have resulted in social exclusion and/or negative discrimination.

### 2.1.viii Birth defects of the axial skeleton in recognised syndromes

A number of recognised syndromes are associated with specific defects of the spinal column and the maxilla. These are presented in tabular form below.

Table 2.1 Skeletal defects within syndromes, with associated soft tissue anomalies

Syndrome	Post-cranial skeletal defects	Oro-facial defects	Soft-tissue defects
Nail-Patella Syndrome (hereditary osteo-onychodysplasia)	spina bifida, pelvic bone spurs patella hypoplasia	-	nail anomalies
Acrodysostosis syndrome	small vertebrae, spinal curvatures, short hands,	small nose	mental retardation
VATER association	many vertebral defects, polydactyly	-	cardiac anomalies, fistulae
Oto-palato-digital syndrome (Taybi syndrome)	-	cleft palate, frontal & occipital prominence, partial adontia	mental deficiency, broad distal digits, deafness
Coffin-Lowry syndrome	anterior/superior vertebral margin anomalies, thoracolumbar scoliosis, flat feet	downslanting palpebral fissures, bulbous nose, hypertelorism	mental retardation
Cohen syndrome	lumbar lordosis and mild scoliosis	prominent incisors, high nasal bridge, maxillary hypoplasia, mild micrognathia	hypotonia, obesity, mental retardation
Contractural Arachnodactyly syndrome (Beal's)	arachnodactyly, short neck, kyphoscoliosis	occasional micrognathia	joint contractures, esp. knees, elbows and hands; crumpled ear

Facio-auriculo-vertebral anomalad	vertebral dysplasia inc. hemivertebrae, hypoplastic vertebrae; limb and rib anomalies occasionally	Cleft palate and/or cleft lip; hemifacial dysplasia, usually unilateral; and Goldenhar syndrome	mental retardation
Mucopolysaccharidosis VI <i>or</i> Hurler's Syndrome (when seen with mental retardation)	flattened vertebrae with anterior wedging of T12-L1; lumbar kyphosis; broad ribs; epiphyseal anomalies	macrocephaly	stiff joints; clouded cornea & coarse facies in infancy; frequent URTI, cardiac complications.
Mucopolysaccharidosis IV (Morquio syndrome)	severe kyphosis; knock knees; stunted height; flattened, ovaloid vertebrae, some with anterior projections; scoliosis, short neck & trunk; platyspondyly	-	mild coarse facies; clouding of cornea; cardiac problems; hearing loss; mental retardation occasionally
Multiple epiphyseal dysplasia syndrome	small irregular epiphyses; restricted stature; blunted, ovaloid or flattened vertebrae	-	stiffness in joints, osteoarthritis
Progeria syndrome (Hutchinson-Gilford Syndrome)	small ribcage; coxa valga; ovoid vertebrae	delayed eruption of deciduous and permanent teeth; facial hypoplasia; micrognathia	atherosclerosis; severely premature aging - "elderly" by teen years
Robinow syndrome (foetal facial syndrome)	hemivertebrae; short forearms	flat facial profile; high arched palate; crowded teeth; hyperplastic alveolar ridges; frontal bossing; macrocephaly	hypoplastic genitalia, no mental retardation
Stickler Syndrome	flattened vertebrae; some anterior wedging; distal tibia has under-developed epiphysis; spondylo-epiphyseal dysplasia	marfanoid appearance	hypotonia; hyperflexible joints; subluxation of hip; flat facies
X-linked spondylo-epiphyseal dysplasia syndrome	flattened vertebrae; small iliac wings and short neck; kyphosis, scoliosis with central hump, short femoral neck	-	

Warkany (1971:571) stated that "anomalies of the skeleton are relatively important complications of CHD" (congenital heart disease). He listed examples of skeletal anomalies associated with congenital heart disease, of which kyphoscoliosis (forward and lateral abnormal bending of the spine) is the only one affecting the vertebral

column. Sherk *et al.* (1982) attempted to document spinal anomalies that were found in association with specific facial abnormalities. They reviewed twenty-six patients with hemifacial microsomia and Goldenhar Syndrome (see above). Of these, eight patients also had a cervico-thoracic scoliosis and hemivertebrae in this region. One patient had an L4 paraplegia with paralytic scoliosis (lateral bending of the spinal column resulting from the L4 defect) because of a myelomenigocele. One patient had some cervical fusion with cervical ribs, and another showed cervical *spina bifida* (cleft neural arch) with congenital fusion of some cervical elements. They also reviewed ten patients with Apert's Syndrome (a craniofacial anomaly with craniostenosis - premature fusion of the cranial sutures, a cranial base deformity and midface hypoplasia). Four had symmetrical block fusion of areas of the cervical spine without scoliosis. Two patients exhibited absence of the posterior atlas arch (cleft neural arch) (Sherk *et al.*, 1982:526). Apert's Syndrome patients were noted to have "a high degree of correlation with upper cervical spine anomalies and non-deforming failure of segmentation of portions of the cervical spine as well as syndactylism [webbing of the fingers]" (Sherk *et al.*, 1982:528). It was noted that Goldenhar Syndrome and hemifacial microsomia patients also showed high rates of congenital heart disease. The authors suggested that these associations were due to developmental faults during the fourth to the eighth weeks of embryological life. The areas affected are derived from the first branchial arch and its pouch, which fuse across the midline and give rise to the maxilla, the mandible, part of the external ear, the malleus, the incus and portions of the lip, cheek and salivary glands. Other elements caudal to the neck develop from other branchial arches. The close physical association of these elements and their precursors in the embryo is combined with the

contemporary resegmentation of the cervical spine. Difficulties in one area of differentiation thus often involve associated structures (Sherk *et al.*, 1982:528).

### **2.1.ix Summary Of Axial Skeletal Defects In The Literature**

- Some, but not all, axial skeletal defects are reported in the literature
- it is possible to associate certain soft tissue anomalies with certain skeletal defects through these reports
- by observing such factors as pain, reduced or extra mobility and so on, in modern patients, it is possible to infer the presence of similar problems among a proportion of affected individuals in the past
- there are recognised syndromes which include axial skeletal defects which may be diagnosed in archaeologically-derived bone
- many of the “minor” morphological defects are not noted in the literature because they are either not diagnosed, or they are not considered meaningful in terms of the patient’s health prospects. This is a hindrance to biological anthropologists as they attempt to relate soft-tissue disease to the presence of these defects. This situation may be addressed by biological anthropologists impressing on clinicians the importance of recording such anomalies, an exercise which will probably only be successful if biological anthropologists make more effort to explain their efforts to the medical profession.

## 2.2 BIRTH DEFECTS IN HUMAN PREHISTORY AND HISTORY

Congenital defects have long been the subject of much curiosity to humans. In the past, individuals who had the misfortune to be “abnormal” in some way, usually in appearance, were often the subject of special attention. Even today it is not uncommon for individuals to be treated differently, often unfavourably, due to their unusual appearance. The view that affected individuals were in some way “abnormal” was confirmed by the nature of the title given to this type of anomaly - defect. This word is derived from the Latin *deficere*, meaning “to fail”. Even the term “abnormality” is derived from the Latin *ab*, meaning “away from”, and applied to what is considered to be the norm by the majority of the [unaffected] population.

The earliest known example of recording of a congenital condition is a marble statuette from the Neolithic site of Catal Huyuk in southern Turkey. It depicts laterally conjoined twins (“siamese twins”), and dates from about 6500 BC (O’Shea, 1995; Warkany, 1983). Other representations of congenital defects have been found in numerous archaeological contexts. Dicephalic (two-headed) children have been recorded in many countries - for example, clay figures from Mexico dating from 500 BC to 800 AD, and chalk and wood carvings from sites in Oceania (O’Shea, 1995). An Egyptian tomb in Thebes dated to 1500 BC contained paintings of an Egyptian Queen and daughter with achondroplasia, and a Roman bronze depicting an achondroplastic gladiator is exceptional as an accurate representation of the condition (Warkany, 1983).

Mystical and religious explanations generally accounted for the occurrence of such malformations, and the arrival of deformed babies into families and communities was often considered bad luck (Dryden, 1978; O'Shea, 1995; Warkany, 1971, 1983). Whilst some defects, such as achondroplasia (dwarfism) have in the past been the subject of benevolent interest, many others were attributed to malevolent forces such as the anger of the gods, devils and demons, indecent posture by the mother during pregnancy, "corrupt seed", or seen as signs of witchcraft (Anderson, 2000; Dryden, 1978; O'Shea, 1995; Warkany, 1983). Obviously this could have had potentially devastating, if not occasionally fatal, effects on the individual and even their family. Warkany refers to the maltreatment of affected children and their parents by their neighbours "who thought that killing the messenger could prevent the coming calamity" (Warkany, 1983:20).

Religion and superstition played contradictory roles in the lives of sufferers. Whilst superstition (and occasionally harsh religious views) often seem to have led to problems for affected individuals, religious institutions offered care and acceptance in the form of monastic or guild hospitals, for instance (Covey, 1998, cited in Roberts, 2000). Warkany (1971,1983) emphasises that there are still modern-day superstitions associated with birth defects, even among western societies, and Roberts reminds us that it is still common to find outer appearances being viewed as determinants of the inner state (Roberts, 2000).

There is illustrative documentary evidence for conjoined twins (see fig. 2.6) from 16<sup>th</sup> century England (Anderson, 2000) and Germany (Herrlinger, 1970). Medical

illustrations depicting "monstrous births" were often published as a warning against sin, or for sensational journalistic purposes rather than for medical enlightenment (Herrlinger, 1970; Anderson, 1994b). Written and illustrative accounts from the past often included some attempt at explanation or interpretation of the "monstrosity". For example, an illustrated document of 1568 AD describes an infant with a midline cleft, *genu recurvatum congenitum* (the leg growing up towards the top of the body) and *spina bifida cystica*. The author of the document stated that the child's deformity was God's punishment on the moral deficiency of the mother, who was unmarried (Anderson, 1994b), although no mention was made of the moral deficiency of the father. The likeness of some defects to animals is reflected in the names given to defects in the past, many of which are still used today, for example, hare-lip. The belief in animal-human hybrids was widespread in the past. It was considered acceptable in India and Egypt, but the Judaeo-Christian tradition considers hybridisation a deadly sin (Warkany, 1983:20).

Pliny the Elder, writing in the 1<sup>st</sup> century AD, noted the transmission of defects through generations of affected families, but could not understand how "sound parents may have deformed children and deformed parents sound children" (Warkany, 1983:25). Leonardo da Vinci suggested that study of the foetus could shed light on the origin of congenital malformations in his work "*Quaderni d'Anatomia*". This was followed by Geronimo Fabricius' "*De Formato Foetu*", which proposed similar ideas. Ambroise Pare, a French surgeon, wrote *De Monstres et Prodiges* in 1573 and a later version in 1582, works which described causes of "monstrosities". He echoed the superstitions of the time, but showed some



knowledge of embryology (or good deduction) when he stated that after forty-two days of gestation the risk of malformation had passed (Warkany, 1983:26).

William Harvey, the British physician and surgeon, in his 1651 work "*De Generatione Animale*" proposed that defective development of the embryo lead to malformations (O'Shea, 1995). James Blondel, writing in 1729 in *The Power of the Mother's Imagination over the Foetus Examined*, said "the prosperity of the foetus does depend on the welfare of the mother...whatever is detrimental to her is directly, or indirectly, prejudicial to the other" (Oakley *et al.*, 1982:17). This view of the mother's moods affecting the foetus still held even into the twentieth century. A deformed baby was believed to be due to the emotions of the mother, and if she allowed herself to be upset, angry and so on, this would transmit to the foetus and result in abnormality (Oakley *et al.*, 1982:17).

In 1822 an important work was published in France. "*Philosophie Anatomique des Monstruosites Humaines*", by le Chevalier Geoffroy St.-Hilaire, covered many aspects of the study of birth defects still addressed today. The title page announced that the work covered:

"a classification of monsters; a description and comparison of the principal types; a reasoned account of the phenomenae and of the primitive mechanisms which produced them; new perspectives on the nutrition of the foetus and other circumstances of its development; and the determining role of the various elements of the sexual organ, in order to demonstrate by this the unity of composition, not only of monsters, in which the altered form renders the organism unrecognisable, but of both sexes, and in addition, that of birds and mammals" (Smithells, 1980, in the original French: translation by Sture).

Between 1832 and 1837 Geoffroy St.-Hilaire's doctor son published further work on birth defects in three volumes, with an accompanying atlas. The shortened title of this work was "*Traite de Teratologie*". The younger author was the first to coin the term "teratology" (Smithells, 1980). He commented on this in his far-sighted

preface, in which he justified his position in considering that “knowledge about monstrosities” should be a distinct science.

Environmental influences on the foetus were first recognised and have been studied in a systematic way only in the last sixty years. Following the rediscovery of Mendel’s work at the turn of the twentieth century, it was believed that defects were solely due to genetic factors. It was not until events such as the rubella (German measles) epidemic in Europe during 1939-40 that exogenous causes came to be recognised, and the multi-factorial aetiology of defects came to be appreciated. The bombing of Hiroshima and Nagasaki at the end of the Second World War then led to studies on the effects of radiation on infants born to mothers who had been pregnant at the time of the bombing. The thalidomide disaster of c.1960 in Britain led to the realisation that drug therapy was not without price (Poggel & Gunzel, 1992; O’Shea, 1995). However, Warkany (1971, 1983) considers that the twentieth century view that drugs are one of the most frequent causes of defects is a superstition in itself, as many such claims are unsubstantiated, but the public is willing to believe them.

### **2.3 BIRTH DEFECTS OF THE AXIAL SKELETON IN THE ARCHAEOLOGICAL LITERATURE**

The study of congenital abnormalities has largely concentrated on interesting cases rather than on large-scale population studies. As a result of this, numerous case studies are available in the palaeopathology literature, but very little in terms of work that seeks to explain the frequencies of congenital malformations in past populations.

Wells (1964), Brothwell (1967), and Brothwell and Powers (1968) published reviews of the skeletal examples of congenital diseases then available in the archaeological literature, finding that anomalies of the vertebral column and the skull were the most commonly described in the literature. This remains broadly the case today. They cited vertebral and other anomalies reported between the 1920s and 1960s, covering examples of normal variation, posterior midline clefts, separated neural arches and dwarfism amongst other examples.

Turkel (1989) described the congenital diseases in palaeopathology. Roberts and Manchester (1995) devoted a chapter to the congenital diseases, and Anderson (2000) has reviewed them more recently. Such reviews have naturally focused on those conditions which are relatively well known in medicine and the medical literature, for example, congenital hip dislocation, cleft defects, club foot, anencephaly and *spina bifida*, amongst others. No previous authors except Barnes (1994) and Sture (1997) have focused on those defects that appear on the skeleton, but not usually in the medical literature, such as cleft neural arch, and border shifting. These are precisely some of the congenital anomalies under further examination by the author, as a means of quantifying rural and urban environmentally-mediated defects.

Anderson (2000) commented on the possibility of comparing past and present frequencies of congenital defects by considering the total number of excavated individuals that have been recovered within living memory, and estimating the number with congenital anomalies. However, this is not a reliable, or feasible, method given that so many cultural and archaeological factors could prevent the appearance of affected individuals in burial grounds (although Anderson does cite

poverty as a reason for hidden burials) or in the literature. Infanticide, for example,

may well have removed some facial cleft sufferers from the record, and non-recognition of the less well-known defects by excavators (or palaeopathologists) is highly likely to have artificially reduced the apparent frequencies of defects among excavated populations. In addition, frequencies should be reported as percentages of bone elements studied, rather than of individuals, as that presupposes the presence of all the elements in all the examined skeletons. Anderson did, however, make the useful point that a database of congenital (and neoplastic) diseases would assist in the quantification and understanding of any changes in frequency over time and across space.

Ortner and Putschar have referred to the noticeable differences in frequency of certain conditions between populations, for example the higher prevalence of detached L5 neural arches (spondylolysis) amongst Eskimo groups, compared to East Africans (Brothwell & Powers 1968:195; Ortner & Putschar 1981:358). Post (1966) also quantified population differences in the frequency of *spina bifida occulta*, although this was done as an exercise in genetic research. Farkas *et al.* (1976) assessed former Yugoslavian material and commented on the sex-related frequency of defects and on the occurrence of single/multiple defects in individuals, which is useful (see below). Unfortunately they did not make clear their diagnostic criteria.

Ortner and Aufderheide (1991) made useful suggestions as to future directions in the study of biological anthropology, arguably the most important of which was the need to relate observed conditions to the clinical base, which is vital for the understanding

of skeletal congenital anomalies. Barnes (1994) focused on the axial skeleton and its developmental defects in the first serious attempt to analyse observed congenital anomalies based on observations of Native American populations and to relate them to their embryological origins. Her work quantified observed defects but did not address the causative factors behind embryological variation. Nevertheless, Barnes' work was the inspiration for this author's research. Black and Scheuer (1996) and Usher and Christensen (2000) have since considered cases of vertebral anomalies from the same perspective as Barnes, trying to understand the condition from an embryological viewpoint, although they did not apply their findings to any population. Sture (1997) also addressed two English late Medieval populations from this perspective. In addition, various workers have cited examples of congenital defects among British/English excavated populations, usually including conditions such as lumbo-sacral border shifting, fused vertebrae, spondylolysis and spondylolisthesis, non-metric traits and especially *spina bifida occulta* (eg. Powell, 1996; Dawes, 1980; Lee, 1987; Mays, 1991; Malim & Hines, 1998 and others).

### **2.3.i General congenital skeletal defects**

#### **2.3.i.a Dwarfism**

There are a considerable number of publications relating to dwarfism in its various forms. Reports often included consideration of the social implications for the sufferer, and occasionally, his/her family (Hunt, 1973). Johnston (1963) outlined changing cultural attitudes to dwarfs in human history, illustrating their "descent" from a position of deity through to court jester, referring to specific examples from the literature and the archaeological record. Hrdlicka (1943) described a Peruvian

female “midget” of normal proportions but of tiny size. Hodge (1969) also reported on an eighteenth century German dwarf, illustrating the high regard in which many dwarfs were held in the past. Dasen (1988) reviewed the iconographic evidence for dwarfism in the classical world and considered the medical history of the condition, relating the different types to iconographic representations. Arcini and Frolund (1996) reported on two Swedish dwarfs suffering from the rare spondylo-epiphyseal dysplastic type of the condition. The authors noted a link between this form of dwarfism and cleft defects and club foot (*talipes*). Unusually, but usefully, they also noted that soft-tissue defects such as myopia and retinal detachment are associated with the disorder. Similarly, Wells (1979) reported on a female Anglo-Saxon dwarf from a northern English monastery who also had a cervical rib, and offered suggestions as to its possible association with atrophy of her arm. Frayer *et al.* (1988) described an adolescent, the earliest known case of chondro-dystrophic dwarfism from an Italian Upper Palaeolithic cave context.

### **2.3.i.b** *Limb anomalies and fusion of elements*

Absence of limbs, in whole or in part, have been reported occasionally. The appearance of such anomalies in archaeological contexts is interesting, as they are rare even in living populations (Mann *et al.*, 1998). Chadwick-Hawkes and Wells (1976) described a case of absence of the pectoral girdle and arm (phocomelia - absence of the proximal portion of a limb), in an Anglo-Saxon cemetery and commented on the possible socioeconomic status of the individual. They likened the observed defects to those associated with thalidomide, and also to clinical cases described by Balard (1938). Similar examples were reported by Lamers (1937) and

Puschel (1963) (cited in Chadwick-Hawkes & Wells, 1976). Bennike (1986) reported a case of dislocation of the shoulder joint possibly due to malformation in a Medieval adolescent male. Mahieu (1984) reported a case of tarsal fusion in an adult from a French Chalcolithic site. Leonard (1974) related tarsal coalition and spastic flat foot, noting archaeological cases from the prehistoric Americas. Mann *et al.* (1998) reported a case of congenital absence of the ulna with humeroradial synostosis from Moundville, Alabama.

### 2.3.i.c *Other anomalies*

Some work has been done by various workers focussing on cranial anomalies, particularly during the early 20<sup>th</sup> century, when the preoccupation with cranial metrical data and morphology was still a significant field of research in European scientific circles. More recently, possible cases of hydrocephaly and Down's Syndrome have also been described, probably as a result of the improved medical understanding and general awareness of genetic anomalies and congenital syndromes.

Richards and Anton (1991) reviewed the evidence for hydrocephalus. They reported archaeological specimens dating from 10,000BC to AD1670, numbering about thirty cases world-wide. Manchester (1980:80) reported a case of hydrocephalus from an Anglo-Saxon context, a child of 14-16 years, and suggested that such individuals could have contributed to society as modern studies show 57% are in the normal IQ range. Derry (1913) reported on a case of hydrocephaly from an Roman-period Egyptian context, and Murphy (1996) described a possible case of hydrocephaly in a Medieval Irish child. Brothwell (1960) described a case of possible "mongolism"

(Down's Syndrome) from a Saxon population. Seligman (1912) reported on a

“cretinous skull” from Ancient Egypt. Comas (1966) examined several cases of scaphocephaly among native American material. Suzuki and Ikeda (1981) reviewed the aetiology of premature cranial suture closure and related the condition to other classified syndromes in the medical literature. Webb and Thorne (1985) described an Australian Aboriginal skeleton with a congenital meningocele in the skull. The young adult female showed a large cranial vault lesion where the meninges had herniated out of the vault. The fact that she had survived into young adulthood suggested that she had been cared for or at least tolerated by the group. Brothwell (1958) reported a case of absent basi-occipital in a young Romano-British adult involving atlas anomalies, and suggested other conditions with which the anomaly could be associated. Porter (1980) described the lumbar spinal canal among three populations and found racial variation in shape and size, suggesting the possible significance of this in the epidemiology of back pain.

### **2.3.ii Defects of the spine in the literature**

The axial skeleton, particularly the vertebral column and the sacrum, are amongst the most common sites to be affected by morphological defects (Bradt Miller, 1984:327).

There are many reports in the literature referring to these areas.

Gregg (1980) made the important point that occult, as opposed to manifest, skeletal lesions are useful indicators, as they are less likely to be influenced by cultural behaviour which may remove them from, or otherwise dictate their position, in the burial place. Gregg noted that the frequencies of occult anomalies observed among



prehistoric Dakota Native Americans were similar to modern rates, whereas frequencies of manifest defects were not. This was unusual, as incest was a known cultural form of sexual behaviour in these groups, and one would have expected to find more cases of obvious birth defects than were apparent in the record. Along with Anderson's (2000) comments about calculating past versus modern prevalence rates of defects, it will be appreciated that such calculation and inference is problematic. Gregg's point about the more frequent appearance of occult defects in the record supports this author's suggestion that occult defects are likely to be the most useful focus of research, as their entry into the record may be less liable to cultural bias.

### **2.3.ii.a** *Spina bifida and cleft neural arch*

*Spina bifida occulta* appears to be the most frequently reported sacral anomaly in the literature. Many excavated populations have been reported to have numbers of affected individuals, and a selection is reviewed below. The high rate of reporting may well be linked to the fact that the condition is mistakenly believed to be easily recognised, and this has led to a degree of mis-diagnosis. Such diagnosis has, however, been entirely understandable prior to the development of the morphogenetic approach that Barnes used in her major work on the developmental defects of the axial skeleton. Barnes (1994:49-50) made the important distinction between *spina bifida occulta* related to neural tube defect and *spina bifida occulta* which should more accurately be described as cleft neural arch, originating from an entirely different developmental field (the paraxial mesoderm). Unfortunately, most authors have not made this distinction, and without adequate illustration or description of the

individuals cited, it is impossible to say to which group most examples belong. This issue is addressed further in chapter 10.

Although it is generally accepted that *spina bifida occulta* is an asymptomatic condition, sufferers may have died in childhood or adolescence due to tethering of the *cauda equina* and the associated complications. Barnes (1994:50) mentions two cases of *spina bifida cystica* from prehistoric American populations (De La Mata & Bonavia, 1980; Dickel & Doran, 1989). Anderson (1994c) described documentary evidence for multiple congenital anomalies including *spina bifida cystica* in sixteenth-century England. The condition, illustrated in a document depicting “marvels of nature and monstrous births”, showed a child with *spina bifida cystica*, and also hemimelia (gross shortening of distal element) of the upper limbs, atrophy of the left leg, amelia (absence) of the right leg, absence of the hands and feet, and possible absence of penis and tongue. Both parents were reported to be “normal”. Jorde *et al.* (1987) considered the genetics of neural tube defects in American populations from an epidemiological perspective, finding significant familial associations. There are no known published examples of the *cystica* form in European archaeological populations to this author’s knowledge, and its presence in American groups suggests a high(er) level of care or tolerance for the affected individuals, enabling their survival beyond infancy.

Wells (1963) observed *spina bifida occulta* affecting S1-2 in association with pedicle defects in three African sacra/lumbar segments. Ferembach (1963) reported on prehistoric skeletons from Morocco, and observed an increase in frequency over

time, which she attributed to genetic influences. Post (1966) reported on Negroid and Caucasoid spinal columns from US poorhouse cadavers, and compared them with other ethnic groups. He found bifurcations (clefts) in all segments, and suggested that mild clefts may be well-represented in skeletal series due to reduced bias (presumably because they were considered asymptomatic). Bradtmiller (1984) compared two Native American series and noted differences in the frequency and age distribution of *spina bifida occulta* and spondylolysis. He believed that differences in *spina bifida* rates between the two groups indicated that they were biologically distinct (1984:328), and made the point that true frequencies in past populations may be impossible to assess due to cultural bias in burial rites. Saluja (1986) reported on *spina bifida occulta* rates in Irish skeletal remains, and later (1988) on the incidence of *spina bifida* in historic and modern London populations, observing the modern population via radiographs taken for unrelated reasons. Bennett (1972) found *spina bifida occulta* /cleft neural arch in Californian Indians, associated with other anomalies (see below). Ortner's report (1990) on the Terry Collection described an individual with pelvic and leg atrophy as having *spina bifida occulta* of the atlas and the sacrum, and L4-5 spondylolysis. Ortner eliminated neural tube defects, neural spurs and tethered cord in this case. The atrophy of the lower body seemed to result not from the *spina bifida occulta* but from injury. Kelley (1980) recorded sacral *spina bifida occulta* among three Native American groups and believed that the condition was under genetic control. It was suggested that this may also be influenced by diet, including mineral deficiencies (particularly zinc) and water hardness. Schultz (1976) reported a prevalence of 0.3% with *spina bifida* in a prehistoric Mexican group. Goldstein *et.al.* (1976) reported on *spina bifida occulta*

and cleft atlas among two Bedouin sites in Israel. Farkas *et al.* (1976) recorded a single case of cleft atlas in a male. Evison and Hill (1996) found *spina bifida occulta* among Anglo-Saxon material in southern England, in adults and one child. Sture (1997) found two cases at Raunds Furnells (Anglo-Saxon) and six at the later Medieval Chichester hospital site (see fig. 2.7).

Most of the true *spina bifida* cases seen in archaeological populations, certainly those of adults, are examples of the *occulta* form, largely because of the complications preventing most cases of the *cystica* form surviving into adulthood. The possibility of *spina bifida cystica* appearing in the record should not be ruled out, however (Barnes 1994:50), although in the absence of soft tissue evidence, or related motor/sensory neuropathy evidence (such as lower limb atrophy) it is difficult to make the distinction. Atrophy due to non-use of the legs after trauma, or damage resulting from sensory impairment could be the result of other pathological conditions.

Many of the above mentioned cases may well be, in reality, not *spina bifida occulta*, but examples of cleft neural arch. Without a review of the material, this is, however, impossible to quantify. Some reports do show photographs, which can be a considerable help in making a diagnosis. For example, Evison and Hill (1996) describe an Anglo-Saxon skeleton with a defect of the atlas which is clearly a unilateral cleft neural arch (from a photograph), but do not photograph the lumbar vertebrae L3-5 which are diagnosed as *spina bifida occulta*.





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 Xpue und goten vor an. Wan der künig hat es die :  
 Ein gros wunder seichen. und man sagt es sol wil.  
 aber dur. von got. ver hengt werde von des kindes :  
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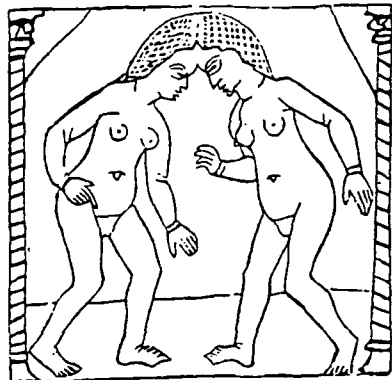


Figure 2.6: Medieval illustrations of conjoined twins (from Herrlinger, 1970)

### 2.3.ii.b *Spondylolysis and Spondylolisthesis*

Barclay-Smith (1910) described a female vertebral column with multiple anomalies, including spondylolysis. The defect was ascribed to her probable activities as a gymnast or contortionist. Associated articular surface anomalies were also noted, and these were referred to by Wells (1963) in comparison to the three African specimens noted above. Wells also described pedicle anomalies in lumbar vertebrae which weakened the pedicles, but which had resulted in a unilateral spondylolysis-type effect in only one specimen. The defect was attributed to a defect of chondrification. Stewart (1931, 1932, 1953, 1956) reported on the prevalence of spondylolysis in Eskimo populations and considered the high frequencies found to be related to their strenuous lifestyle. Merbs and Wilson (1960) also considered the defect among the Eskimo. Farkas *et al.* (1976) noted cases of spondylolysis among Avar culture (Yugoslav) skeletons, affecting males almost four times as often as females. Bradtmiller (1984) found different frequencies of separate neural arch between two native American skeletal series, and linked the condition to both genetic and mechanical stresses (1984:329). The group with the highest rate of *spina bifida occulta* also had a higher rate of separate neural arches. Osteoarthritis and osteophytosis were found to be associated with the frequency of spondylolysis (1984:331). Mulinski (1974) found a rate of 25% lumbar spondylolysis amongst Alpayeyma burials in Washington state, U.S.A.. Among these, 30.8% of the observable vertebral columns also had a sixth lumbar vertebra, and the majority of those with the extra element also had some degree of sacralisation. This picture is similar to that of the Modoc Indians (see below). Kelley (1980) found varying frequencies of spondylolysis with or without spondylolisthesis among three Native

American skeletal populations. Trembly (1995) reported a prevalence of 38% with L5 spondyloysis in males and 14% among females in a population from the Mariana archipelago. Spondylolysis of L4 was found to affect 5.5% of individuals overall. Trembly suggested that the condition may be related to the outstretched leg position involved in paddling canoes, similar to that practised by Eskimo groups who also have a high rate of spondyloysis. Ozonoff (1995) commented on the non-appearance of lumbar spondylolysis in human infants on radiographic examination, but stated that when affecting the cervical spine the lesion is always congenital in origin. Merbs and Euler (1985) described an Anasazi middle-aged female with L5 bilateral spondylolysis, in whom the L5/S1 disc had ossified, rendering theolisthesis permanently recorded. She also suffered other spinal anomalies (see below). Merbs (1996) reported on a 4% prevalence of spondylolysis of the sacrum in a group of North American Inuit skeletons, and suggested that the lesion may have been common due to activity patterns, and possibly delayed maturation. Arriaza (1997) described a spondylolysis prevalence of 21% among Guam natives from the 13<sup>th</sup> - 16<sup>th</sup> centuries AD, also attributing the prevalence to activities, in this case, building large houses from huge stone blocks. Sture (1997) found a low rate of spondylolysis at Raunds Furnells (around 5% mean male/female rates) and a higher rate at Chichester (around 9% mean male/female rates).

### 2.3.ii.c *Segmentation errors - block vertebrae*

Merbs and Euler state that congenital fusion of vertebrae is a familial trait, often accompanying atlanto-occipital fusion (1985,388). Farkas *et al.* (1976) recorded block vertebrae in two Avar (former Yugoslavian) males, one of which affected the

cervical vertebrae. The location of the other block case was unspecified. Schultz

(1976) found that 2.5% of a skeletal prehistoric population from Mexico had congenital fusion of vertebrae. Wade (1981) found five cases of cervical fusion among 29 native American (Kayenta Anasazi) skeletons dated AD 600-1200, all of whom were male, and two females with thoracic fusion. Wade noted that affected individuals often had lumbo-sacral border malformations, but did not distinguish any of these as being derived from a different developmental field. Merbs and Euler's Anasazi female (1985) had a fusion at C2-3 and a hyper-extended neck, reducing the width of the cervical neural canal. They suggested that she may have suffered neurological deficits. Schultz (1976) reported on two individuals from prehistoric Mexico with congenital fusion of vertebrae. Reed (1981) reported three more Anasazi cases with cervical and thoracic block vertebrae, as did Miles, with two cases (1975). Akins (1986) described an individual from Chaco Canyon with a C2-C3 level block. Palkovich (1980) reported an adult and two infants with cervical blocks from New Mexico material. One of the infants was 10-11 months and the other a neonate. Barnes (1994:71) described an Anasazi male with a block incorporating C7-T4. Other American cases have been reported by Anderson, (1989, cited in Barnes, 1994), Gregg and Gregg (1987) and Matthews (1891). Sture (1997) found fused elements in three females at Chichester (see fig. 2.8) and in five individuals at Raunds (see fig. 2.9).



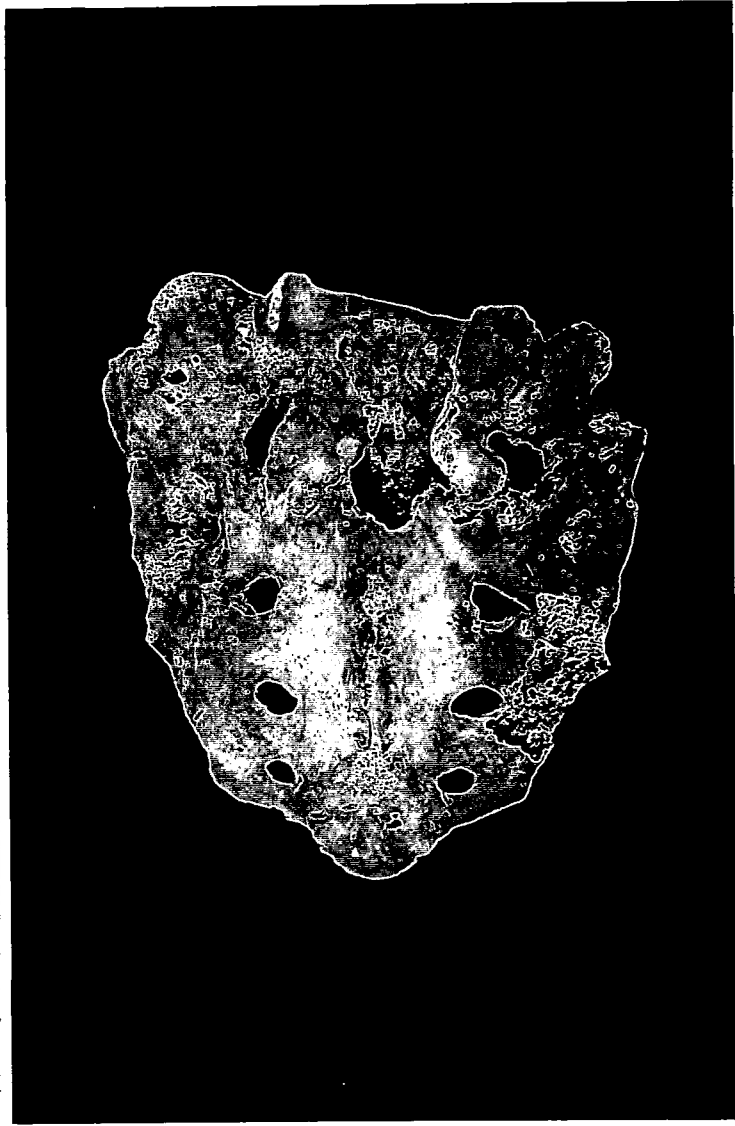


Figure 2.7: Partial lumbarisation of S1 with spina bifida occulta - a mature female from Chichester (C304)

### 2.3.ii.d *Segmentation errors - Cervical ribs*

Turner (1870) reported cervical ribs on cadaver specimens, and others followed, including Lucas (1915), Cave (1929, cited in Finnegan, 1976), Gladstone & Wakeley (1932), Brues (1946, cited in Finnegan, 1976) and Allbrook (1956, cited in Finnegan, 1976). Finnegan (1976, 1978) reported on a case of cervical rib with associated disuse atrophy in an Archaic American male aged 45-55 years. The left humerus was found to be less robust than the right, although bilateral cervical ribs were present. The right arm appeared unaffected, and the left forearm was of a normal size and shape. The defect may have been the result of soft tissue injury. Finnegan suggests that the individual must have been well-integrated into the group as his hunting skill would have been reduced. This does not account for his possible value to the group in other ways, however. Black and Scheuer (1997) reported a case of cervical rib from post-Medieval London. Barnes (1994:100) followed the classification outlined by Honeij (1920) which divided cervical ribs into four classifications depending on the size and shape, and commented on the likely neurological impairment which could result from neural compression of the brachial plexus which supplies the arms. She stated that 50 – 75% of affected individuals remain asymptomatic, but this is contradicted by other authorities who state that such individuals are the exception (Pizzutillo, 1983; Smith & Micheli, 1995). She cited cases from several Native American collections which had cervical ribs, including those identified by Denninger (1931), Palkovich (1980), Reed (1981) and several which she herself had identified among Southwest native Americans (1994:102).

Lumbar ribs are rarely reported in the archaeological literature. They are far less common than cervical ribs and appear to be always asymptomatic (pers.comm. RW. Smithells). MacCurdy (1923) described several cases from Peru, and Barnes (1994) found several cases among the Southwest native Americans.

### 2.3.ii.e *Segmentation errors - lumbosacral anomalies*

Border shifts at the lumbo-sacral border are the most commonly reported segmentation errors, and there appear to be population variations in frequency. Farkas *et al.* (1976) noted 8 cases of lumbo-sacral border shifting in Avar material (7 sacralisation and 1 lumbarisation). Bennett (1972) reported on high rates of lumbarisation among Modoc Indians associated with supernumerary vertebrae and spina bifida occulta of the extra vertebra (L6). He attributed the high rates of lumbo-sacral anomalies to genetic inbreeding, commenting on the typical settlement size of the Modoc, varying from 30-150 individuals at a time. Mulinski's observations of the Alpayeyma correspond to these findings. Both groups are located on the west coast of the USA and there may well be a genetic link. Merbs and Euler (1985) described atlanto-occipital fusion along with cervical fusion (C2-3) in a middle-aged Anasazi female, who also had spondylolysis. Goldstein *et al.* (1976) reported several cases of "synostosed" vertebrae among Bedouin groups which they said included sacralisation. Coyne (1981), Akins (1986), and MacCurdy (1923) all described cases of lumbo-sacral transitional vertebrae among Anasazi collections. Barnes (1994) found numerous examples of border shifting at all segmental borders in southwestern native American populations, as did, among these and other populations, Akins (1986), Anderson (1986), Bennett (1972), Coyne (1981), Gregg and Gregg (1987), Merbs and Euler (1985), Miles (1975), Palkovich (1980), and

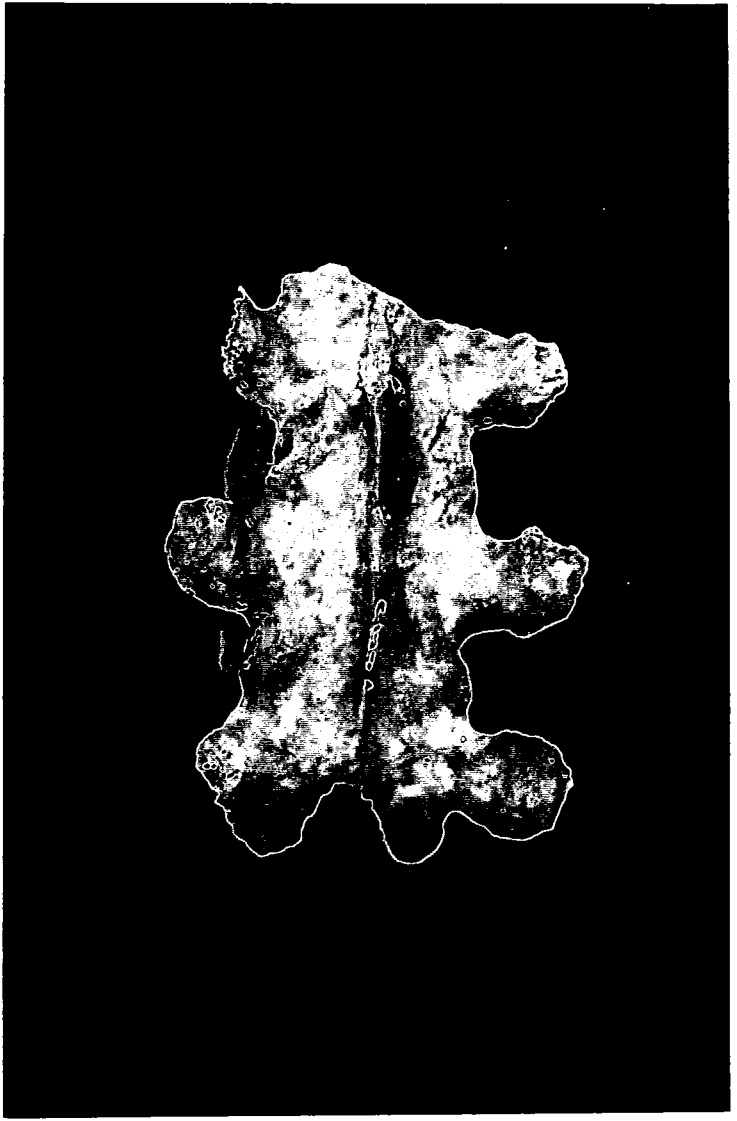


Figure 2.8: Fused thoracic vertebrae: adult female from Chichester (C137a)

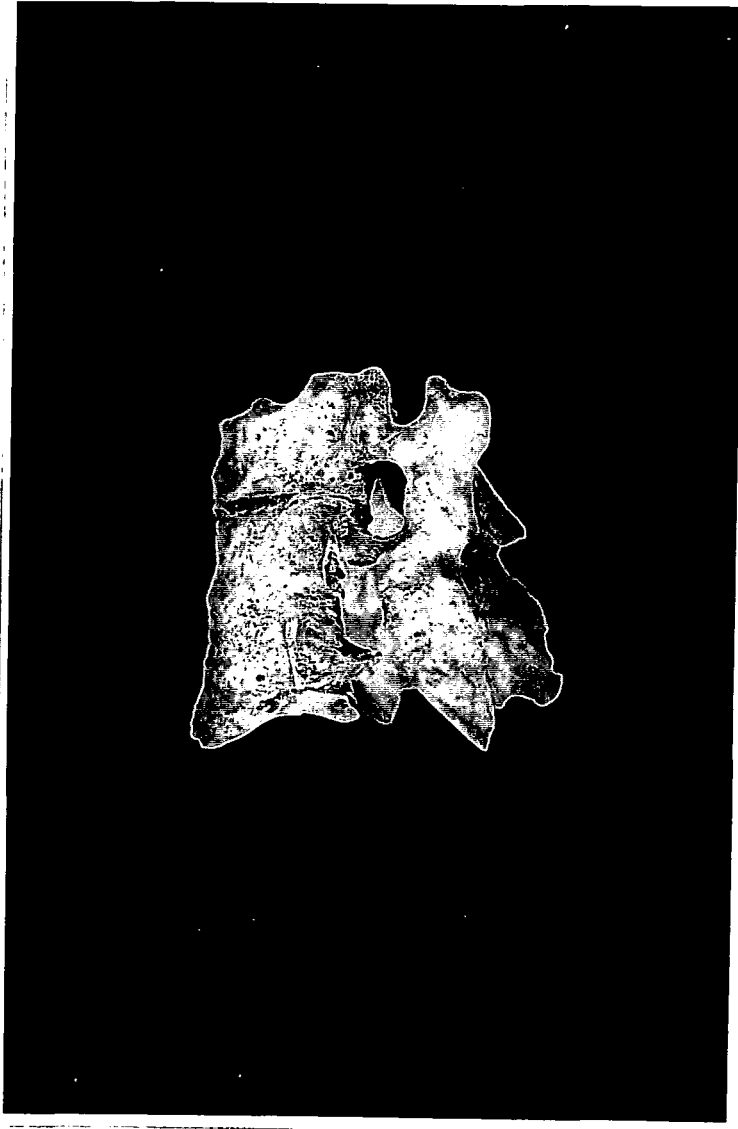


Figure 2.9: Fused cervical vertebrae: female adult from Raunds (5187)

Reed (1981). Black and Scheuer (1996) described a case of occipital-atlanto fusion in a nineteenth century female from St. Bride's, London. Sture found 48 cases of L5 sacralisation at Chichester (see fig. 2.11) and 20 cases at Raunds (see fig. 2.12). She also found 34 cases of lumbarisation of S1 at Chichester and 12 cases at Raunds.

### **2.3.ii.f**      *Segmentation errors - numerical variations in vertebral elements*

There are usually 24 pre-sacral vertebrae in the human spine. True numerical anomalies appear to be less common than apparent numerical anomalies. Many vertebral columns with an extra lumbar vertebra, for example, have fewer thoracic or sacral elements present, which is in fact simply a border shift, often without involving any abnormal synostosis of the elements. Genuine extra or absent elements result from an uneven number of somites in the blastemal stage, and may only be identified when either at least the whole segment is present, or the entire spine. Extra elements appear to be more commonly identified than absent elements, and, of these, the lumbar spine seems to be the commonest site affected.

Numerical anomalies in the pre-coccygeal vertebral column are usually mentioned in reports of individuals with other associated vertebral anomalies. For example, Bradtmiller suggests that numerical variation in the spinal column may be related to spondylolysis (1984:332), due to idiosyncratic stresses on the vertebral column.

Wells (1963) noted the presence of a sixth sacral element in one of three African sacra with pedicle anomalies. *Spina bifida occulta* was present in S1 in the affected sacrum. Usher and Christensen (2000) reported a young adult female from a Danish Medieval context who suffered multiple spinal anomalies including two extra pre-sacral vertebrae (thoracic and lumbar) and an extra sacral element. Also present were

block vertebrae (thoracic), extra ribs, border shifting at the thoraco-lumbar and

lumbo-sacral borders, and a deformity of the sternum. The authors of this report used Barnes' morphogenetic approach to analyse the origins of the defects, which made their work very useful, although it still focused on a single individual. Stewart

(1932) found true extra elements among Eskimo skeletons, and Allbrook (1955)

noted the same in a collection of East African origin, as well as some examples of shortened columns with only twenty three vertebrae in the pre-sacral spine.

Bornstein and Peterson (1966) reported on numerical variation in the Terry collection material, and made comparisons between Caucasoid, Negroid and Mongoloid groups, but this was largely unsuccessful because they discounted sacralized or occipitalized vertebrae. However, at least it was an attempt to make some sort of comparison on a population scale. Reed (1981) and Wade (1981) found extra vertebrae among southwestern Native American remains, as did Barnes (1994). Merbs (1974), Shore (1930) and Willis (1929) all reported cases from other American or African populations. Sture (1997) found examples of columns with absent vertebrae in six individuals at Raunds (see fig. 2.11) and in 22 individuals at Chichester.

### **2.3.ii.g**     *Cleft Defects in the literature*

It appears from the palaeopathological literature that facial or maxillary clefting was not common in antiquity, but this is more likely to be a result of non-identification due to inadequate understanding of the condition, than a true reflection of the frequency of the problem (Barnes, 1994). In addition, the popular view that cleft palate only affects the back of the mouth is a hindrance to diagnosis. The existence

of secondary cleft palate associated with cleft lip, or of cleft lip alone, has not been adequately addressed at all, certainly among European populations. Barnes identified a few cases of anterior maxillary clefting among Native American populations, but few cases have been identified previously among Old World populations save those mentioned below.

This is probably due to the misconception that cleft lip cannot be identified on bone alone. There are likely to be many more undiagnosed cases of cleft lip lying unrecognised in skeletal collections. Recognition in archaeological contexts may also be hindered by the occurrence of post mortem damage to the maxilla which is a fragile element, and by the effects of specific infectious diseases such as leprosy, tuberculosis and syphilis which can cause destruction or alteration of the maxilla.

Derry (1938) described two skulls with absence of the premaxilla, a formation defect related to cleft palate and cleft lip. Berndorfer (1962) described a 25-30 year old female Hungarian skull (c. fifteenth century AD) with a cleft lip. The premaxilla was rudimentary with the upper incisors being lost, and the nasal bones were assymetrical. No cleft palate was present. Berndorfer discussed the skull with a view to using it as a teaching tool for surgeons considering the optimal age for repair of cleft lip. Thompson Brooks and Hohenthal (1963) describe cases of defective palate formation from California, and also suggested the use of archaeological material in helping doctors to better understand cleft defects. Brothwell and Powers (1968) cited Elliot Smith and Dawson's 1924 review of a Nubian case, along with a 7th century Anglo-Saxon specimen from Burwell, Cambridgeshire; they also referred to three cases reported by Jorgensen in 1953 from a Greenland Eskimo group.



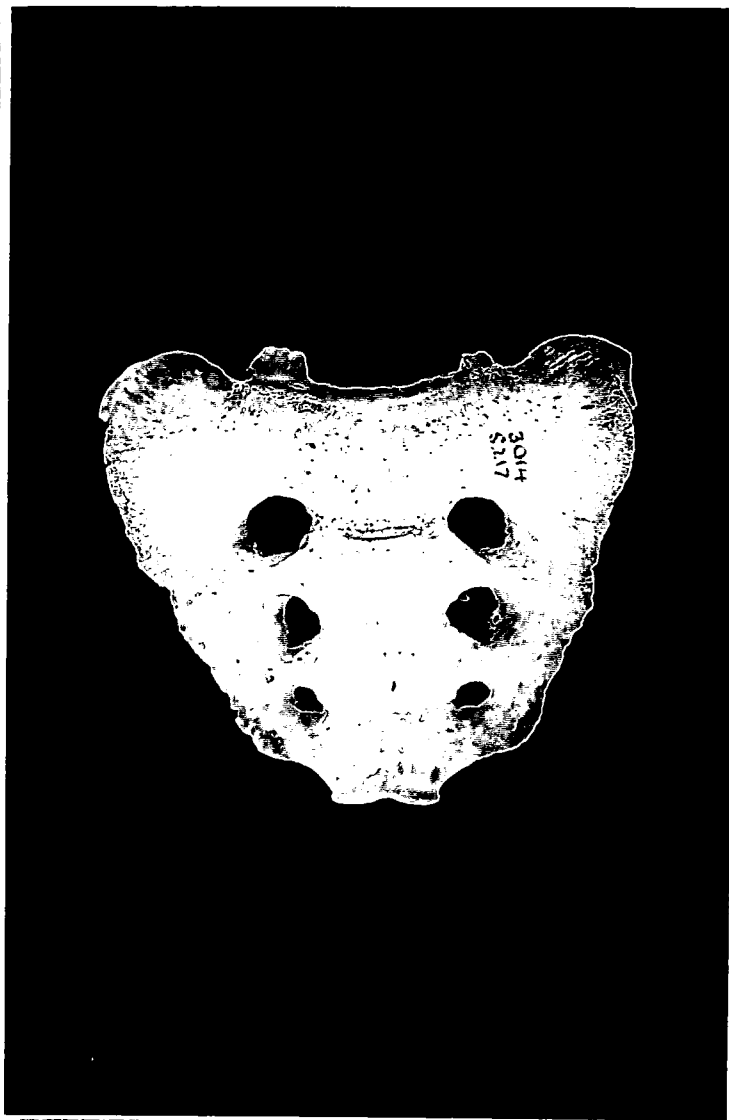


Figure 2.10: Absent vertebra (4 sacral elements only): a female adult from Raunds Furnells (5217)

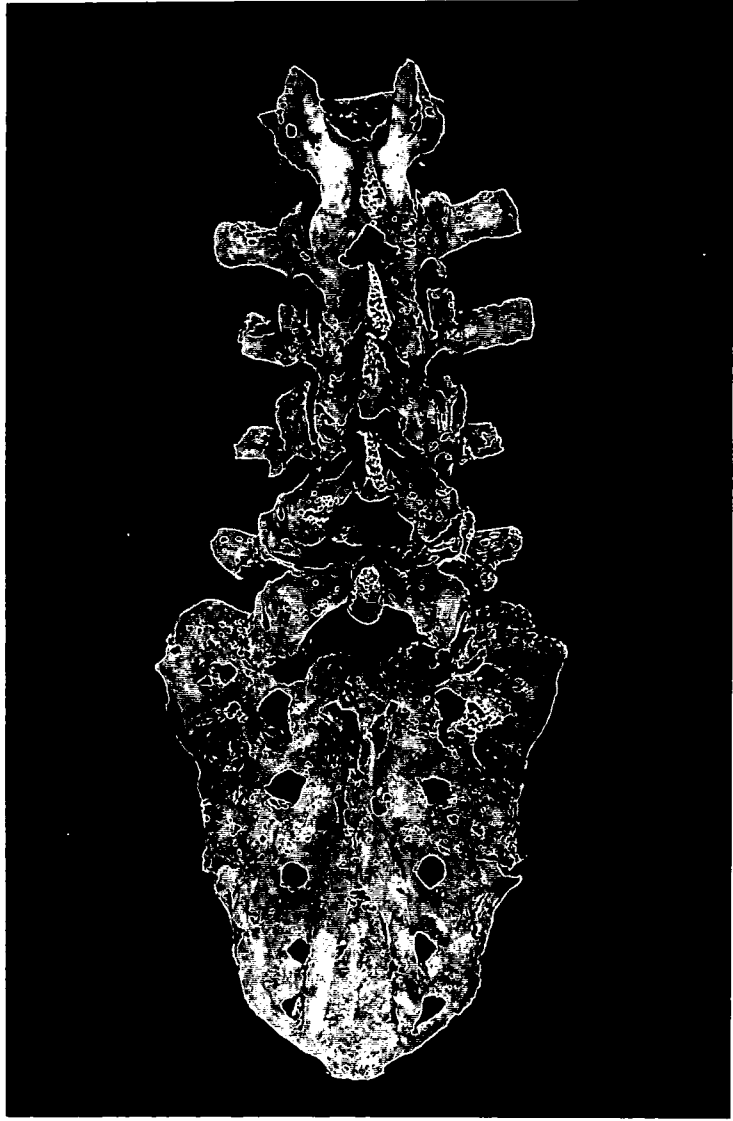


Figure 2.11: Supernumerary vertebra (6 sacral elements):  
a male from Chichester (C73)

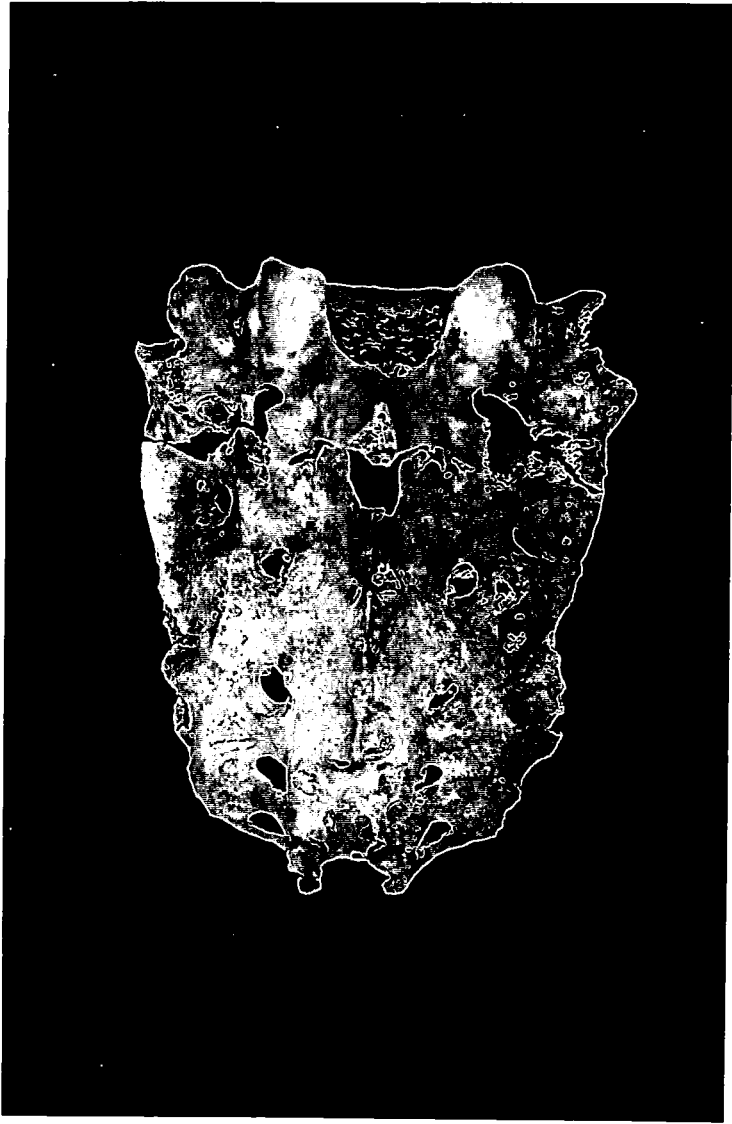


Figure 2.12: A vertebral column with segmental numerical anomalies (6 cervical vertebrae, and 6 sacral elements, with S1 being completely lumbarised): a male from Raunds Furnells (5202)

Fiori (1983) described cleft palate in an Egyptian mummy of the Middle Empire of Old Egypt (2445 - 1731BC), and also a small statuette of an actor with a cleft in the lip and nasal features. He also cited the earliest surgical repair of a cleft lip, in 390BC, by a Chinese surgeon on an 18- year old male peasant, which was apparently successful. Anderson (1994a) described a Medieval adult male of 40-50 years of age with a cleft palate and lip. He also cited documentary evidence of Anglo-Saxon surgery to a case of cleft defect involving excision of the edges of the cleft followed by suturing (stitching) with silk. Documentary evidence was reviewed for Medieval surgical intervention in later cases also, with patients undergoing surgery without anaesthetic. At least one individual survived for about twenty years after the operation, despite being unable to eat or drink easily for weeks after surgery. The outcome for the cleft repair is unknown (Anderson 1994:468-9). Anderson also noted the documentary evidence for Chinese surgical intervention (see above), and artistic representations of the condition dating to the 4th century BC. He also referred to the Huth Collection, a series of illustrative Elizabethan ballads in the British Museum, depicting "marvels of nature" including "monstrous births". Powell noted a possible cleft palate in a Raunds Furnells juvenile (1986), but on re-examination by Sture (1997), this was found to be a case of post-mortem damage. A further possible case was identified by Sture from Raunds, but the diagnosis is tentative, and may also be a case of post-mortem damage. Barnes (1994) described several cleft defects among Native American populations, including primary and secondary cleft palate, and cleft lip. She noted that there appears to be a nutritional link with the defect, and that unilateral cleft lip is more common than a bilateral defect among modern populations (1994:184). Ferguson (1977, 1978) showed that the embryological origins of the two forms of cleft palate (with cleft lip at the front of the mouth, or in isolation at the

back of the mouth) are completely different entities. He also stated that isolated cleft palate, affecting only the soft and hard palates, is 80% environmentally-related, as opposed to the aetiology of cleft palate in association with cleft lip, which is 80% genetically-derived. Ferguson points out that not all societies considered malformations “monstrous”, and mentions South American historical populations who believed that cleft lip and palate sufferers should be held in high regard, which may explain the relatively high frequency with which the condition is found amongst them in individuals who had survived the difficulties of infancy. Gilkey (1978) described three pre-Columbian skulls from Colorado and Peru. All three show asymmetric fusion of the nasal septum giving unilateral access between the nasal and oral cavities. All are presumably adults.

A selection of papers focussing on craniofacial anomalies appeared in 1987. These included examples of Treacher Collins Syndrome (Kolar *et al.*, 1987), Prader-Labhart-Willi Syndrome (Meaney & Butler, 1987), Apert’s Syndrome (Richtsmeier, 1987), and others featuring familial craniofacial defects and/or metrical analysis of them (Bookstein, 1987; Sudha *et al.*, 1987; Ward & Bixter, 1987). The publication of such papers *en masse* reflects the (arguably) current generally-held view among biological anthropologists that specific defects (when they are considered at all), particularly where they can be linked to recognised syndromes, are a suitable focus of birth defect studies among past populations. As such syndromes have typically very low frequencies even among modern populations, there is probably little to be achieved by actively searching them out among archaeologically-derived human material.

## **2.4 MEDIEVAL LIFESTYLES – A BRIEF SUMMARY FROM THE HISTORICAL LITERATURE**

As the review of the medical literature has shown, many of the birth defects relevant to this study are possibly derived from nutritional and/or infectious agents. By outlining the socioeconomic and environmental milieux in which Medieval populations such as those under examination in this study lived, this section aims to provide a picture of the variations of conditions which could give rise to differential exposure to pathogens and to nutritional impairment. For example, population density was greater in urban environments than in rural ones (Dyer, 1994:190), with the later Medieval period seeing the increased crowding of buildings and less open space in towns, with the associated latrines, cess pits and open drains in close proximity. Animals were attracted to rubbish, both vermin and farm animals (Dyer, 1994; Bond & O'Connor, 1999) increasing the number of methods of disease transmission in towns. As high population density brings with it increased rates of infectious diseases (Roberts & Manchester, 1995), populations living in urban environments may be expected to express higher rates of birth defects if the hypothesis of this study holds true.

There is a substantial literature available that describes Medieval English social, religious and economic life from the historical and archaeological perspectives. The entire body is too large to review here, but several sources have been consulted (and referenced in the text) to produce the following over-view. Living conditions, diet, occupational activities and economic factors, all of which affect health status, are considered below.

### **2.4.i Medieval towns and urban life**

The development of towns in the early Medieval period was dependent on the revival of the economy following the abandonment of Britain by the Romans in the fifth century. There were some centres which could be considered “urban survivals” from this period, often retaining their importance due to the presence of an ecclesiastical or commercial focus, such as York (Hodgett, 1972:55; Grenville, 1997). By the eleventh century, urban life appears to have been on the rise, probably because agricultural efficiency and output enabled this to occur (Hodgett, 1972:56; Hodges, 1982; Grenville, 1997). By the end of the thirteenth century, approximately 120 towns had been “planted”, or established decisively, for economic purposes in England. These towns included, for example, Ludlow, Richmond (Yorkshire), Bury St. Edmunds, Portsmouth, Liverpool and Stoney Stratford amongst others (Hodgett, 1972:57). Urban centres were distinguished from larger rural settlements by, among other factors, their lively corporate life, often focussing on ecclesiastical and religious centres, and the nature of the occupations of the inhabitants (Reynolds, 1977, Dyer, 1994; Schofield & Vince, 1994). The period of urban expansion seems to have ended by the late fourteenth and early fifteenth centuries, with ports and corporate towns dwindling in size and population (Hodges, 1982; Schofield & Vince, 1994). However, the economic system seems to have been maintained by the power of the urban corporate monopolies and vested interests (Postan, 1972; Schofield & Vince, 1994), maintaining towns and cities as major attractions for those in search of economic betterment.

Most towns depended on their role as regional distributors and marketers for their survival (Reynolds, 1977; Holt & Rosser, 1990), and the largest urban centres tended

to be those sited furthest from equal-sized competitors (Reynolds, 1977:59). York, Chichester and Hull were such places during the Medieval period, particularly until the late fourteenth and early fifteenth centuries (Reynolds, 1977; Goldberg, 1992). Reynolds states that only a handful of towns would have held populations in excess of ten thousand by 1300 AD, with many smaller ones managing populations of five thousand or less (Reynolds, 1977:62); nevertheless, these are still huge numbers compared to village population levels.

Documentary evidence tells us the types of crafts and industries which were focussed in towns, particularly the victualling (food and drink), metal, leather and textile and construction trades (Swanson, 1983; McGregor *et al.*, 1999; Morris, 2000). Trade tended to become woven into the political, religious and legal functions of the town or city. These functions drew people and trade into the urban centres and enabled them to flourish (Reynolds, 1977:64). Not all industry moved into or stayed in urban centres, however. A mix of rural and urban industrial locations was often employed. For example, cloth production is known to have largely moved into towns from its predominantly rural, domestic-level early Medieval production, but fulling mills required large amounts of water, and this was most readily available in the countryside. Hence, fulling mills tended to remain in rural areas, serving the industry in towns. Cloth was transported between the two localities in production, and returned to the towns for sale (Reynolds, 1977:59), ensuring contact between workers in both locations. Conversely, up until the twelfth and thirteenth centuries many towns contained within their boundaries open spaces for the production of foods, and in the early years of urban development, much food production was carried on within towns (Hodgett, 1972:56).



The textile, metal-work and leather trades were well-established in later Medieval York (Reynolds, 1977; Richards, 1993; Stocker, 1999). Most craftsmen are believed to have conducted all levels of production and selling of their goods themselves, from the procurement of the raw materials through the manufacturing processes and on to the selling of the finished article (Reynolds, 1977). Craftsmen in Medieval towns employed large numbers of individuals in their service to assist in all these processes. Unskilled porters, apprentices and journeymen (paid on a daily basis for work done) all appear in documentary records in urban centres and were largely drawn from outside the town (Reynolds, 1977:59; Goldberg, 1992: 86).

Pedestrians “set the scale of towns” (Hodgett, 1972:57). There was little wheeled traffic, with most people walking or travelling on horseback within the town. Streets were essentially used as a means of access to houses, and houses were considered more important than streets (Hodgett, 1972:57). Examples of narrow streets may still be seen today in places such as the Shambles, York. Most northern European towns displayed similar patterns of urban street design, with typical street widths of about 4.5 metres, with the surrounding houses crowding into the available space. Upper storeys tended to jut out across the street, reducing daylight below. Houses typically featured one or two rooms on the ground floor with the same on the floor above (Hodgett, 1972:57; Hall *et al.*, 1988; Dyer, 1994; Grenville, 1997). Properties were often long and narrow, with the short frontage facing the street, a pattern that reflects the Medieval burgage tenure. Many streets in York still demonstrate this pattern today, for example at Stonegate, which illustrates a continuity in the maintenance of property boundaries over many centuries. Craftsmens’ houses were typically of two storeys with a ground floor shop at the front where goods were sold, and workshops behind this. Living accommodation was upstairs. Yards at the back of the property

extended back in the long and narrow pattern and were often used for growing vegetables, keeping an animal, or for storage or refuse disposal. Wooden out-houses often occupied these areas (Grenville, 1997). Sanitation in towns consisted of latrines in back yards, and virtual open sewers, as available water courses became polluted with refuse. As cess-pit emptying was expensive, there was probably little incentive to have this done regularly until absolutely necessary (Dyer, 1994:209). Water courses within towns often became fouled with waste, for example the “Queen’s Dyke” near Aldwark, York. From the twelfth century onwards in Aldwark, excavations have shown timber and stone buildings with yards at the back full of rubbish in close proximity to wells (Bond & O’Connor, 1999:319). Public hygiene was further eroded by the practice of butchers, who would kill their animals in the street and leave the remains in the public thoroughfare. Civic regulations later appeared, enforcing butchers to clear up and restricting slaughtering to private yards (Dyer, 1994:191). Craftsmens’ areas in town generally developed into specific “quarters”, as more fellow-craftsmen came into the town from the countryside and congregated together. For example, the Shambles in York was the butchers’ quarter (hence the name).

By the twelfth and thirteenth centuries the open spaces within towns had declined and much residential building was thrown up to cater for the influx of workers. Many such buildings were of poor quality, or were allowed to fall into decay quickly. The tenements on Aldwark, York are a good example of this. Private landlords, in the case of Aldwark, the Vicars Choral of the Minster (Hall *et al.*, 1988; Richards, 1993), let the properties to poorer working people for whom such rented property was the only form of housing available. Rented accommodation for poorer workers was

typically a single room, often sub-let, and cramped (Dyer, 1994). Lady Row, Goodramgate, in York, is one of the few surviving blocks of this type which may be seen today. Although it looks picturesque now, it did not make for good quality accommodation in the fourteenth century (Grenville, 1997) and represents one of the poorer types of housing in York. Grenville points out that even poorer quality housing would have been found in the city, but such buildings have long since disappeared. Suburban areas (by Medieval standards) such as Bootham were noted for their squalor, with Bootham being infamous for the stink of pig-sties (Grenville, 1997:193). Apprentices were often housed with the craftsman's family, but pieceworkers or other "casual" labourers had to fend for themselves (Reynolds, 1977).

Much urban building work focussed on complex and rich buildings (Swanson, 1983). In rural areas a carpenter and thatcher were sufficient to construct a house, but in towns where large-scale churches, civic and legal buildings were required, as well as the houses of the rich merchant class and the town-houses of the nobility, a wider range of craftsmen was employed (Swanson, 1983). Skilled craftsmen such as carpenters, plasterers, and thatchers were in constant demand. Although some early town houses were constructed entirely in stone, such as the Jew's House in Lincoln, these were in the minority throughout most of the Medieval period. Houses were usually built in timber or timber and stone (Dyer, 1994; Schofield & Vince, 1994; Grenville, 1997). The rate of decay of timber and thatch in the English climate ensured constant employment for construction workers. Natural building materials such as these encouraged infestation with vermin, and were highly susceptible to damp, which would have impaired the quality of air inside the houses. There would

also have been less likelihood of rented accommodation being well-maintained than private or more well-off houses (Hall *et al.*, 1988; Grenville, 1997). Masons were usually employed in the construction and upkeep of the largest civic and particularly the ecclesiastical buildings such as York Minster. Various grades of workers were employed in the industry. Unskilled labourers such as hewers, mortar mixers and barrow-men came below an intermediate level of wallers or setters, who were in turn below skilled hewers, or free-masons. These worked on free stone. In addition, pavers, tilers, plasterers and brickmakers were all required in abundance.

The urban diet had the potential to be more wide-ranging than the rural diet, as the town acted as a market venue for farm produce and imported foods from a large catchment area, although it was subject to seasonal availability (Spencer, 2000), as was the rural diet. The best meat, for example, was generally sent to towns for sale (although only the better-off could afford it), leaving the lesser quality meat for the rural producers' own consumption (Dyer, 1994). Fruit consumption was generally higher in towns also, with greater amounts and types of fruit available for sale in towns (Dyer, 1994:197), including apples, plums, cherries, grapes, gooseberries and blackberries. Following the initial onslaught of the Black Death in the fourteenth century, the urban diet was, on paper, better than that of the rural peasantry. An Act of 1363 stipulated that the servants, artisans and tradesmen of towns and cities were to have meat or fish at least once a day, along with bread, cheese, and ale (Spencer, 2000). A typical day's food would consist of a breakfast of bread and ale, a midday meal of roast meat, stew, fish, or soup, plus bread, cheese, and ale, and a supper of bread and cheese, perhaps with some cold meat, and ale again. Water provision was usually polluted, so ale or beer were the drink of preference (Spencer, 2000).

Archaeological evidence of diet can be found in various sources which describe and quantify the remains of animal, fish and bird bones (for example, Bond & O'Connor, 1999). Such evidence can only be considered as representative of a general consumption within a population, not for specific individuals. Urban dietary quality and source was linked to changes in the urban environment during the Medieval period. In the early period (up to the early thirteenth century) open spaces in town enabled the production of foods. For example, the Bedern in York (adjoining Aldwark) was under light agricultural usage in the early thirteenth century, but this was lost in the fourteenth and fifteenth centuries as stone and timber buildings were constructed over the open areas (Bond & O'Connor, 1999:317). Animal remains from Coppergate, Fishergate and Aldwark included cattle, sheep, pig, domestic fowl and goose, with the relative proportions of these changing over the Medieval period at the Coppergate site (Bond & O'Connor, 1999:339, 373). Cattle numbers appear to have risen (34% - 64% fluctuation) as sheep numbers fell (19% - 30%) between the twelfth and fifteenth centuries, with pig maintaining a fairly regular proportion of the diet (c. 10-16%) as expressed by excavated bones. The Fishergate material (including fish and eel) came from latrines, indicating human consumption. The Aldwark site had evidence of butchery on-site, as well as cooking. In addition to the cattle, sheep and pig remains, horse, dog and cat were also present at this site (Bond & O'Connor, 1999:21-33). Some deer, fowl and goose were also present, with fish in only tiny amounts (Bond & O'Connor, 1999: 21-33). Social status also dictated diet, as evidenced by the differences between the bone deposits at the Bedern and other areas of York. The Bedern housed the College of the Vicars Choral of the Minster, and as such, was a higher status site than its neighbour, Aldwark. Here, there was a large

proportion of sheep, but documentary evidence shows that this was probably related to wool production rather than to diet. The Bedern community had its own beef source, separate from York market, and thus obtained a higher quality meat from this (Bond & O'Connor, 1999:363). In addition, the Bedern community also ate quantities of fallow and roe deer, and a greater proportion of fish (mainly marine) than elsewhere in the city, with fish increasing in importance in their diet as the Medieval period progressed (Bond & O'Connor, 1999:363). This pattern of fish consumption contrasts with the Fishergate fish bone pattern, where the fish were mainly of freshwater origin. The York evidence suggests that sheep were kept as wool-sources for between four and six years before slaughter (there must have been large numbers of lambs), whereas young pigs were eaten and older ones kept for breeding. Fowl, wild birds and geese bones probably originated from winter hunting on flood meadows, which is believed to have played a minor role throughout the Medieval period (Bond & O'Connor, 1999: 373ff).

The downside of the urban diet could arguably be that less food was prepared and consumed by the family unit (i.e. migrant workers may have had to rely on bought or provided food), although this is an assumption of the author's for which archaeological evidence is lacking. As many workers living in towns had left their families behind to seek economic betterment, they would have been reliant on food prepared from other sources. Court rolls tell us that prosecutions for the adulteration of foods and beer were commonplace, suggesting that the quality of consumables was a serious issue (Goldberg, 1992). Inadequate cooking and storage of foodstuffs would also have been a major hazard to health. Those selling food and drink for

profit would have had more of an interest in making money than in disposing of foods past their best.

Markets took place in urban centres on provision of a grant to hold a market from the king, bishop or landowner during the Medieval period. Not only did local craftsmen sell through the market but farmers and others from the countryside came to town every week to sell their wares. Fish markets, meat markets and others often specialised in produce (for example, Billingsgate and Smithfield markets in London, and Haymarket, Grassmarket and so on in other cities also). Many Medieval towns have a "Newmarket" - these are often in a widened main thoroughfare and take their name from the late twelfth-early thirteenth century charters which allowed for more market activity at that time to accommodate the larger economy and population (Hodgett, 1972:58). These markets served to draw in greater numbers of people to the urban centres and provided an economic basis for the expansion of the town or city, at the same time increasing the contacts between urban and rural dwellers. Cities which acted as ecclesiastical, political and legal centres always had large numbers of travellers and visitors passing through, which guaranteed a steady demand for services and goods. Added to the town's role as distributor and market-centre for the local region, this encouraged the growth and sustained the importance of the centre.

Town-based work offered by the textiles, metalwork and leather industries also attracted many people from the countryside. Amongst the 126 different occupations listed in the 1379 York tax records, were glaziers, goldsmiths, embroiderers, spicers, cooks, saucers (sold fast-foods e.g. pies and meats in the city), builders, masons, tilers, potters, plasterers, carpenters etc. The variety of work was vast, but conditions

of work were not controlled by the authorities in terms of health and safety as they are today. Metal workers (and their close neighbours), for example, would be exposed regularly to the hazards of metal dust and fumes.

Metal working involved the processing of lead, tin, copper and iron from all parts of the country (Hodgett, 1972:157; Hall *et al.*, 1988; Richards, 1993). Iron was particularly important to the economy as it was used for ploughshares, tools, cauldrons, axles, anchors, nails and arms. Miners were held to be of higher status than agricultural workers, and a way to obtain free status was to become a miner, or a craftsperson in a town (Hodgett, 1972:160). Metal from surrounding areas was sent into towns to be worked. For example, metal from the dales and the High Peak of north Derbyshire (lead) and iron from many areas, especially the West Riding, was sent to York for working. Recorded craftspersons related to metal work include cutlers, lorimers, spurriers, pinners, nailers, and bell-founders (Swanson 1983). Much of this work was done on a small scale with metal workers producing their own goods for sale from independent workshops. Metal workers in towns were also involved in smelting in small-scale furnaces, and working metal in hearths and ovens (Hodgett, 1972:162). The extraction of silver from lead was also commonly performed on a small scale. The fourteenth century development of the blast furnace increased pollution, with smoke and dirt even more of a hazard than previously (Hodgett, 1972:164). Foundries such as that at the Bedern, York, would have polluted substantial areas around them (Hall *et al.*, 1988; Richards, 1993).

The textile industry is typically the first to develop in industrializing economies and this appears to have been so in York (Hodgett, 1972:137; Goldberg, 1992). The



woollen and linen textile industries in York are known to have employed many single women and the majority of these are believed to have come from the outlying rural areas (Goldberg, 1992; Mays, 1997). Industries in cotton and silk were on a smaller scale due to the smaller demand, as these were expensive items (Hodgett, 1972:137; Goldberg, 1992). Surveys of contemporary documents reveal large numbers of single females whose occupation may be identified by their surnames, such as Kempster (seamstress) and Glover (Goldberg, 1992). Women were also employed in the production of textiles as spinsters (spinners), carders and websters (weavers) (Goldberg, 1992:21-22). Spinning was traditionally done by women using the distaff and spindle until the thirteenth century when the spinning wheel appeared (Hodgett, 1972:137). Weaving produced broadcloth, with two women seated at a loom, or narrowcloth, with one. Working hours were long and hard, being all “daylight hours” with a one and a half hour break during the day. Hence, in winter the working day was about eight hours and in the summer about thirteen (Hodgett, 1972:137). Fulling and tentering (stretching out cloth to dry after fulling) was a heavy job and often sited outside the town or in the country in order to use adequate clean water supplies, and for space. Dying involved the use of mordants of potash, alum and fuller’s earth without any consideration of the health effects of these. Weavers, fullers and dyers were considered “dirty-hand workers” and not allowed entry to the merchant guilds in some towns (Hodgett, 1972:137), suggesting that they were in daily contact with foul, poisonous or contaminated materials. Obviously, workers would have been greatly exposed to any pathogenic impurities in the wool.

Thus, urban centres provided a wide variety of work opportunities for residents, but at the price of frequent over-crowding in poor-quality living accommodation (Hall *et*

*al.*, 1988), poor sanitation, exposure to larger numbers of people and their pathogenic agents, poorer quality food, unsafe industrial practices (Goldberg, 1992) and less of the personal support from immediate family which does so much to enhance psychological well-being and a feeling of personal value (Brothwell, 1994; Ursin, 1994). As the immune system is increasingly believed to be mediated by perceived stress as well as by pathogenic agents (Hiramoto *et al.*, 1999), psychological factors may well have been a significant factor in the health of many urban dwellers, removed from the security of family and a small community.

#### **2.4.ii Rural and agricultural life**

Many agricultural techniques practised during the Medieval period had remained virtually unchanged since the Romano-British period (Hodgett, 1972:13). By the ninth century AD, the pattern of ploughing and crop-raising in northern Europe was established. The development of the heavy plough, which was capable of turning heavy clay-rich soils, enabled ploughing to be done two or three times a year. By the eleventh and twelfth centuries, the three-course rotation of fields (the three-field system) was commonly used (Hodgett, 1972:15), with lighter ploughs used for lighter soils, and sheep-rearing largely overtaking cattle herding (Welch, 1992). The common-field system was prevalent throughout western Europe during the medieval period, and comprised four elements: arable and meadow lands divided into strips; common pasturing in arable and meadow when lying fallow or after the harvest; common pasturage or waste where stock may be grazed, and timber, peat and other natural resources may be collected; and an assembly which regulated the system - usually the manorial court or a village meeting (Hodgett, 1972:166ff). Open fields and common agriculture remained the pattern in many areas. The heavy plough

required a team of four to eight oxen to pull it. This was a sizeable investment and was often shared by a village, enabling every family to benefit (Hodgett, 1972:15).

Crops were much as they had been for centuries before, although generally improved in quality. They included spelt, rye, oats and buckwheat, as well as barley. The latter ripens well in a wide range of conditions and was the staple ingredient in bread, beer, ale and porridge, as well as being used as a mash for horses (Hodgett, 1972). It could be sown in spring or winter and was therefore a versatile and reliable food source.

Vegetable crops included turnips, peas, beans and lentils, providing substantial amounts of vitamins, minerals and protein (Hodgett, 1972:22). Forest fruits were directly available to rural inhabitants, both for themselves and for their animals, particularly pigs, which pannaged in woodland. Rivers, streams and lakes provided a rich source of protein from fish. Meat was probably not regularly obtained by the killing of farm animals. The family cow, pig, sheep or goat would have been valued for its secondary products, and even in lean years would probably have been sustained in anticipation of the benefits it would confer when things got better – a practice employed in developing countries today (Harris, 1975). Protein was available from fish and pulses, which was fortunate as meat was not considered such an important part of the diet as it is today. Excavation has shown that cattle were the most predominant farm animals at Wharram Percy (and other Yorkshire rural sites), closely followed by sheep (Bond & O'Connor, 1999:415). The age of the animals at time of slaughter suggests that some calves (heifers and bullocks) were probably sent to town for sale, but that the majority of traded animals were adults (Bond & O'Connor, 1999: 415ff), leaving younger animals for consumption on the farms. The evidence from Wharram Percy suggests that a similar pattern was apparent with

sheep and that pigs were generally consumed young also (Bond & O'Connor, 1999: 415). In addition to the growing of food crops, the rural economy also benefited considerably in some areas from the raising of dyer's plants, including dyer's weed, woad, madder, saffron and teasels, as well as the cultivation of flax and hemp for woven textiles and cordage. Marshy areas also produced osiers for fencing and rushes for matting and lighting (Hodgett, 1972:22).

The manorial system declined during the fourteenth century, not only because of the plague, but due to factors already in train before this, such as marginal arable land failure, a succession of poor harvests and a worsening climate. At the same time the economy was becoming more money-based, rendering labour services less valuable (Postan, 1973:89).

#### **2.4.ii.a**      *Lifestyle of peasants*

Hodgett estimates that one knightly family with its own manor and lands would require between fifteen and thirty peasant families to provide the necessary support (1972:167). The "peasant class" consisted simply of those who were not nobility or landed gentry. Thus the majority of the population were peasants. The term itself did not imply poverty. Peasants owed various duties to the lord, for example, "week-work" which involved putting in several days work regularly on the lord's demesne (area of the manor); or "boon-work" which included extra days working for the lord such as during the harvest (Hodgett, 1972:168). These duties varied over time, and many were commuted for rent from the eleventh century onwards, almost totally disappearing by the late fourteenth century (Hodgett, 1972; Postan, 1973:52). By the thirteenth century, however, they began to be called back into practice for a period. In

other words, a peasant had to spend some of his labour on lands which were not his own, and expend energy towards crops which he would never eat.

Medieval peasant houses used to be considered insubstantial constructions of poor quality, but a reassessment over the last twenty years now reveals that many rural families probably lived in stone and timber constructions of a variety of forms (Aston, 1989; Grenville, 1997). At Wharram Percy the two toft excavations have revealed longhouses of a substantial nature, with cross passages and hearths (Milne, 1979 a and b). The general assumption has been that animals occupied one half of such houses, at least in winter. Welch states (1992) that this was not necessarily so, but was considering settlements far south of Wharram, so the harsher northern climate may well have entailed the over-wintering of some livestock in the houses for at least part of the time. Grenville says that the byre end of the house may well have undergone a change in use over time, from byre, to agricultural store to extra accommodation (1997:137). However, the houses were relatively dark, with small windows, probably a central chimney, and despite regular sweeping, probably a smokey and damp atmosphere.

Medieval farming was very labour intensive. The three field system was commonly used but this did not leave adequate fallow time for the land, and as population increased, so did the pressure to produce enough food. Some areas practised a two field system, in which half of the land was left fallow every year, but again, population pressure (on a national scale) made this difficult (Hodgett, 1972:184). During the thirteenth and early fourteenth centuries population pressure was “very severe” (Hodgett, 1972:197), with the rural population probably as high as in the

early eighteenth century. A large number of people were living on the edge of starvation during this period, and mortality was high during years of poor harvest (Hodgett, 1972:197). Manuring was the best form of fertilisation, adding large quantities of nitrogen to the soil, but there is evidence that at times during the Medieval period the practice declined (Hodgett, 1972:184). This is interpreted as an imbalance between arable farming and livestock rearing - too little manure was available. Alternative fertilisers included seaweed and the practice of burning the vegetation (Hodgett, 1972:184).

The dietary staple for most peasant communities was black bread, made from rye or mixed corn. Accompaniments to bread were probably few for the majority of peasants during the early Medieval period (Hagen, 1999:355), consisting of a small amount of meat or offal, and fats, and cereal stews (all on a seasonal basis). Porridge and gruel made up a substantial part of the diet. White bread was a feature of the last three centuries of the Medieval period (c. AD1200-1500) but was only eaten by the urban or rural rich (Hodgett, 1972:194). From the twelfth century onwards, legumes played a greater part in the diet. Peas, beans and vetches were not only good for human consumption but also made up good feed for animals and provided a greater amount of manure for the fields. Vegetable-based stews could remain in the pot over the fire for days (Spencer, 2000), being added to and taken from as necessary (hence, “pease pudding hot, pease pudding cold, pease pudding in the pot, nine days old”). Obviously much of the nutrient value of such stews would be lost before being consumed. When possible, fats from butter (sheeps' milk) or from oily seeds such as linseed, were added to improve the taste and nutrients where available (Spencer, 2000). Leeks, lettuce, onions, spinach, kale and cabbage were also typically grown

by peasant farmers, and these were added to the pottage. Protein was available in curd cheese, whey and eggs for the peasant population (Spencer, 2000). Herbs such as mint, camomile, fennel, garlic, parsley, rue, sage and thyme were available for flavouring food. Fruit crops such as apples, peaches, pears and cherries were raised with success in various areas (Hodgett, 1972:194). In the earlier Medieval period (as represented by the Raunds population in this study), food could be scarce seasonally, as food production was more precarious (Hagen, 1999:354); there is evidence that women and girls were subject to discrimination in the distribution of available foods during such times (Hagen, 1999:354) and this would have had an impact on the reproductive health of undernourished women (Allen, 1986).

Demographic changes substantially affected the life of the peasant. The years 1314-17 had already been famine years, and the fourteenth century went on to be characterised by the deteriorating climate that had adverse health effects. A reduced amount of sunshine must have led to Vitamin D deficiencies (Hodgett, 1972:202), and the increasing dampness must have raised levels of infection and exacerbated arthritic complaints. Damp in houses must also have increased. The Black Death arrived in England in 1348 and maintained a presence in the population for the next three centuries (Hodgett, 1972:199-200; Kiple, 1997). The initial onslaught is believed to have killed one third of the population of England, with up to 40-50% losses in some local areas (Carmichael, 1999:61), leading to a huge drop in the available workforce. Labourer wages rose significantly as a result, and the surviving poor did very well in consequence. However, three subsequent epidemics of the plague in the same century slowed the demographic recovery (Hodgett, 1972:200; Kiple, 1997).

By 1380 a surplus of land was in evidence, with too few people to farm it. Much land went out of agricultural production and a sizeable proportion of arable land returned to pasture (Hodgett, 1972:206). This led to a wide disparity in peasant landholdings. Labour was expensive as the demand was high. Peasant landholdings only increased if there was a family big enough and vigorous enough to manage it and keep labour costs down (Hodgett, 1972:208). During this period, with high wages and a demand for their labour, peasants did very well, with a greater income than before and the ability and opportunity to spend it on an increasing amount of consumer goods and better clothes (Hodgett, 1972:213). This in turn supported the expansion of towns, with their craftsmen and markets. Eventually a higher standard of living was enjoyed by peasants farming on good land. These people enjoyed a higher-protein diet, eating more meat and dairy products, presumably because the pressure to expend energy on arable crops was reduced slightly and they had a higher income to spend on better food (Hodgett, 1972:213). This was typical of the changes in food consumption patterns that occurred throughout the Medieval period. For example, in the late fourteenth and early fifteenth centuries, bread consumption fell and legume consumption rose, along with the rise in consumption of dairy products and meat. Within this picture, beef consumption increased, but pork consumption fell (Hodgett, 1972:198).

#### **2.4ii.b**     *Economic changes for peasants in the Medieval period*

Generally speaking, prices rose in the early medieval period and tended to fall during the later period, reflecting agricultural prices, which tended to be higher up until the fourteenth century, and lower after this (Postan, 1973:7). These facts accord with the



drop in population after the epidemics of the Black Death, although there was a recognisable trend of falling population even before the early fourteenth century (Postan, 1973:12). Economic benefits did not always pass directly to the peasant class. Agricultural and industrial price movements did not always synchronize. During the twelfth and thirteenth centuries industrial prices did not rise as high as wheat prices for example, but neither did they fall or remain steady in the later Medieval period (Postan, 1973:9). In effect, the economy for the average peasant during the Medieval period was fairly unsteady, with the economy reacting to, and being driven by, the demographics of the population. The periods before and after the epidemics of the fourteenth century were characterised by fluctuations in population levels, economic demands and agricultural output. Following the epidemics of this period, there was a contraction of agricultural effort, with much land being abandoned and a period of agricultural depression setting in (Postan, 1973:13). The worsening climate and the poor harvests of the first two decades of the fourteenth century paved the way for the problems experienced following the epidemics (Postan, 1973:13).

In rural areas, the increased costs to farmers incurred by the rise in wages for labourers lead to some cutting back on employment. Added to this, the labour-intensive farming practices which had developed in the pre-plague era were adapted or abandoned because the food requirements of the post-plague population were not as great. Farmers, therefore, released many men and women from agricultural labour, freeing them to look for work in towns, but removing a source of income from those who had to stay in the country.

Postan suggests that some of these pressures may have been exacerbated by changes to the family structure during the early Medieval period. He suggests that the nuclear family may have begun to replace the extended family, and resulted in more numerous individual landholdings (Postan, 1973:16). The social effects of this would have had implications for the health of the old, the young and the sick, with the members of smaller families having to work more in order to produce enough food, and having less time or opportunity to concentrate on childcare and nursing.

#### **2.4.iii Women in the later Medieval period**

As the bearers of children, womens' health and associated lifestyles and opportunities are of particular interest in this study. However, interpreting the fortunes of Medieval women, affected by the vicissitudes of rural and urban economies during the pre- and post-plague periods, is described by Goldberg (1992:8) as an "intellectual minefield". There is limited source material on the lives of urban women, as they are largely "hidden" by their husbands in documentary records, unless they were single or widowed. Rural women are easier to study because they are better represented in the literature, but this is complicated by the fact that married women had a different legal status from unmarried ones (Goldberg 1992:83-84). However, there is much documentary evidence of female traders in towns from the 13<sup>th</sup> century onwards, particularly in relation to market trading. Spinning, laundering and traditional female tasks were expanded in the late 14<sup>th</sup> century into wider employment (Goldberg 1992:86).

Documentary evidence, for instance regarding wages and the increased appearances of women in court rolls and poll tax returns, is also a useful source of information,

showing that economically-speaking, women's standing in society could be said to have undergone a change as a result of the demographic effects of the Black Death from the fourteenth to the fifteenth centuries. However, despite the mention of women in documentation of the period, there are problems with placing too great a reliance on such records (Goldberg, 1992:82). Apparent data are frequently distorted due to women not being recorded in their husbands' records. If the husbands' businesses were not recorded adequately for any reason, then there is no possibility of accounting for the associated women involved. Many women appear in court records when presented for prostitution, or debt, and can only be followed through such entries, often across many years. Legal regulations for debt and prostitution were stringent, and prosecutions pursued vigorously, so women falling short of the standard in these areas were highly likely to appear. Women were heavily represented in their own right, however, in the brewing, bakery and petty retail trades, and appeared often in court rolls and mentions of business dealings in relation to these.

Undoubtedly the plague had a significant effect on the fortunes of women in Medieval society. In both town and country, their value as a labour force was enhanced by the general shortage of workers following the various episodes of plague. For instance, York experienced an acute shortfall of labour in the years immediately following plague visitations and as a result, wages paid to available labourers rose greatly. A greater proportion of money in the hands of the poorer members of society led to an increase in demand for manufactured goods, which in turn led to more employment in the city producing goods to meet the demand. In this way, York, like many other Medieval towns and cities, recovered economically from the plague (Goldberg, 1992:7). Rural women were more active on the farmland

in the post-plague period, at least during harvest-time, probably due to the labour shortage. This would probably affect mothers and older women mostly, if the younger, freer ones were able to go off to work in towns. Much labour of rural women would have been unpaid, for instance within the family. Rural women were heavily involved in spinning and weaving, which would entail contact with dirty fibres, but on what basis this was done (domestic or "commercial" for the whole village) is not certain (Goldberg, 1992:12).

During the endemic period of the plague, women were increasingly absorbed into the general workforce to make up numbers (perhaps a similar situation to the requirement of female labour during the two World Wars of the twentieth century), and this involved the migration of many women into towns and cities in search of well-paid employment in manufacturing industries. It is believed that male and female workers were probably paid the same wages during this period (Goldberg, 1992:12). Documentary evidence shows that women increasingly delayed or avoided marriage, and the birth rate appears to have fallen (Goldberg, 1992:7) - or at least recorded births fell. Increasing numbers of older mothers producing babies would have led to an increase in infants born with birth defects due to the maternal age factor. Women were effectively liberated from dependence on marriage to survive, as many were able to be self-financing by virtue of the value of their own labour. It should be noted, however, that the mass demographic movements associated with the post-plague period were not solely rural-to-urban in direction. There was also significant migration from one rural area to another, with farm workers and their families often travelling considerable distances to meet the demands for tenants and labour in areas which had been particularly badly affected by the plague (Razi,

1980:117). However, the mid-fifteenth century (approximately one hundred years after the first visitation of the plague) saw the demographic profile of the country reach such a low point that the growth in the recovering economy could no longer be sustained, and the status of women began to revert to its previous situation. As the male population increased the labour shortage fell, and men appear to have had to fight for their own employment and position in society. This effectively reversed the trend for female employment, with women excluded from the labour market by various means, and strongly demarcated divisions between “male” and “female” work roles developing (Goldberg, 1992:7).

## **2.5 INFANTICIDE - ECONOMIC, RELIGIOUS AND CULTURAL ASPECTS**

Our views on infanticide are influenced by the culturally embedded ideals that our society determines for us (Lee, 1994:65). Infanticide has not always been considered “bad”, as past societies (and some modern ones) viewed the practice as an economic or religious necessity, with different constructed roles for parents and children (Lee, 1994:65). It is a commonly held view that deformed individuals were probably “eliminated” as infants by past cultures due to superstitious beliefs. Archaeological evidence suggests that this is not strictly true, with various examples of individuals surviving into childhood or adulthood (Barnes, 1994; Webb & Thorne, 1985; Chadwick-Hawkes & Wells; 1976). It could be argued that cultural perceptions of such individuals varied between societies, allowing, for example, the survival of individuals with visible cleft defects in one culture but not in another. These tolerance differences may have been strongly influenced by religious ideals or beliefs, or these may have been used as a “smoke-screen” to hide infanticide as an economic necessity. Traditional populations today appear to have differing views regarding

infanticide. For example, Blainey (1976, cited in Webb & Thorne, 1985) stated that infanticide was an automatic and universal response to malformed children among nomadic groups. This statement is contradicted by findings from various parts of the world, however. Australian aboriginal remains include adults with cleft defects, dwarfism and club foot among other conditions (Webb & Thorne, 1985), and several Native American skeletal series contain individuals with cleft and other obvious defects (Barnes, 1994; Jorgensen, 1953; Thompson Brooks & Hohenthal, 1963). In modern contexts, as it probably was in the past, infanticide appears to be influenced principally by economics. As recently as the late nineteenth century, female infants in India, Japan and China were frequently killed at birth if the economic situation did not favour the presence of girls in the family at that time, and the practice is said to continue in these areas amongst the poor today (Harris & Ross, 1987). The Chinese one-baby per family rule is also leading to the selective infanticide of girls, who are perceived to be less economically valuable than boys. This is leading to a significant reduction in the number of females in the population of certain areas and could have severe demographic effects, resulting from a principally economic-driven behaviour.

Infanticide as practised among the poorer sections of some societies today appears to be related to the requirements for child labour at any given time, whereas richer families view the survival of female infants in the light of land control, marriage and inheritance issues (Harris & Ross, 1987:97). Clearly, some of these considerations are pertinent to the Medieval period, to both richer and poorer families, particularly during the time of economic and demographic recovery following the onslaught of the Black Death in the fourteenth and fifteenth centuries (Chambers, 1972).

Limitation of family size during such periods would be occasionally necessary for the

survival of those already living. The majority of families in under-developed or transitional economies have long been recognized as living at or near the subsistence level (Lorimer, 1954), and recovering economies such as that in England after the plague had decimated the population must have been similarly classified. It has also been suggested that illegitimacy rates rose in the post-plague era, and that fines for illegitimate births were levied on females producing such offspring (Razi, 1980:138). Clearly this could be seen as an inducement to poor women (and poor families) to conceal unwanted births - how much more so if the infant was obviously deformed. Illegitimate births to older widows in the post-plague era rose, a fact that Razi attributes to the new opportunities for dowerless young girls to marry at an early age, thus leaving many widows with fewer chances of remarriage than would have been the case pre-plague (Razi, 1980:139). This rise in recorded illegitimate births to older women would probably have also produced a higher rate of infants with birth defects due to increased maternal age (Levene & Tudehope, 1993), and the figures therefore probably represent only a percentage of actual infants born to these older mothers. Alternatively, child labour has long been recognised as an important element in the welfare of the family in agricultural communities, with many tasks such as grinding and pounding nuts and seeds, weeding, hoeing, bird-scaring, collecting natural resources, and a variety of domestic and child-care duties falling to children from an early age, freeing the older members of the family for heavier or more complex work (Harris & Ross, 1987: 39f).

Female infants in non-industrial or developing countries today are the most frequent victims of selective infanticide (Harris & Ross, 1987). Along with this, females are also frequently the victims of nutritional discrimination (Lindenbaum, 1977). During

times of economic stress, including periods of under-availability of adequate food supplies, females in the past may well have received less food than males, a situation which continues today in many under-developed countries (Harris & Ross, 1987; Lindenbaum, 1977) and has been recorded in historical populations (Hagen, 1999). This nutritional discrimination is believed to have affected female children as well as female adults, thus setting up a pattern of female undernutrition which must have jeopardized the reproductive health of the population in the past just as it does now (Allen, 1986). Girls within such communities today suffer a higher mortality rate than boys, which may be at least partly related to this nutritional discrimination (Harris & Ross, 1987:42), although cultural attitudes towards health care (professional or domestic) may also have a significant sex-based effect. For example, Piers (1978) documents the selective indirect infanticide (by neglect) of girls rather than of boys, in impoverished South American communities.

It seems reasonable to suggest that the practice of infanticide was probably common in England during the Medieval period, as the country underwent similar economic and social fluctuations to that experienced by non-industrialized societies today, as well as suffering the devastating effects of the plague. Infanticide in the English Middle Ages was, of course, illegal, but the practice could easily escape detection (just as it does today). Overlaying, or the “accidental” suffocation of a baby sharing the parents’ bed, would be virtually impossible to prove as a premeditated act; exposure may have feasibly escaped undetected during long winter nights or cold days, and neglect by wet nurses was common (Chambers, 1972; Harris & Ross, 1987; Piers, 1978). Razi, using contemporary records from Medieval Halesowen, noted a discrepancy in family sizes depending on the amount of land available for



families to farm (1980:143-4). One conclusion from this observation could be that the families farming smaller acreages may have been routinely practising infanticide in order to survive, as in all other respects the parents were living under the same economic and social conditions as their neighbours with more land. Razi comments that during the pre-plague period the children of richer families within the village survived at a higher rate than those of poorer families in Halesowen, and that post-plague, fewer children of richer families survived into adulthood (1980:144-6), but does not consider the possibility that poorer families may have always had to practise infanticide periodically and that richer land-owning families post-plague may have been considerably worse-off as a result of the higher costs of labour during the post-plague period. Lee states that richer families in the past, having access to better food, may have had higher fertility rates and have had to practice infanticide more regularly than the poor as a normal state of affairs (Lee, 1994).

Demographic studies by Chambers based on post-Medieval parish registers have been shown to include only a proportion of actual births. Stillbirths were not usually entered into the registers, and those surviving only a few hours may have been entered as a "chrisom child", but there are suspiciously few of these recorded. Chambers points out that this situation takes no account of abortions (spontaneous or induced) or infanticides, and is therefore a poor record of the actual number of conceptions (Chambers, 1972). In view of the fact that the Church must have been aware of the practice of infanticide, and that economic considerations could be harsh, it does not require a great leap of the imagination to consider that deformed babies could have been relatively easily disposed of by the parent(s). The notion of children being acknowledged as a distinct social entity appears to have been the norm by the

late Anglo-Saxon period, with children being considered worthy of separate burial from adults in many burial grounds (Lucy, 1994). This would have probably placed a greater strain on families “killing” their children.

In the late eighteenth century it was not unusual to see the corpses of infants lying in the streets or on dunghills (Langer, 1972: 98), a practice which may well have been of longstanding. Evidence from sewers beneath a Roman period bath house/brothel in Ashkelon revealed the presence of up to one hundred infants, interpreted as the victims of infanticide (Faerman *et al*, 1998). In the late twentieth century newborns are still being disposed of in similar ways, with dead infants routinely discovered in North American city sewer systems or rubbish bins (Piers, 1978; Lee, 1994). The Ashkelon evidence indicates the selective saving of females, which accords with Roman documentary evidence of the rearing of girls as courtesans for work in brothels. Similar sex-selective infanticide is also documented in many developing countries, particularly in shanty towns around huge cities. In South American *barrios*, innumerable impoverished parents have been documented as practising indirect infanticide on the seventh or eighth child, usually a female, and often dark-skinned. This is interpreted as an economic decision, boys having more to offer economically than girls. By neglecting the unwanted child it falls prey to malnutrition, stunted growth and developmental delays, both emotional and mental. By the time the child dies of neglect, such a distance has grown between the child and the family, who consider it to be almost non-human, that the death is not seen as a great tragedy (Piers, 1978).

Cultural responses to stress are many and varied, but often appear irrational or unusual to the modern western eye. Infanticide as a means of population control is not the only anomalous method practised throughout human history. For instance, populations under stress due to population pressure (whether due to high birth rate and /or low mortality, lack of resources, or both) are known to temporarily adopt changed behaviour in order to counteract the causes of the stress. Documented changes include a community shift from heterosexual to homosexual relations for a period of time, or for a certain number of days of the year, in an attempt to reduce the birth rate (Harris & Ross, 1987). Other recorded responses have included the rise of culturally-lead misogyny, by which women are considered as “polluting”- this is believed to be the origin of many Medieval witch-hunts, the vehemence of which was predominantly associated with times of economic stress in the later Middle Ages when population pressure was high and agricultural output low (Harris & Ross, 1987: 66f).

Added to these “irrational” responses to stress, the popular belief in the sick being “cursed by God” or of abnormalities being related somehow to occult powers (Dryden, 1978; O’Shea, 1995) was a strong influence within society during the Medieval period. Religion was heavily tainted by superstition (Daniell, 1996) and there was no general understanding of the biological processes involved in reproduction which could result in congenital defects. It was generally accepted that the outward form of the body reflected the condition of the soul (Daniell, 1996), therefore any visibly deformed infant would have been considered spiritually unclean, and “non-Christian”. Daniell states that “sudden death was feared because the lack of preparation was possibly injurious to the soul in the afterlife” (1996: 71).

If, however, the soul was already considered beyond redemption, sudden death would seem appropriate - the idea that “bad” people died suddenly was well established. In addition, the mode of death (i.e. with or without preparation) symbolised the spiritual standard of the individual’s life, and was considered to indicate the destination of the soul - to Heaven or Hell - so there exists a circular argument by which an individual dies because he is evil, but is evil because he dies suddenly (Daniell, 1996: 71).

Those infants born with visible deformities such as cleft lip and palate, or extra digits, may have often attracted unwanted attention, and perhaps been killed or allowed to die as a means of removing a “malevolent” presence from within the family and community. Likewise, the belief that the spilling out of internal organs was related to evil (Daniell, 1996:83) - there are numerous examples in the Bible of this fate befalling evil men - would have impacted on babies born with conditions such as *spina bifida cystica*, which involves the exposure of the meninges through the lower back. The prime candidacy of deformed infants for this kind of treatment may explain the paucity of cleft, cranio-facial and severe neural tube defects in archaeological populations, although a proportion of infants affected by cleft palate may not have survived infancy anyway due to an impaired ability to suckle, and a predisposition to continuous ear, nose and throat infections (Bacher *et al.*, 1996).

Infants with myelomeningocele would not have naturally survived longer than a few hours or days, but may have still been the victims of infanticide if they were considered “evil” due to their exposed internal features. The opportunity offered by religion to consider such infants as being the products of evil may well have eased the parents’ role in practising infanticide - this ability to “separate” from the infant enables the poverty-stricken parents in poor countries to “allow” their own children to die and is likely to have similarly empowered Medieval parents (Piers, 1978).

It is suggested today that childhood mortality from disease, along with direct or indirect infanticide (active or passive killing) would have been the most effective means of controlling family size in the past (Harris & Ross, 1987), and that the active control of family size was directly related to economic conditions. Sex-selective infanticide is well-documented and recognized in modern as well as historic contexts (Harris & Ross, 1987; Faerman *et al*, 1998; Lindenbaum, 1977). Religious belief and superstition played a significant role in relation to matters of life, death, and spiritual value in the past (Daniell, 1996) and were a crucial determinant in the fate and perceived value of adults and children alike. Therefore, when considering the fortunes of many deformed babies in the past, the likelihood of infanticide should be taken at the very least as a distinct possibility, if not a likely “predestined” fate. The implications for archaeologists involve a lack of the more obvious deformities in the archaeological record, especially in the formal contexts of burial grounds. Not only may deformed infants have been excluded from burial in holy ground due to their perceived spiritual status but also due to their illegal manner of death. Parents would not be in a position to either register the birth, or to mark the death in any public way. This accords with the current situation in the study of congenital defects in archaeological populations. Birth defects involving obvious soft tissue manifestations are very few, whereas those defects which are invisible, such as vertebral anomalies and minor clefts at the back of the mouth, are represented relatively well (for instance, Barnes, 1994; Sture, 1997). If this hypothesis, that many obviously deformed babies were victims of infanticide and those with invisible defects were not, holds true, then we may expect to see many of the latter in the archaeological record.

## CHAPTER THREE

### Embryology and anatomy of the axial skeleton

#### 3.1 EMBRYOLOGICAL DEVELOPMENT OF THE VERTEBRAL COLUMN

This section is based on information taken from Larsen (1993), Barnes (1994), Moore (1978) and Moore and Persaud (1998).

The vertebral column and the maxilla develop in the foetus through three stages: the blastemal stage, which is the most complex; the cartilaginous stage, in which the bone elements take on their working shape; and the osteogenic stage, in which the immature bones undergo ossification and become true bone. This final phase continues into early adulthood to allow normal growth. The correct timing of embryological events is vital in order for the foetus to develop normally. Any interference in the phases of development may be traced to the time of gestation at which they occurred, by observing the type of defect present. *Dysplasias* can be hypoplastic (under-development), aplastic (absence of an element), or take some other form of malformation. *Hypoplasia* results from some interference in the development of the *anlage* (the primitive bone element), which prevents it growing to its normal size or shape. *Aplasia* is a result of interference which prevents the development of the *anlage* itself, which means there is no precursor present from which an element may subsequently develop. The majority of congenital defects are caused by insults to the foetus during the period of *organogenesis* (the developmental period of organs and systems) which lasts from the fifteenth to the sixtieth days of gestation. Defects caused by developmental delays are the commonest congenital malformations of all, particularly in the vertebral column and central nervous

system, due to their complex formation processes during the embryonic stage of foetal life. Cleft palate and cleft neural arch are common examples of developmental delay defects.

Normal development of the blastema (the conceptus during the blastemal stage) is required for the subsequent normal development of the cartilaginous or membranous precursor of bone. Any interference with this development can result in abnormal bone formation. Problems in one developmental field can have an effect on another field, for instance, a meningocele (a herniation of the meningeal membranes through a weakness in the vertebral canal) will produce a defect in the vertebral column (spina bifida), because bone forms in response to soft tissue formation. The timing of the disturbance can affect the severity of the defect. If development is almost complete when interference occurs, then the defect may not be as severe, although it could lead to the development of a severe defect in another field whose development is dependent on the first. Defects are frequently multi-factorial in origin, and when this is taken into account along with the effects that timing has on development, the aetiology of many congenital conditions is almost impossible to understand fully.

### **3.1.i The blastemal stage**

The blastemal or early cellular phase, which lasts from approximately the 16th day of gestation to the sixth week, is the source of many congenital defects in the skeleton, as it is during this stage that the genetic pattern is laid out in the foetus, and during which the foetus is highly susceptible to interference. Many women are unaware of their

pregnancy during this stage, and may unwittingly expose the foetus to hazards which they would otherwise avoid. Problems with correct formation of the skeletal elements at this point result in malformations in the foetus because the building blocks of the bones are inadequate for subsequent normal development. Such defects may be so severe that the foetus is subject to spontaneous abortion, while others reach birth with some form of dysplasia which may not allow them a normal life-expectancy.

On the 15-16th day of gestation, cells diverge from the primitive streak to form the notochord, which eventually develops into the neural plate, giving rise to the neural tube and the central nervous system (see fig. 3.1). The notochord or *chorda dorsalis* is the framework around which the *paraxial mesodermal* cells form the blastemal vertebral column. Its remnants will become incorporated into the nucleus pulposus of the intervertebral discs and the apical and alar ligaments of the axis vertebra.

The neural tube develops from the neural plate, which develops along the length of the notochord (see fig. 3.1). As the plate grows, its margins form neural folds (the first signs of brain formation), which eventually form a groove in the plate. This groove deepens into a canal-like structure, the submerged neural tube (ultimately the spinal cord), which lies cranio-caudally in the embryo. The ends of the tube remain open until the 4th week, and it obtains its nourishment from the amniotic fluid (the fluid surrounding the foetus *in utero*). Closure of the ends of the tube coincides with development of its vascular circulation and the formation of the embryonic cerebro-spinal fluid (the fluid that circulates around the spinal cord and brain within the sub-arachnoid space, between the arachnoid mater and the pia mater, the two inner meningeal membranes).



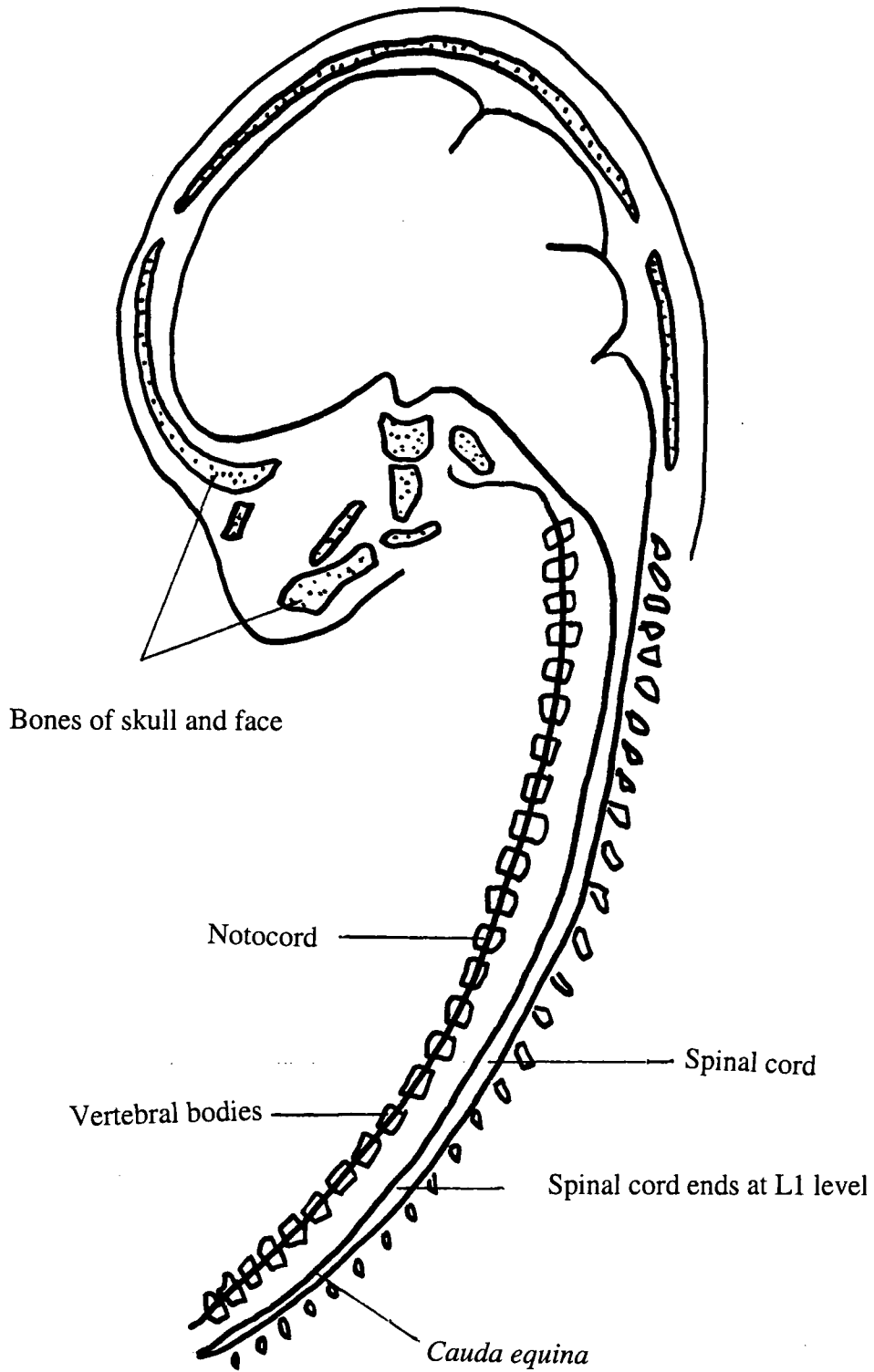


Fig. 3.1 Schematic drawing of the Central Nervous System and axial skeleton in a rat foetus at 18 weeks gestation (after Kjaer, 1998)

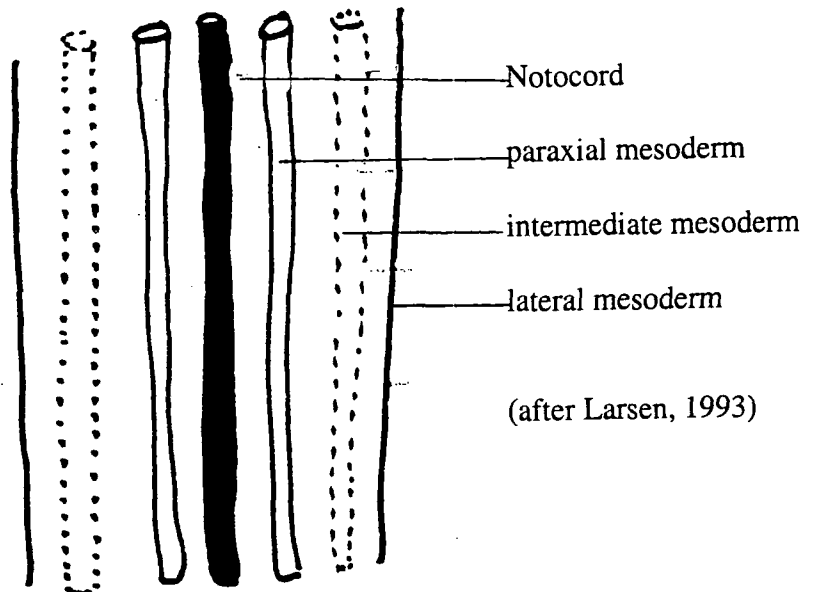
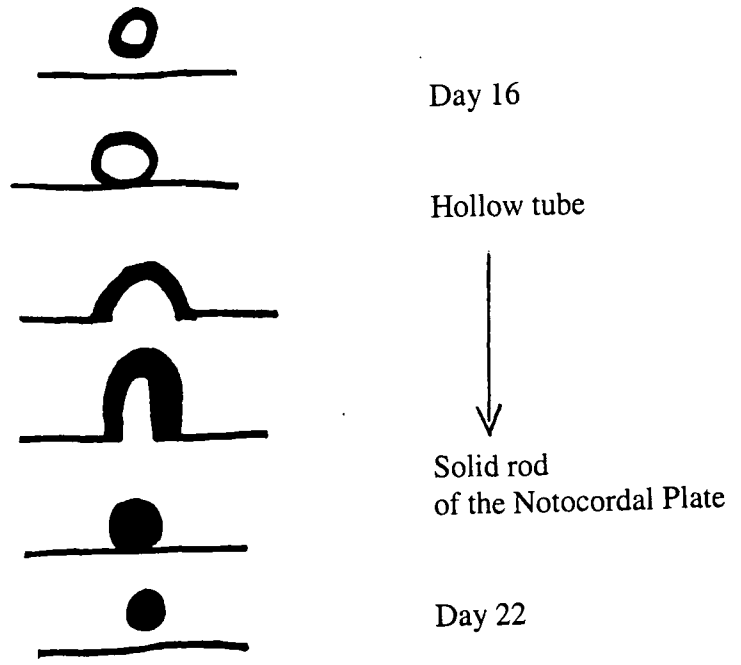


Fig. 3.2 Development of the notochord and the mesodermal layers (after Larsen, 1993).

Faulty development of the vascular circulation can lead to neural tube malnourishment. This may result in failure to close, which gives a neural tube defect such as *spina bifida*, a *myelomeningocele* or *meningocele*, a herniation of the spinal cord and/or meninges out of place inside the vertebral column (see fig. 2.3).

Work by Roth (1976) suggested that bony elements form in response to neural stimulation, thus observed defects in the skeleton may be traced back to interference with the normal development of neural pathways in the embryonic stages. More recent work has shown that the primitive central nervous system and the developing axial skeleton are in fact a developmental unit, both relying for their subsequent normal development on the notochord (Kjaer, 1998). Faulty development of the neural tissues derived from the notochord may be identified by the area of bone or tooth anomaly observed in the infant/child. Cleft, dental and spinal defects may be identified as the sites of compromised development along the original track of the notochord in the foetus (Kjaer, 1998).

### **3.1.i.a** *Common sources of defects arising from the blastemal stage*

The notochord is completely formed by day 20, and then transforms from a hollow tube into a solid rod by day 22 (see fig.3.2). This solid rod is called the *notochordal plate*. Whilst the initial precursors of the vertebral bodies coalesce around this feature, it contributes no material to the bony elements of the vertebral column, as any notochordal cells trapped in the vertebral bodies die and disappear (Larsen, 1993:52). It is, however,

important in the induction of the vertebral bodies, and failure in this inductive interaction results in many common vertebral defects (Larsen, 1993:52). The notochord is commonly referred to as the precursor of the nucleus pulposus, which forms the intervertebral discs. This is only true in the embryo, foetus and young child. Early in childhood, those nucleus pulposus cells of notochord origin are replaced by adjacent mesodermal cells (Larsen, 1993:52). Thus, intervertebral disc problems may not be all attributed to notochord problems.

The paraxial mesoderm forms in a “cylindrical condensation” (Larsen, 1993:54) around the notochord in the third and fourth weeks of gestation, starting cranially and progressing caudally. Lateral to the paraxial mesoderm is the *intermediate mesoderm*, and this is in turn encased in the *lateral mesoderm*, the whole of the mesodermal structures enclosing the notochord (see fig.3.3). The paraxial mesoderm gives rise to the axial skeleton, voluntary musculature and parts of the dermis (skin), whilst the intermediate mesoderm gives rise to the urinary system and parts of the genital system. The lateral mesoderm divides into two layers, giving rise to the linings of the body cavities and the structures within them such as the heart, lungs, kidneys; it also gives rise to parts of the limbs and most of the dermis. Problems with the differentiation of the mesoderm thus lays the foundations of systemic and visceral dysplasia.

### **3.1.i.b** *Formation of the blastemal vertebrae*

The paraxial mesoderm consists of two columns of cells grouped around the long edges of the notochord (see fig.3.2). At 21 days gestation these begin to segment into

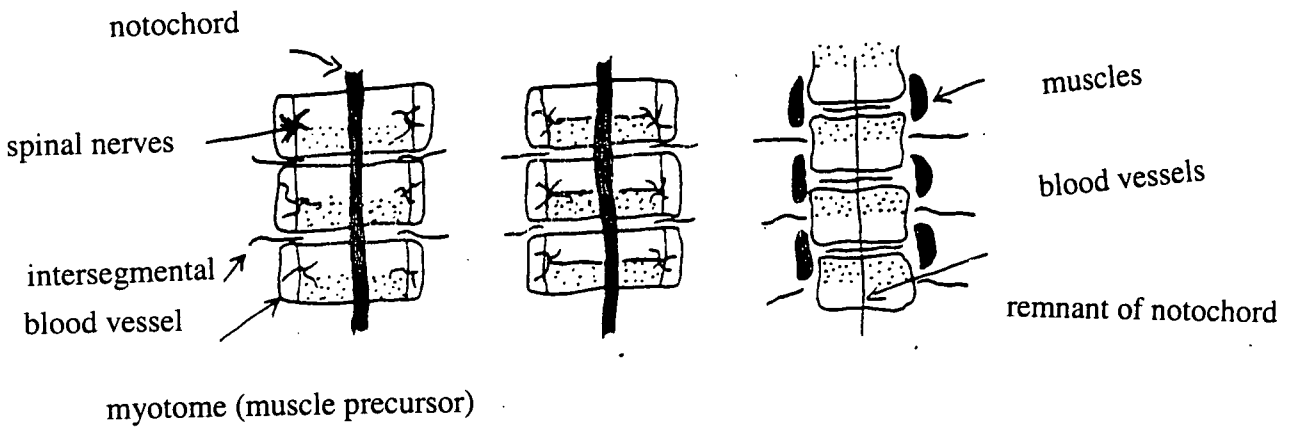
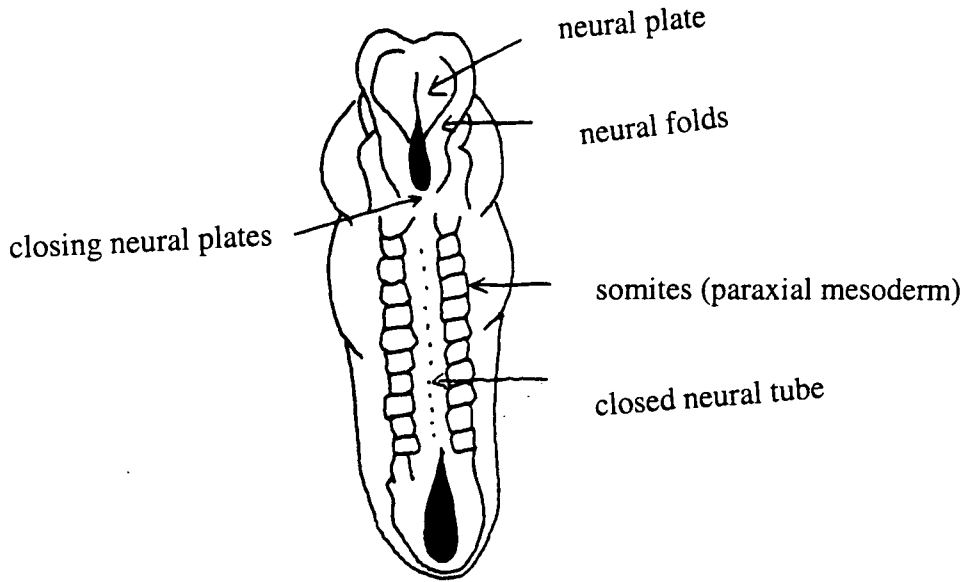
hemi-metameric pairs to form block-like *somites*. This begins at the cranial end of the embryo and progresses caudally. By the end of day 30 there are 42-45 pairs of somites, each separated by an intersegmental septum. This stage is the source of numerical variation in the numbers of vertebrae. The cranial-most four pairs (more can occur as a non-metric trait) develop into parts of the occipital. The remainder develop into the vertebral column. Thus, 8 pairs form the cervical vertebrae, 12 pairs form the thoracic vertebrae, 5 pairs form the lumbar vertebrae, 5 pairs form the sacral vertebrae and 8-10 pairs form the coccygeal vertebrae, which at this point measures  $1/6$  the length of the embryo. After a further four weeks the tail regresses to form the coccyx. Each somite develops independently of its pair, and then the pairs unite together around the notochord.

Three cell types are derived from the united somites, of which one type, the *sclerotome* cells, develop into the vertebral column. These are the *ventromedial somites*, which multiply rapidly and form a mesenchymal (pre-cartilaginous) sheath around the notochord. They eventually surround the neural tube to develop into the vertebral column. Vertebral bodies and intervertebral discs are formed by the division of sclerotome cells (see fig. 3.3). The cranial segment of each somite develops less densely than the caudal segment, and the two split apart. The resulting fissure then fills with mesenchymal cells from the cranial element. By three months gestation, these cells have surrounded the notochord remnants and eventually form the intervertebral discs. The denser caudal segment joins with the cranial segment of the next sclerotome to

form the anlage of the vertebral centrum, which grows into the vertebral body. Cells from the dense caudal segment extend dorsally to form the neural arches. Extension ventrolaterally forms the transverse and costal processes. The costal processes in the thoracic elements continue to grow into ribs.

After segmentation of the cranial and caudal halves of the sclerotomes, *differentiation* occurs. This involves the development of each vertebral element into a specific type of vertebra (cervical, thoracic, etc). Anomalies in the separation and unification of the cranial and caudal halves of the sclerotomes results in confused differentiation between vertebrae, both in numbers (too few or too many), and in type, giving malformations such as cervical ribs at C7, or T1 without ribs, for instance.

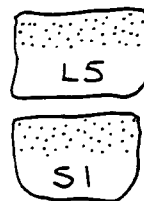
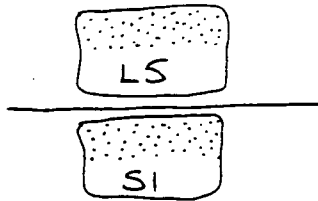
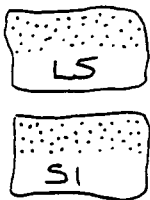
The atlas and axis develop differently. The anterior and posterior arches, and the lateral masses of the atlas, and body of the dens, are thought to be derived from the caudal segment of the first cervical sclerotome. The cranial half is assimilated to the exoccipitals and also forms the tip of the dens. The body, posterior arch and the transverse processes of the axis are derived from the second cervical sclerotome. Cells from the outer layers of the neural folds of the embryonic brain develop into specific elements of the head. The maxilla and mandible are formed during the 5th to 8th weeks of gestation as the face and pre-maxilla develop anteriorly to the growing forebrain. Problems here can result in malformations of the maxilla such as cleft palate.



somites

somites divide

vertebral bodies



normal border position

caudal shift (lumbarization)

cranial shift (sacralization)

Fig. 3.3 Development of vertebral bodies and intervertebral discs (after Beck, *et al*, 1995 and Barnes, 1994)

### 3.1.ii *Chondrification*

This stage begins in the vertebral column by the 6th week. Cartilage cell development follows the pattern laid down by the blastemal precursors. Delay in the blastemal cells reaching their “critical size” for cartilage formation to begin can result in any of the following: delay in, or absence of, cartilage cell replacement, which can result in hypoplasia or aplasia (under-developed or absent body-parts); premature fusion of cartilaginous elements where the blastemal precursor is too thin, which can lead to abnormal bone formation, particularly in the base of the skull, or abnormal separation or anomalous union of cartilaginous elements of the skeleton, again leading to bone malformations, such as cleft neural arch. As the cartilage model is the “blueprint” for the bone, normal development at this stage is critical for skeletal mineralization and development (Reddi, 1982:180). It is already recognised that low levels of available phosphates and magnesium are problematic in achieving this normal development (Reddi, 1982:180).

The vertebrae chondrify (turn into cartilage) from two paired centres in each blastemal vertebra. One pair diverges to form the rudimentary neural arches, the other pair joins to form the centrum, which eventually develops into the vertebral body. Failure of the centrum pair to unite causes hypoplasia or aplasia of the vertebral body in the ossification stage. Chondrification begins at the cranial end of the column and proceeds in a caudal direction. During the third month of gestation, the neural arches close over the vertebral body and enclose the spinal cord.



### 3.1.iii Osteogenic stage

The vertebral column begins to ossify during the 8<sup>th</sup>- 9<sup>th</sup> week of gestation and continues throughout childhood and adolescence. Centres of ossification do not match the chondrification centres. In the vertebral body ossification begins at one centre in the centrum (the precursor of the vertebral body). The process begins in the centra of the thoracic and lumbar elements, and moves upwards and downwards from there, reaching the cervical vertebrae by the 12<sup>th</sup> week. The vertebral arch halves each have an ossification centre situated in the *pars articularis*. Defective development in this area is associated with many cases of spondylolysis. Arch ossification starts in the cervical and thoracic areas, and after a short delay, continues in the lower thoracic, lumbar and upper cervical regions. This primary ossification completes by the 20<sup>th</sup> week of gestation and is complemented by secondary ossification in puberty. At birth the vertebrae consist of a bony centrum and two bony arch halves, all of which are un-united. Fusion of arch halves (synostosis) occurs between the first and third years of life, and fusion of arches to centra between the third and sixth years. Ossification centres for the vertebrae including the transverse processes and the articular processes appear at about 16 years and unite by about 25 years. The sacral elements fuse starting with S1-S2 at about 16 years and complete by about 30 years. The coccygeal elements ossify and fuse between 1 and 18 years. The mandible and maxilla ossify directly from mesenchymal tissue, therefore by-passing the chondrification stage. This ossification begins at the 6<sup>th</sup> week of gestation with the pre-maxilla (the anterior portion of the upper jaw), containing the origin of the incisors. It fuses with the maxilla at the incisive suture as the bones come together in the formation of the mouth.

### 3.2 ANATOMY OF THE VERTEBRAL COLUMN

The vertebral column typically consists of thirty-three elements (vertebrae), although numerical variation is not uncommon (see fig. 3.4). The segments comprise seven cervical vertebrae, twelve thoracic vertebrae, and five lumbar vertebrae. The sacrum consists of five rudimentary vertebrae which are fused together into one segment. Below the sacrum is the coccyx, which consists of four or five small bones making up the vestigial “tail”. These are rarely found archaeologically, although occasionally the first coccygeal element is fused to the fifth sacral bone.

#### 3.2.i The functions of the vertebral column

- to provide a strong bony protection to the spinal cord by the formation of the neural canal, which is made up of the vertebral foramina along the length of the column.
- to allow safe passage out of the spinal cord for the spinal nerves via the intervertebral foramina. These are spaces formed between adjacent vertebrae which give bony protection to the spinal nerves.
- to support the skull, which is protected from shocks by the presence of the intervertebral discs between the vertebrae.

to form the axis of the trunk, and to give attachment to the ribs, the shoulder girdles and the upper limbs, the pelvic girdle, and the lower limbs.

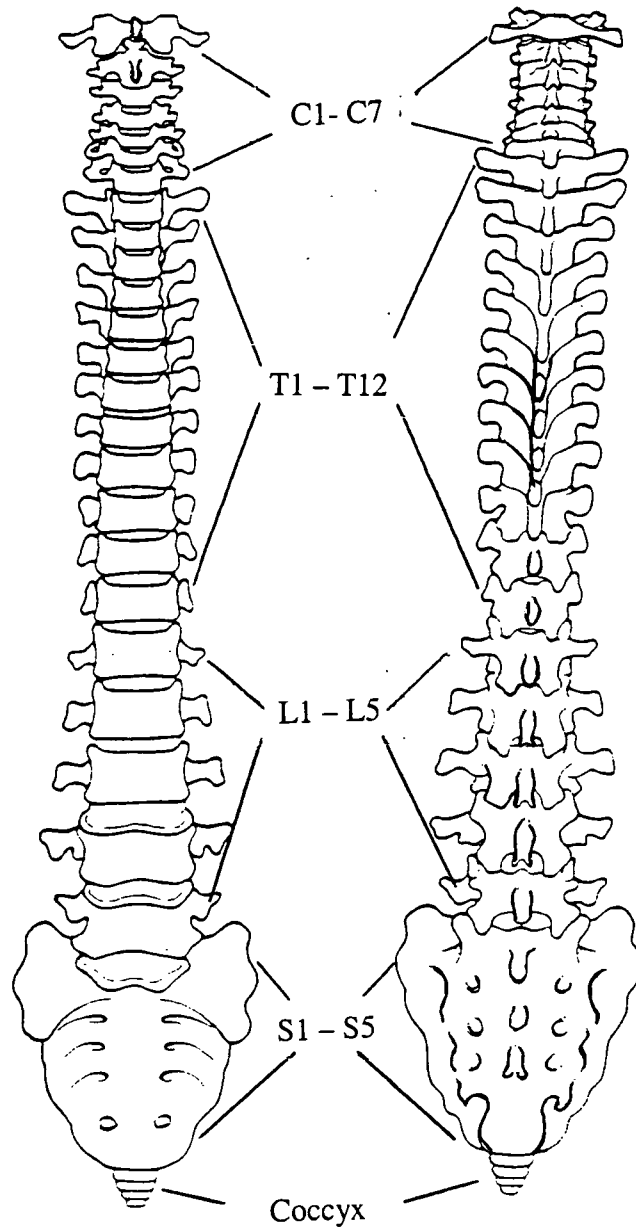


Fig. 3.4 The vertebral column: 1 - 7 = cervical; 8 - 12 = thoracic; 13 - 17 = lumbar; 18 - 22 = sacral).

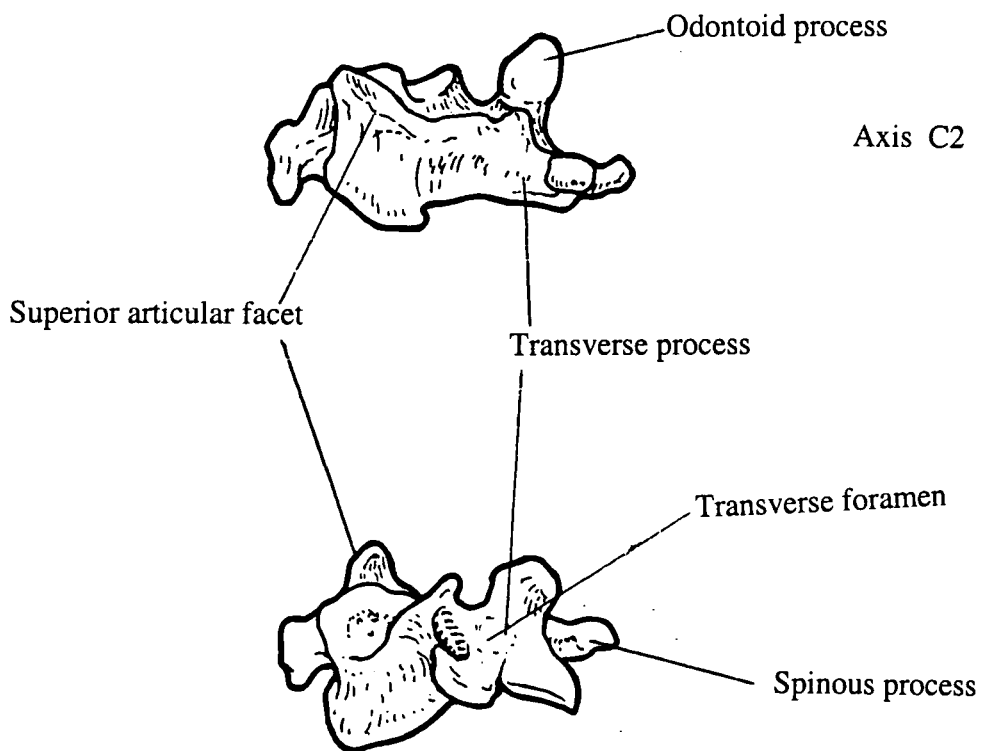
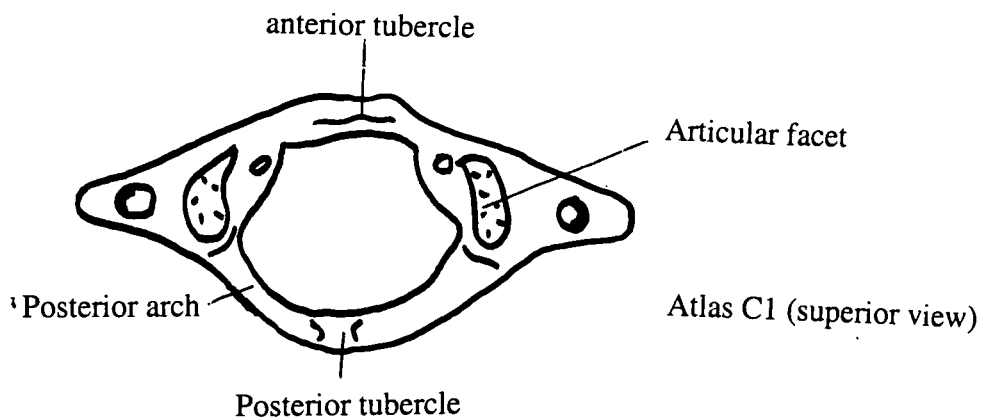


Fig. 3.5 The cervical vertebrae

### 3.2.ii.d The sacrum (See fig. 3.7)

The sacrum is made up of five rudimentary vertebrae that fuse together in adulthood to form one bone. The anterior surface is concave. The posterior surface has five ridges running down it, representing the vestigial spinous processes of the vertebrae (the median ridge); the fused articular processes (the intermediate crests on either side of the median ridge), and the fused transverse processes (the lateral crests). The foramina carry spinal nerves from the lower end of the spinal cord. The upper sacral body articulates with the fifth lumbar vertebra, and the lowest point of the sacrum articulates with the coccyx.

### 3.2.ii.e The coccyx (see fig.3.4)

The terminal end of the spine is made up of between three to five coccygeal vertebrae, which fuse together to form one triangular-shaped bone, the coccyx. This is the vestigial tail. It articulates with the base of the sacrum.

### 3.2.ii.f. The spinal cord (see fig. 3.1)

This is the principal nerve pathway of the body. It is contiguous with the brain, leaving the skull through the foramen magnum and progressing down the spine through the neural canal. The spinal cord itself extends from the upper border of the atlas (C1) to the lower border of the first lumbar vertebra. It divides below this point into a multi-stranded set of nerve fibres which pass downwards through the lumbar neural canal. This terminal portion of the cord is the *cauda equina* (horse's tail). The entire spinal cord and brain are enclosed within three layers of protective tissue known as the

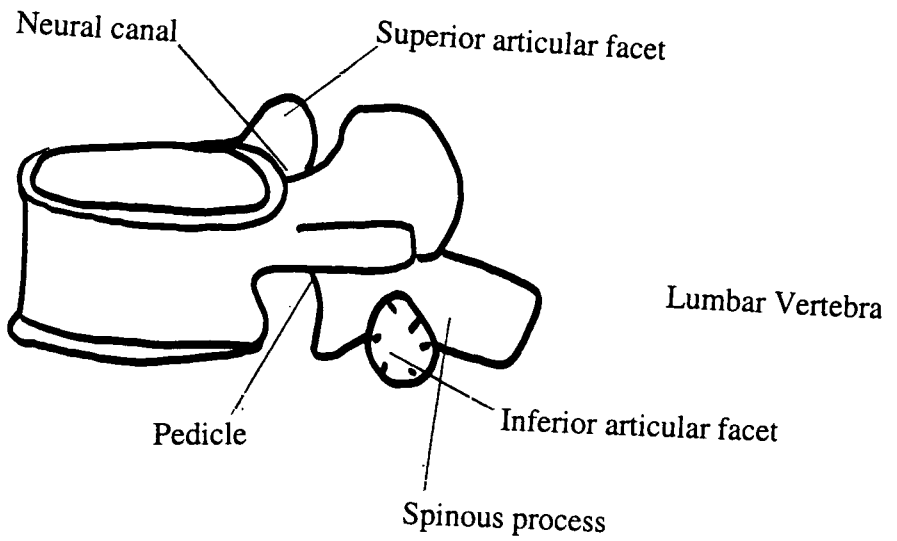
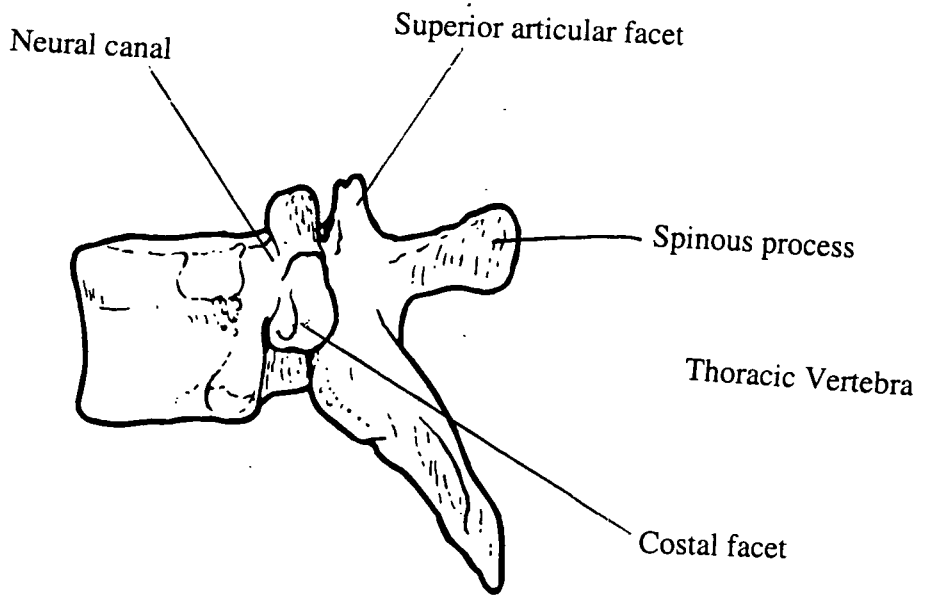


Fig. 3.6 The thoracic and lumbar vertebrae

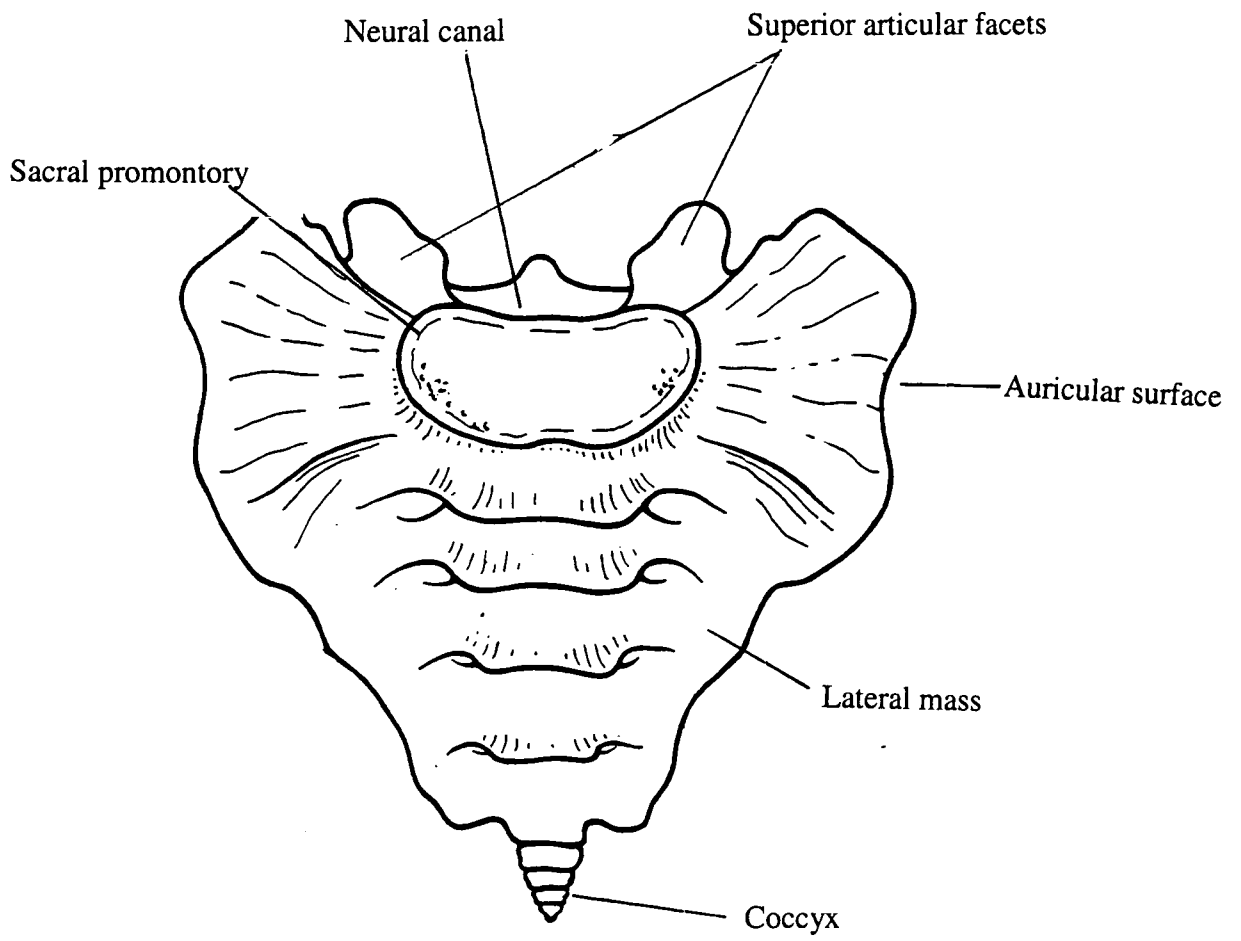


Fig. 3.7 The sacrum

meninges. These are the *dura mater*, the *arachnoid mater* and the *pia mater*. Within the space between the arachnoid mater and the pia mater, flows the cerebro-spinal fluid, which acts as a cushion against shocks and as a source of nutrients for the neurological tissue. The brain is enclosed in the cranium and the spinal cord in the neural canal of the vertebral column.

### **3.2.iii. Movement** (see fig. 3.8)

Movement in the column is restricted to flexion, extension, and lateral flexion. Rotation is possible to some degree without putting tension on the spinal cord, but movement of all these types is greatest in the cervical and lumbar regions. Because of the close physical association between the spinal cord and the vertebral column, it can be seen that any problems with the formation and/or function of the vertebral column could have serious implications for the health, lifestyle, and possibly survival of the individual. Any abnormal flexion or extension of the column may rupture the meninges and/or break the spinal cord, thus causing paralysis or severe neurological deficits below the level of the injury.



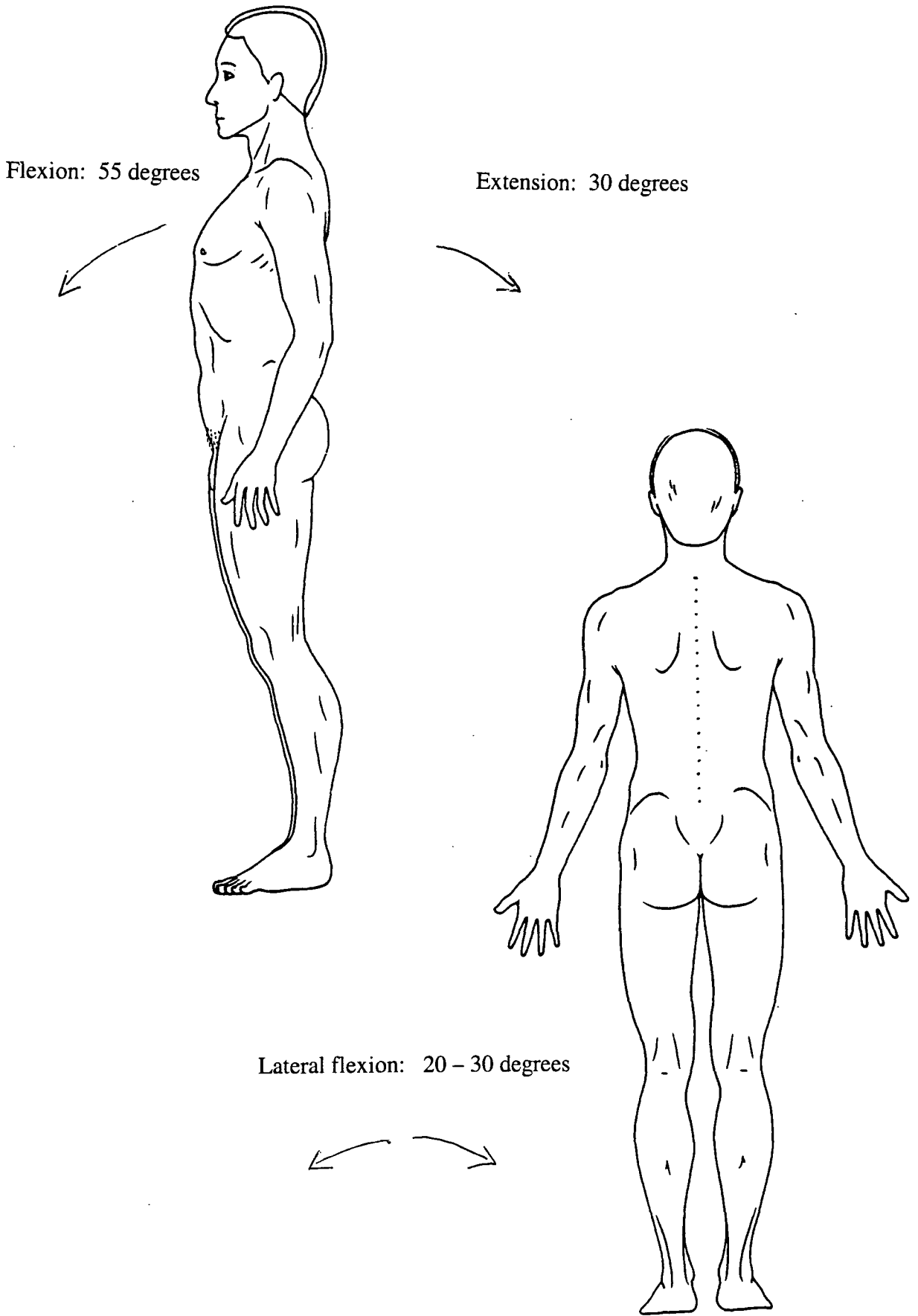
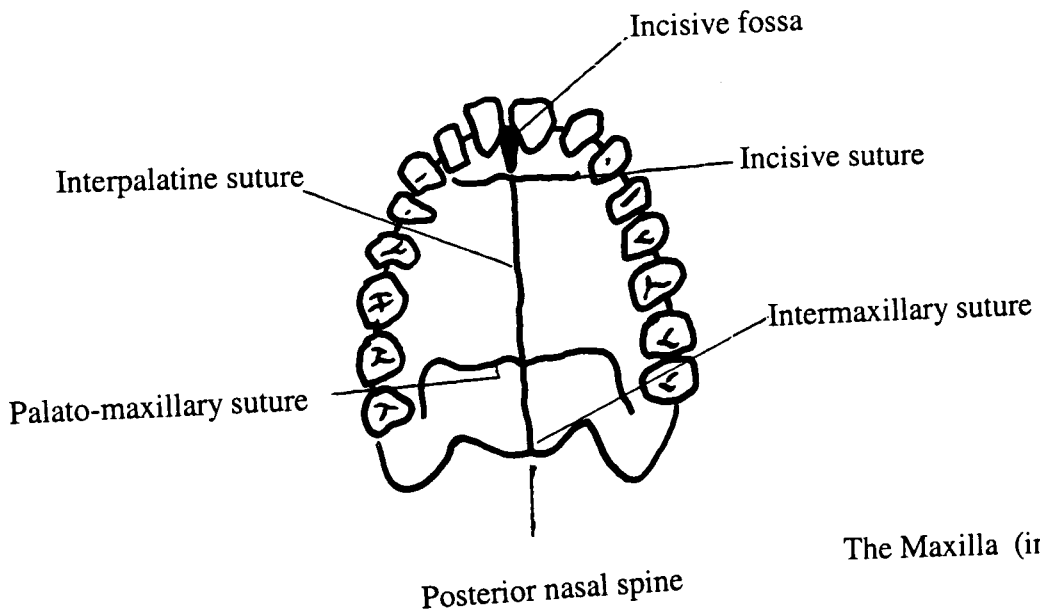


Fig. 3.8 Range of movement allowed by the vertebral anatomy

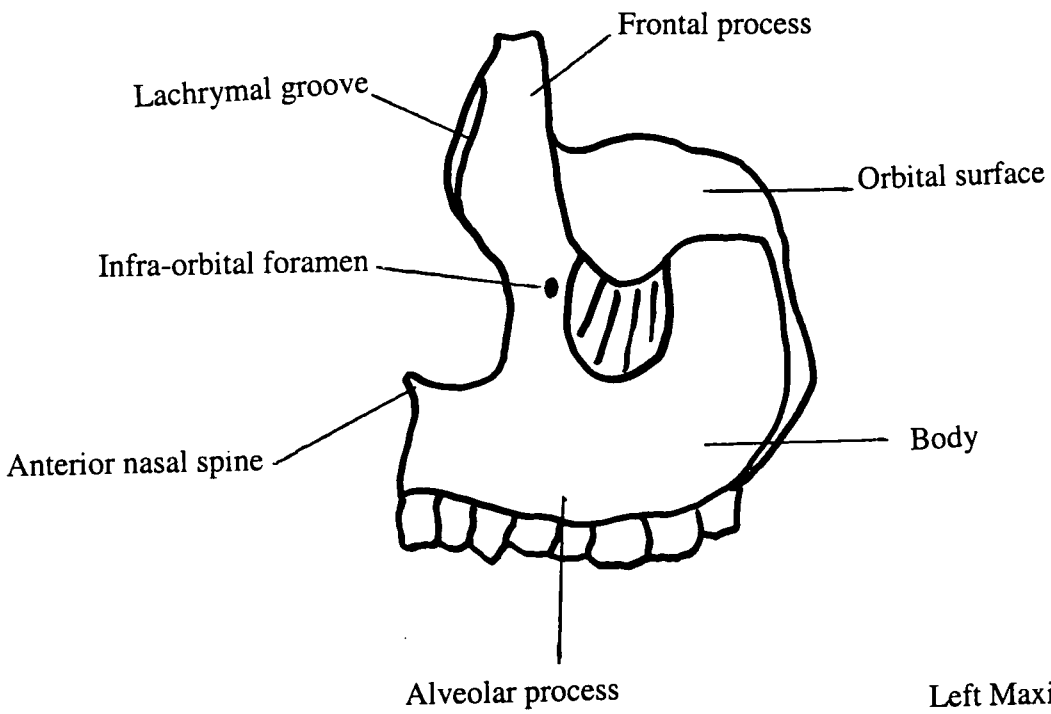
The normal range of movement in the vertebral column allows anterior flexion up to approximately 55°, extension of approximately 30°, and lateral flexion of approximately 20°-30°. The degree of movement generally diminishes with increasing age as the vertebral column becomes subject to osteoarthritis, reduced physical fitness, or injury.

### **3.3 ANATOMY OF THE MAXILLA (see fig. 3.9)**

The upper jaw, or maxilla, comprises two bones which fuse together before birth to form one element. The maxilla is one of the largest bones making up the face, and forms parts of several other facial elements including the hard palate (the roof of the mouth), the orbital floors, and the lateral walls of the nasal cavities. It also carries the alveolar process which support the upper teeth. The body of the maxilla contains the maxillary sinus, which reduces the weight of the bone, and aids resonance of the voice. The infraorbital foramina allow passage of the infraorbital nerves and blood vessels out of the skull. The anterior nasal spine is a sharp projection arising in the midline below the nose. The frontal processes rise from the body and form the lateral walls of the nasal fossa. The zygomatic processes are lateral projections from the body which articulate with the zygomatic bones (cheek bones). The palatine process projects medially from the body of the maxilla, fusing with its opposite half to form the roof of the mouth. The incisive foramen is a foramen or canal in the midline formed by the two halves of the maxilla, just behind the central incisors. The alveolar process forms inferiorly from the body and holds the upper dentition. It forms a U-shaped arch and generally holds eight adult teeth.



The Maxilla (inferior view)



Left Maxilla (lateral view)

Fig. 3.9 The anatomy of the maxilla

Two of the four maxillary sutures are used in age estimation, although this method is not particularly reliable. The incisive, interpalatine, intermaxillary and palatomaxillary sutures are well-defined at birth with spaces along their margins. The incisive suture, running between the lateral incisors and the canines, touches the posterior portion of the incisive foramen. This suture thus delineates the pre-maxilla. The degree of obliteration of this suture, which begins at the teeth and moves towards the incisive foramen, has some role as a simple age indicator, with increasing obliteration as age advances. The interpalatine suture is a continuation of the intermaxillary suture, continuing posteriorly from the palatomaxillary suture to the posterior nasal spine. This also obliterates with increasing age.

Archaeologically the palatine process survives better than other parts of the maxilla, although it is often recovered in two pieces, having fallen apart at the intermaxillary suture. This has led in the past to mis-diagnosed cases of cleft palate, when the condition of the bones in fact represents post-mortem degradation.

## CHAPTER 4

### **Birth defect research: medical and epidemiological issues**

#### **4.0 MEDICAL RESEARCH: BACKGROUND TO THE STUDY**

The hypothesis of this study implies that birth defects are associated with reduced nutritional and immunological status in the mother, either before or during (or both) pregnancy. The nutritional and immunological systems operate in a symbiotic (mutually beneficial) relationship, and disturbances in this symbiosis often result in reduced efficiency in both systems (Chandra, 1980; Scrimshaw, 2000). Such reduction in efficiency may be expected to result from the increase in pathogen load, exposure to infectious agents, poor quality nutrition, and other hazards of increased population density (Chandra, 1978, 1979, 1980; Ericksen & Hubbard, 1993; Frisancho *et al.*, 1977; Roberts & Manchester, 1995; Scrimshaw, 2000). One of the principal health-mediating issues that distinguish the urban lifestyle from the rural is arguably that of population density, in addition to, or over-riding, potential differences in housing, sanitation, food storage and preparation, family support networks, and so on (Bogin, 1988; Coleman, 1995; Goldberg, 1992; Little & Leslie, 1993; Roberts & Manchester, 1995). This effect is well-associated with health problems (Roberts & Manchester, 1995; Kiple, 1997) such as increased infection rates and higher disease rates in general. Coupled with the issues of poor quality food due to inadequate storage and cooking, and the adulteration for commercial gain that is frequently reflected in court records throughout the Medieval period, the nutritional and immunological status of urban populations is likely to have been relatively compromised. To date,

little has been done to investigate the effects of the nutrition/immunology relationship with birth defect aetiology. Knox & Lancashire (1991) considered the possible effects of viral infections on birth outcomes, and Saxen (1983) investigated the effects of the influenza virus similarly. Neither authority found broad associations with birth defect rates, but they had focused particularly on specific viral agents rather than on a generalized increase in non-specific pathogen exposure. Therefore, the author aims to correlate aspects of nutrition, immunology and teratology in an attempt to understand birth defect aetiology and prevalence, by using data derived from English Medieval populations. This chapter will outline the background of teratological investigation, and briefly summarize the immune and nutritional systems in order to clarify the arguments upon which the hypothesis is founded. The aim of this chapter is to provide a framework within which the hypothesis of the study may be understood. The final section will address aspects of epidemiological study as it investigates environmental exposures, and shows how biological anthropologists utilising the biocultural approach may, in effect, conduct useful epidemiological studies, that may have implications for health research in modern populations.

#### **4.1 TERATOLOGY: THE STUDY OF INSULTS TO THE FOETUS**

Teratology may be defined as the scientific investigation of agents causing birth defects in human populations. A teratogen is defined as “any substance, agent or process that induces the formation of developmental abnormalities in the fetus” (Martin, 1994). To attribute a single, simple cause to any defect or family of defects is, however, still usually impossible. Teratology relies largely on a sound knowledge of embryological development, particularly of the blastemal stage and

the period of organogenesis (approximately 15<sup>th</sup> day to the 60<sup>th</sup> day of gestation, see Chapter 3). The origins of birth defects have been identified during the twentieth century to a greater degree than in any previous period of human history. This is due to a combination of factors, including an ever-increasing knowledge of embryological development, and a series of international events that were subsequently studied and found to be causally related to the appearance of birth defects. Among these were the European rubella epidemic of the early twentieth century, the nuclear bombing of Japan in the 1940s, and the thalidomide disaster of the 1960s.

The development of teratology as a discipline arose from the realisation that certain agents acting on pregnant women appeared to lead to congenital defects. The term “teratology” was coined by a French scholar, Isidore Geoffroy St.-Hilaire in his three-volume work plus atlas, that appeared in 1832, 1836 and 1837 (Smithells, 1980). The work, that had a cumbersome main title but an alternative one of “*Traite de Teratologie*”, followed on from work published by the author’s father in 1822, that had focused on human monsters: “*Philosophie Anatomique des Monstruosites Humaines*”. Geoffroy St.-Hilaire the younger was far-sighted in his ideas. He believed that teratological study should be a multi-disciplinary science, and more recently, Smithells has supported this idea, envisaging an increasing diversity of involved disciplines. During the twentieth century, teratology has become an established research discipline and has produced much valuable work, often arising from such major health catastrophes as those mentioned above. In addition, the search for causal factors has broadened, tending now to move away from the search for a single external cause for a

specific defect family, and considering instead more complex and subtle aetiological factors. For example, in recent years the effects of raised temperature (that is a mechanism of the immune response during infectious episodes) on the development of the embryo have been studied, showing that malformations of the axial skeleton may be induced by exposure of the mother to raised temperatures on specific days of gestation (Angles *et al.*, 1990; Kimmel *et al.*, 1993).

#### **4.1.i Intergenerational factors and defect rates**

Emanuel and Sever were among the first to consider intergenerational issues in birth defect aetiology, and to look for complex aetiologies rather than to single teratogens as the causes of most defects. For example, instead of focusing on poor quality potatoes (a popular suggestion as a cause of central nervous system defects in the 1960s and early 1970s) it was their suggestion that an interference with the growth and developmental processes of mothers of affected children could be a significant aetiological factor in neural tube defects (1973b: 329). They considered that such interference could be “short- or long-lived” and could include “*nutritional deficiencies, severe illness, poor sanitation, and emotional and sociocultural deprivation*” (1973b: 329). They proposed a unifying hypothesis that brought together three types of causal factor: genetic, immediate and intergenerational (1973b: 329). This meant that effects mediated by all three types of factor act upon the genetic and environmental status of the mother. They did not, however, consider the possible role of paternal genetic and environmental input into affected babies, that is surprising, as spermatogenesis is ongoing, and surely more susceptible to environmental variables than female ova, that are all in place at birth, and may only have been adversely affected during the nine month



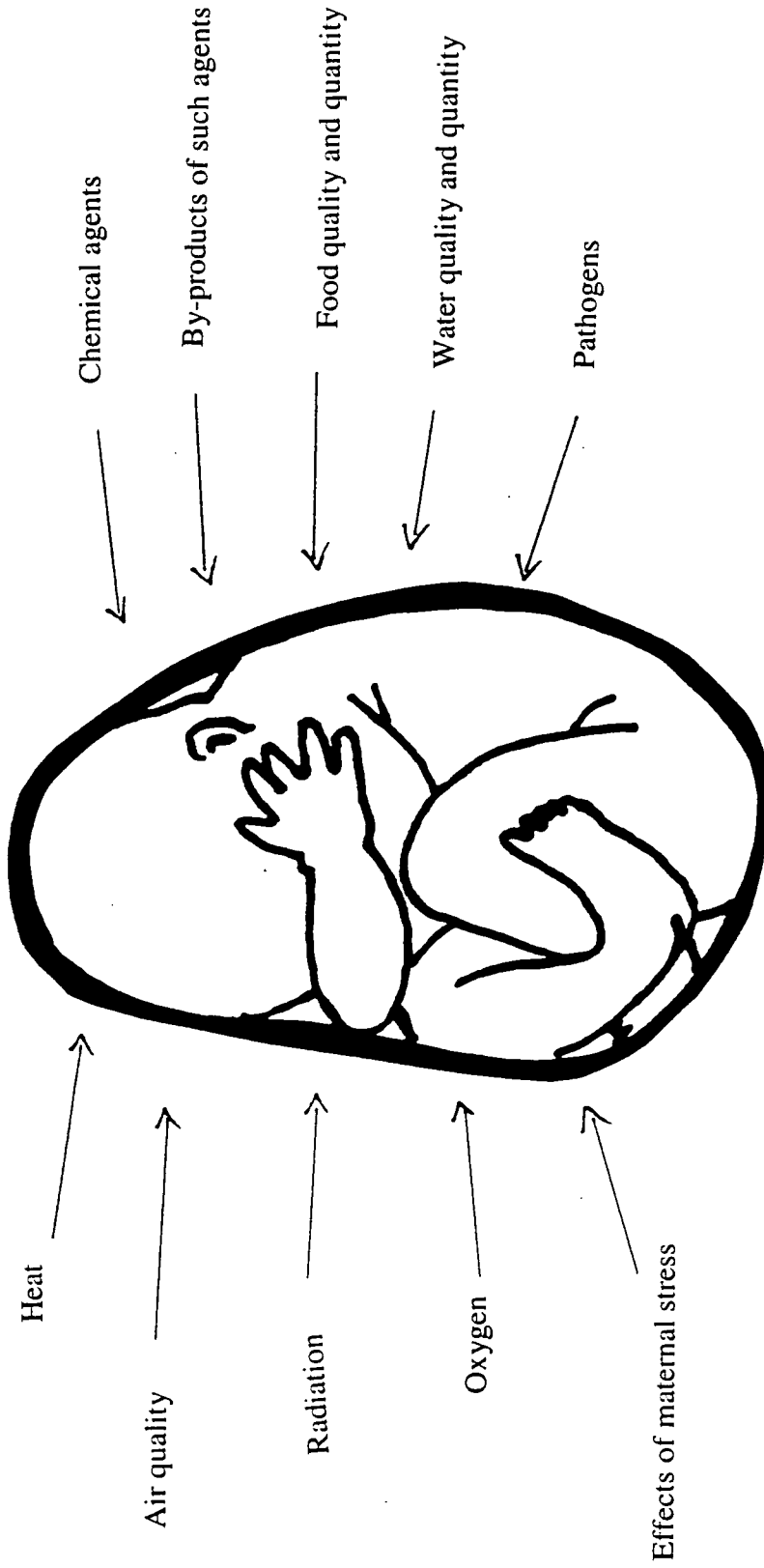


Fig. 4.1 Environmental agents acting on the foetus

period of gestation. However, Emanuel and Sever's approach was far-sighted and is still being developed today. The intergenerational perspective on reproductive health has since been taken up and expanded by Barker (1994, 1998), cited elsewhere in this study.

#### **4.1.ii Normal foetal development and potential interference** (see fig.4.1)

Human development requires certain environmental conditions functioning within "normal" ranges. These include temperature, oxygen tension, quality of food, quantity of food and water (Hahn, 1979). Any variation of these norms outside the acceptable limits within which normal development may take place may therefore pose a threat to the normal development of the embryo. For example, prior to placental assumption of foetal nutrition (around 12 weeks' gestation), the embryo is fed directly from the surrounding maternal tissues (Hahn, 1979). Once the placenta takes over, any alteration in the nutritional supply to the foetus via the maternal circulation, or any significant alteration in maternal and therefore foetal body temperature occurs, may result in compromised development.

Numerous experimental studies since the 1960s have shown that many vertebral anomalies can be induced in mice by exposing the pregnant female to variable temperatures, levels of oxygen and so on (e.g. Angles *et al.*, 1990; Bennett, 1972; Kimmel *et al.*, 1993). It may be supposed that similar consequences affect pregnant human females to some degree, but this is difficult to identify, as many people find they are unreliable in their recall of episodes of ill-health (Barker & Hall, 1991).

The presence of infection in a pregnant woman will, of course, lead to systemic and localised immune response activation, including a raised temperature. The work of Angles *et al.* (1990) and of Kimmel *et al.* (1993) showed that rat embryos developed anomalies of the neural plate and of the somites when exposed to raised temperatures. They also induced vertebral anomalies such as fused vertebrae, absent vertebrae, and other defects of vertebral/rib morphology. These results led to the suggestion that pregnant women with raised temperatures should not be exposed to ultrasound examinations as this would further raise the temperature around the foetus (Angles, 1990). Other axial skeletal defects were observed in chick embryos when alterations in bone morphogenetic proteins were induced during the somitic stage of development (Nifuji *et al.*, 1997), resulting in vertebral formation anomalies including malformations affecting the lateral and dorsal aspects of vertebrae.

From the psychological perspective, stressors acting on the pregnant woman may also induce a neuro-immunomodulatory effect within her body, in which her perception of stress and her ability to control it may result in reduced ability to fight infection, thus exposing her to longer and/or more intense periods of pathogenic activity (Ursin, 1994).

#### **4.1.iii Teratogenicity and research - some problems**

Hogue (1984) outlined some difficulties with research into human exposure to teratogens. These included:

- rates of adverse outcomes vary according to exposure levels
- one hazard may be a confounding factor in the study of another

- a common exposure may produce a synergistic effect with an environmental agent
- documenting human exposure accurately is problematic (Hogue, 1984:45).

For example, both smoking and alcohol consumption are associated with spontaneous abortion, low birthweight, perinatal mortality and developmental defects. But most women will have been exposed to both substances for significant periods, and isolating the effects of one from the other is often impossible (Hogue, 1984:46). In view of this difficulty, this study of Medieval population samples is not intended to isolate any one aetiological agent, but intends to assess the physical responses of those population samples to two different types of environment (i.e. urban and rural) in terms of population density and its associated hazards to health.

#### **4.1.iv Critical periods and organizational processes: how defects develop**

Development of the embryo consists of a series of irreversible events in which the organism moves towards greater complexity of form and function. The organizational changes that occur in the embryo have been described by Scott (1986: 182ff) as involving *maintenance processes* and *developmental processes*, that together lay the foundations of the final life-system - the infant. Scott's work addressing the phenomenon of *critical periods* in organizational processes related the general theory of critical periods to the concepts of human growth and development. (1986:181-196). Critical periods were first identified by Stockard in the early twentieth century. He concluded that the stimulus to abnormal development was not the *nature* of the environmental alteration, but the *time at*

*which it was applied* (Stockard, 1921, cited in Scott, 1986). Work by Child in the 1920s (Child, 1921, cited in Scott, 1986) associated heightened vulnerability with those cells undergoing the most rapid change at any given time. Later, Scott's work showed conclusively that "organizational processes are modified most easily at the time that they are proceeding most rapidly" (Scott, 1937). This showed, in effect, that those processes within the foetus that are undergoing rapid developmental change on any given day were the most at risk from external interference resulting in unwanted change leading to malformation. Scott's own work in the 1930s on polydactylous guinea pigs showed that genetic alteration could occur as a growth enhancer (producing extra digits) as well as being a growth inhibitor (such as was later seen in relation to Thalidomide, with limb reductions/absence). This statement obviously applies to many embryological developmental processes, involving the development of all the organs and systems of the human body. It is also apparent that developing systems may be affected by more than one agent, and that the nature of the agent *is secondary* to the time at that it is applied to the developmental process (Scott, 1986:188). This fact alone makes the identification of cause and effect in congenital malformations a monumental task.

#### **4.1.v Critical periods and teratogens in humans**

The concept of time is therefore fundamental to the phenomenon of organizational change. Modification of a living organism may only be possible within specific time-periods, or even within a single specific period lasting only a few hours (embryological change is already recorded in terms of specific days). It is this association of time with the organizational changes necessary for development of

the embryo that leads to the concept of critical periods in human embryological development. Teratogenic agents may only have a detrimental effect if they act on the embryo at or during these “critical periods” of development, such as those of organogenesis between days 15 and 60 of foetal life. A teratogenic agent may therefore have little or no effect on a specific organ should it interact with the mother at any other time in the pregnancy. It may, however, cause a developmental problem with another organ that is due to occur on that day, thus affecting another organ. It must be stressed, however, that it is not yet possible to attribute direct action between teratogen and embryo *at a specific time* during foetal life. Warkany (1971) stated that it is not feasible to attribute a malformation to a single event during a critical period and this opinion still holds today. It is simply possible to say “the teratogen had its effect before the end of the organogenetic period of the structure or organ concerned” (Moore, 1978:134).

#### **4.1.vi Teratogens and genes**

In considering the role of environmental agents in the developmental well-being of the foetus, the role of genetics cannot be ignored completely. Genes are organic molecules that have a fundamental role in organizational processes (Scott, 1986:183). Their role in enzyme production, which influences, among other things, the speed of chemical reactions, is of great significance in the developmental changes taking place in the foetus. The effects of environmental agents on both individual genes and on combinations of genes within the genotypic system are probably the source of many congenital malformations, and although genetic research is forging ahead apace, the understanding of the effects of external agents on the makeup and action of genes (individually and as groups)

is still not fully understood. Certain genes have been identified as being associated with certain congenital problems (e.g. the PAX and KFS genes), but it is the unquantified change to other genes which result in unforeseeable defects which are much more difficult to distinguish.

#### **4.1.vii To summarise teratogenic action on the foetus:**

If a teratogen (an agent or an event) is applied to the human embryo, it has the potential to affect an organizational process within the embryo. If the teratogen only affects a single organizational process it will act in a specific manner, to produce a specific detrimental effect:

- the critical period is the time during which a specific organizational process is taking place
- prior to the critical period, the teratogen will have no effect
- the teratogen affects the embryo if it is applied during the critical period
- its effect on the embryo is proportional to the speed of the organizational change taking place in the embryo and inversely proportional to time i.e. it has most effect when the organizational process is proceeding at its fastest. When applied early in the organizational process, some repair may be possible, thus reducing the effect of the teratogen, but when applied late in the organizational process, there is less chance of reorganization to repair the damage done
- once the organizational process is complete and the critical period past, the teratogen will have no further effect.

#### 4.2. THE IMMUNE SYSTEM (based on Reeves & Todd, 1996)

This study addresses, indirectly, the immunological status of Medieval English populations. Past populations were not under the protection of the public health measures, medical treatment, vaccination programmes and so on which benefit 20<sup>th</sup> century populations. The author suggests that problems arising from the impaired interaction between the immune and nutritional systems could be related to the frequency of birth defects observed in the populations examined.

##### 4.2.i Components of the immune system

The body attacks infection in two ways, via the implementation of *specific* and *non-specific* anti-microbial protective mechanisms, which together form the immune system. *Specific* immunity arises from the altered reaction of the body which results from an initial exposure to a new antigen; this type of immunity is mediated by *antibodies* and *B-lymphocytes*. Once the body has been exposed to the rubella virus, for instance, it forms antibodies that have a specific action against that particular micro-organism. Future exposure results in either a very mild illness, or none at all.

Lymphocytes are one of three types of white blood cell, all of which have a role in protecting the body from foreign matter. Lymphocytes enable the immune system to be specific in its ability to react to antigens. They enable it to “remember”, or recognise, the same antigen on re-exposure; and also give it the ability to recognise “self” cells and therefore limit tissue damage to host cells during the immune response (Reeves & Todd, 1996:38). They are present in the blood and also in lymphoid tissue and bone marrow and are subdivided into B-cells, which



produce the circulating immunoglobulins which fight systemic infection, and T-cells, which act against agents which have actually entered the body's tissues.

*Non-specific* immunity arises from two sources, *molecular* and *cellular*. The nature of this immunity is *innate*, meaning that it is not affected by exposure to an antigen. It is the body's "in-built" capability to fight infection, comprising cells which are already in place ready to counteract foreign materials. Its activities can, however, be affected by certain factors. Non-specific immunity arises from the activities of leucocytes and lymphoid tissues. There are three types of leucocytes (white blood cells). These are phagocytes, or cells which engulf and digest other cells, that are important in defence against parasitic infection as well as fighting bacterial and viral infections; basophils, which contain histamine and are also involved in the allergic/inflammatory reaction, and mast cells, which have a role in the defence of mucosal surfaces (e.g. the linings of body structures) and are the cells which release chemicals which affect the permeability of blood vessels, for instance, in the inflammatory response.

Lymphocytes may circulate freely in the blood, but the majority are to be found in the lymphoid tissues. *Primary lymphoid organs* consist of the bone marrow and the thymus in adults. *Secondary lymphoid organs* consist of diffuse colonies of lymphoid tissue spread around the body: in the lymph nodes, the white pulp of the spleen, and mucosa-associated lymphoid tissue (MALT) such as Peyer's patches in the small bowel, the tonsil and the adenoids. The majority of mature lymphocytes are to be found in the secondary lymphoid tissues, where they come into contact with each other and also the antigen-presenting cells that stimulate

them into producing an immune response. Lymphocytes are also present in a diffuse, thin spread of follicles in the sub-epithelial *lamina propria* of the small intestine.

#### **4.2.ii Immunogenicity - the strength of the immune response**

This is governed by several factors related to the nature of and exposure to the antigen. The nature of the antigen includes its chemical nature, size, the charge on the cell surface, and the degree of “foreignness” i.e. the more unfamiliar the antigen the stronger the immune response. Exposure to the antigen includes the dosage of the antigen, frequency of exposure, the route of entry, and any *adjuvants*, or additional substances taken with the antigen which intensify the inflammatory response. The nature of the recipient also governs the immune response. For example, age is a crucial factor - the very young, in whom the immune system is relatively “uneducated” and the elderly, in whom there may be deficiencies which impact on the efficiency of the immune system, often experience tolerance induction instead of immunity. They therefore fail to defend themselves against the antigen. The genetic makeup of individuals also varies and may differ significantly in response to antigen. The nutritional status of the individual also affects the strength of the immune response. For example, malnutrition, or metabolic disorders such as diabetes, are known to have an effect on the individual’s response to antigen.

#### **4.2.iii Immunity in action**

*Extracellular* bacteria, viruses which undergo a *viraemic phase* (a period of spread via the blood stream), and some fungal infections, all appear to be sensitive

to antibody and phagocytosis. *Intracellular* bacteria, some enveloped viruses, protozoa, and some fungi, may only be successfully eliminated through the action of T-cells, macrophages and natural killer cells (large granular lymphocytes) (Reeves & Todd, 1996:148). Helminths, or *macroparasite worms*, pose a slightly different problem to the immune system. Generally speaking, they are immune to the effects of antibody and lymphocyte cytotoxic cells. They appear to be susceptible to the activities of leucocytes, which mediate antibody-dependent reactions. It is believed that a primary helminth infection is attacked in this manner, and that subsequent infections are mediated by IgG or IgA antibodies (Reeves & Todd, 1996).

#### **4.2.iv Immunity and its role in this study**

Immunodeficiency can be acquired as a result of bacterial, viral, fungal, parasitic or other disease, including nutritional deficiencies (Bendich, 1993; Chandra, 1980; Klurfeld, 1993; Passmore & Eastwood, 1986; Scrimshaw, 2000; Siegel, 1993). Generally speaking, immunodeficiency does not have a *direct* effect on nutritional status, but its *indirect* effects may result from the action of chronic intestinal bacterial, viral, and protozoal infections, all of which may cause chronic diarrhoea, malabsorption of nutrients, and loss of blood and proteins from the gut (Ferguson, 1993: 689). Protein-loss states and anaemia (whether due to iron-deficiency or chronic disease processes) are significant factors in the maintenance of an effectively functioning immune system (Kent & Stuart-Macadam, 2000; Scrimshaw, 2000). Long-term blood loss itself frequently leads to anaemia, which in turn impairs overall health. In chronic cases of mal- or undernutrition, or persistent poor health, the body's capability to produce the working components

of a healthy immune system, such as lymphocytes and leucocytes, are highly likely to be compromised due to impaired haemopoiesis (Janeway *et al.*, 1997; Reeves & Todd, 1996). Malnutrition or undernutrition are therefore likely to impair the ability of the immune system to act efficiently (Kent, 2000; Scrimshaw, 2000). Any circumstance that involves a reduced nutritional intake or reduced absorption of nutrients can lead to mal- or undernutrition. This scenario encompasses individuals who are suffering illness, those who eat a restricted diet for cultural or religious reasons, or those with problems such as alcoholism or drug addiction, which inhibit the normal functioning of the body's organs due to adverse chemical effects.

The disruption of the normal anatomy and physiology of the gut as a response to antigens is a problem that must have been significant in the health of past populations. The protection against micro-organisms and parasites offered by the lymphoid cells in the gut is depleted when tissue damage occurs as a side-effect of the immune response. When dealing with certain infectious diseases, the immune system is known to react with what Ferguson refers to as “the innocent bystander phenomenon” (1993: 687). For instance, tuberculosis and leprosy are known to be associated with damage to the gut wall when the immune system attacks harmless foods, “mistaking” them for antigens. The overstretched immune system of an individual already overloaded with pathogens and parasites will therefore often react in a similar manner to other, non-specific, infectious agents. When this is the case, the immune system is active in its own destruction, as the combined effects of reduced health status and undernutrition rob it of its capacity to fight antigens over a prolonged period. Pregnancy, in the presence of an impaired nutritional

uptake and an already overloaded immune system, would add increased pressures onto the mother which she may not be able to meet by the usual adaptations (Allen, 1986).

#### **4.2.v Summary of the immune response in action**

In order to function effectively, the immune system must have the capacity to:

- recognise antigens from amongst a potentially endless group of organisms
- overwhelm the foreign material by generating a large defence in response to a small stimulation by antigen
- overcome a vast range of antigens
- regulate itself so as to minimize the damage done to “self” tissues by the immune response (Reeves & Todd, 1996:147).

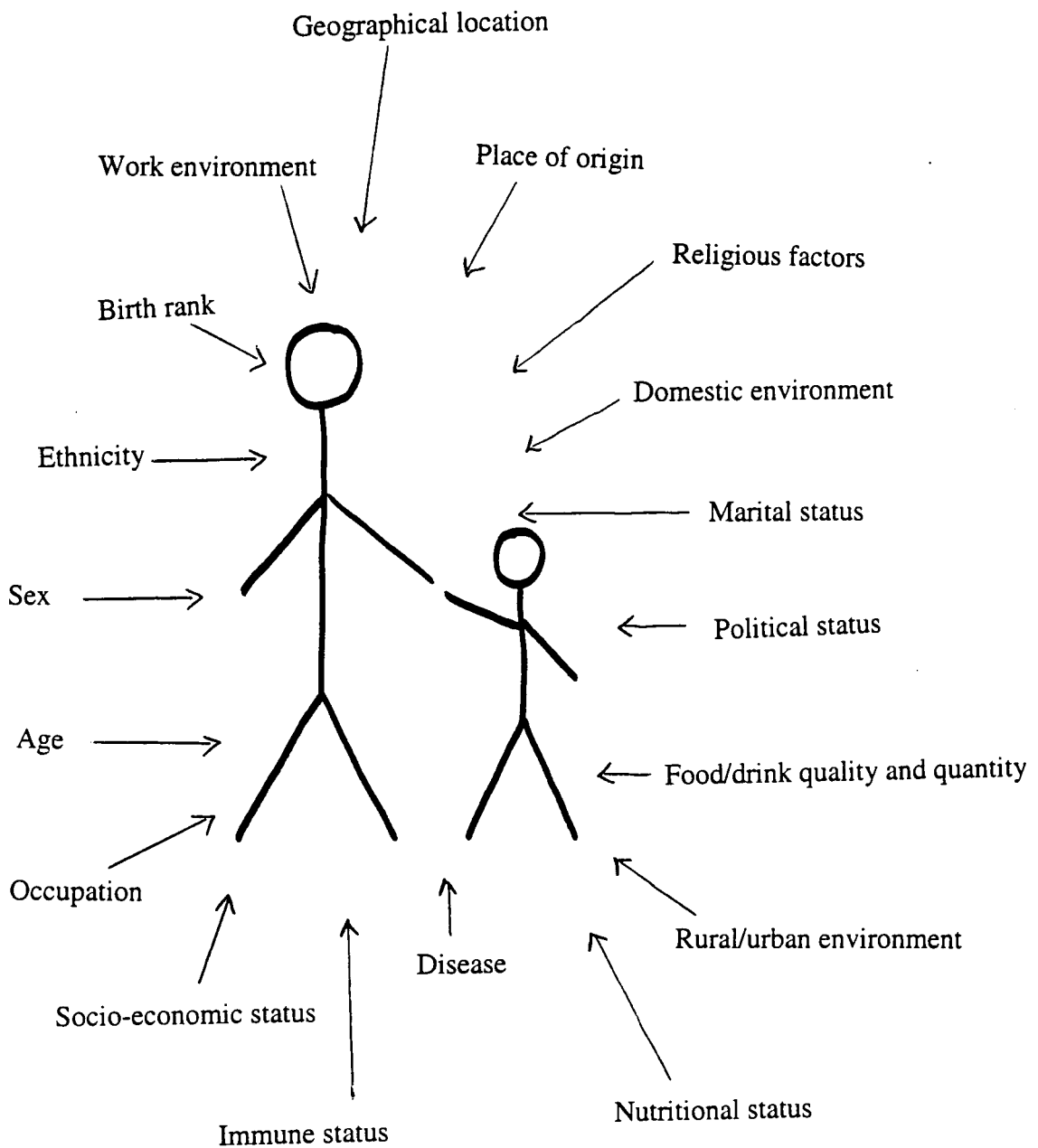


Fig. 4.2 Variables affecting human health

### 4.3 NUTRITION AND HEALTH

“Adequate food and nutrition are essential for proper growth and physical development *from conception to adulthood* [italics mine], to ensure optimal work capacity and *normal reproductive performance*, and also to *ensure the adequacy of immune mechanisms and resistance to infections* [italics mine].....

.....an inadequate diet produces two main types of metabolic nutritional disorder: protein-energy malnutrition (PEM), and micronutrient disorders (mainly deficiencies).....

World Health Report 1997:50-51

#### 4.3.i Nutritional requirements today and in the past

Nutrient requirements today are expressed in several ways, for example, the RDI, or Recommended Daily Intake, as seen on the side of cereal boxes. Those eating diets with this level of the nutrient will not usually be at risk of deficiency. RNI levels vary, with males and females often, but not always, requiring different amounts. In addition, the requirements vary with age, and in females, with pregnancy. In the past, nutritional deficiencies would have played a large part in the health of most populations, for reasons such as seasonally or economically restricted food supply, inadequate storage, preservation or cooking techniques, lack of effective hygiene measures, and health issues causing inadequate nutrient uptake, such as parasitic infection. Unfortunately, an adequate diet is not enough to ensure health if nutrient uptake in the gut is compromised, for example, through under-absorption of nutrients due to vitamin imbalances or damage to the gut lining, or if preparation, cooking or storage techniques destroy much of the nutritional value.

In order to expect successful pregnancy outcomes within a population, mothers require certain standards of nutritional status in order to provide for both

themselves and the growing foetus during pregnancy (Hahn, 1979). Pre-pregnant nutritional status is as important as that during pregnancy, with, for example, a deficiency in folates at the time of conception being strongly associated with raised rates of neural tube defects (Smithells, 1992). Allen (1986) outlined the known effects of certain micronutrient deficiencies on pregnant women. She pointed out that when pregnant, a woman's diet must undergo a qualitative change as well as a quantitative one. Recommended daily amounts of nutrients change, with energy requirements rising by 15%, and those of other nutrients by 15% to 333% (Allen, 1986:267). In addition to deficiencies in micronutrients, pregnant women who suffer protein-energy malnutrition (PEM) are known to produce babies with low birthweight. Maternal effects of PEM include low weight gain during pregnancy, reduced fat storage and reduced placental cell number (Allen, 1986:266). The hypothesis of this study suggests that populations suffering differential nutritional and/or immunological status due to the hazards of increased population density, may be producing infants who have suffered developmental defects as a result of inadequate provision of nutrients whilst *in utero*. This could be due to either malabsorption of nutrients, infectious processes in the mother diverting her energies away from the developing foetus, or a combination of both conditions acting together to interfere with the normal development of the foetus.

#### **4.3.ii Undernutrition**

Chronic undernutrition occurs when the requirements of daily energy expenditure are not met by long-term food consumption. This is not the same as the acute malnutrition seen in populations suffering famine. Body measurements such as



thinness in adults, and stunting in children are indicators of undernutrition. Acute malnutrition (wasting) results from a severe, abrupt reduction in food consumption. Chronic undernutrition is related to many causes, which include problems with food availability as well as health issues such as chronic or multiple infections, which result in non-uptake of nutrients from the gut. As a result, undernourished populations tend to be of smaller stature than they would otherwise be, and to suffer from increased numbers of infections (due to the symbiotic nature of the nutrition:immunity relationship), which are often also more severe. Work capacity also decreases (WHO, 1997:50-51). Protein-energy malnutrition (PEM) is widespread in the developing world, but is not as common as micronutrient deficiencies (low serum levels of vitamins and minerals), which include iron-deficiency anaemia. These problems are likely to have been common in the past, affecting populations of the Medieval period which is under examination in this study. For example, inadequate intake of fresh or suitably-stored produce is well-associated with nutritional deficiencies (Coulter & Davies, 1994), and consumption of such foods would have been common before the advent of refrigeration, freezing, and regular supplies of good quality fresh foods.

#### **4.3.iv Food groups**

Proteins, carbohydrates, fats and micronutrients make up the human diet. Energy is gained from fats, alcohol, proteins and carbohydrates, and the percentage from each source is important in a balanced diet. Typically, about 15% of energy is derived from proteins, and the remainder from fats, of which there are two essential fatty acids (linoleic acid and alpha-linoleic acid), and from carbohydrates, which consist of sugars and starches. Dietary fibre is now

commonly referred to as non-starch polysaccharides (NSP). It is an important component of the diet, ensuring healthy bowel activity, but is also associated with certain micronutrient deficiencies as it binds some minerals and prevents their adequate absorption (Coultate & Davies, 1994:21).

*Proteins* are required for structural maintenance of the body, and their role in the transport of oxygen by haemoglobin, the maintenance of antibodies of the immune system and in food as milk caseins providing nutrition for newborns may all play a part in the reproductive health of women (Coultate & Davies, 1994:69). The continuum-like condition of protein-energy malnutrition (PEM) can be a primary or secondary phenomenon, resulting from an inadequate intake of nutrients or from illnesses affecting the gastro-intestinal tract or the immune response (Kuvibidila *et al.*, 1993:121). Protein is taken up as essential amino acids which are used by the body as required. It may be obtained from animal sources such as meat, offal, eggs, fish and milk. Plant sources include legumes, pulses and soya, but are generally not as valuable as animal sources, as they often lack certain substances. For example, wheat contains some protein, but it lacks lysine, one of the nine essential amino acids. Thus, it requires about double the amount of wheat than human milk protein to provide the requirements of the body. Legumes contain lysine but lack methionine, another essential amino acid. A healthy diet therefore requires both legumes and cereal if meat is low in the diet. Cereals such as wheat, rice and maize “provide twice the food energy for human consumption of all other food sources put together” (Coultate & Davies, 1994:77). Despite this, there are variations in protein quantity between crops grown at different times of the year, which of course may have been a significant nutritional issue in past

populations who relied on cereal-based proteins for at least a part of the year. For example, spring-sown wheat contains 12-14% protein, whereas autumn-sown wheat contains less than 10% protein. Milk proteins come in the form of caseins (undissolved proteins) and whey, or serum proteins (dissolved proteins) (Coultate & Davies, 1994:80).

*Vitamin* requirements vary. In childhood all requirements are lower than in adulthood (Coultate & Davies, 1994:91). Males require larger amounts than females in adolescence and adulthood, but within the sexes, the levels required remain roughly the same throughout adult life. They are essential to normal development and growth in humans and are required throughout adult life to maintain the normal physiological and biochemical functions of the body (Coultate & Davies, 1994:91). There are two forms of vitamins: water-soluble and fat-soluble. Because humans cannot generally synthesise vitamins in the body, dietary intake is vital. Specific deficiencies arise from inadequate intake or uptake. Both insufficiency and excess uptake of vitamins may result in disease. For example, mammalian liver is so high in Vitamin A that pregnant women are advised against eating it today, in order to avoid toxicity. Excess Vitamin A results in liver enlargement, skin and hair problems, skeletal abnormalities and psychiatric problems (Coultate & Davies, 1994:96). Vitamin D excess produces calcification of soft tissues, for example in the lungs and kidneys (Coultate & Davies, 1994:96). This may, of course, have been more common in the past amongst those living a rural, predominantly out-of-doors lifestyle. Excess Vitamin C is associated with diarrhoea. Other vitamins when taken in excess appear to be eliminated from the body in the urine and therefore do not seem to cause any ill-

effects (Coultate & Davies, 1994:97). Loss of vitamins from foodstuffs occurs when freshness is lost, and during cooking and preserving. In addition, inadequate normal intestinal uptake or impaired uptake due to illness such as parasitic infection, for example, may also result in deficiencies. Vitamin content in foods tends to be lost or seriously diminished within twenty-four hours of harvesting, due to bruising, wilting, or over-cooking (Coultate & Davies, 1994:98). The milling process also removes vitamin B content from wheat, as does prolonged baking (Coultate & Davies, 1994:99).

*Minerals*, including sodium, potassium, iron, zinc, magnesium, copper and calcium are found in plant and animal foods in varying concentrations, requiring a varied diet to provide the necessary amounts (Coultate & Davies, 1994:83-4). Reactions with other foodstuffs such as sugars are often either required for mineral uptake, or act as uptake inhibitors, hence intake does not always equal uptake. Iron is the mineral most likely to be lacking in the human body. However, as iron is so abundant in foodstuffs and water, *dietary* lack is very unlikely. Most iron deficiencies are the result of *inadequate uptake* of dietary iron. Anaemia is a late sign of iron deficiency; with about twice as many persons affected by iron deficiency as are anaemic. (WHO, 1997:50-51). Women of reproductive age and children under 5 years are primarily affected by iron deficiency, with prevalences of 40-50% in developing countries, including over 50% of pregnant women (WHO,1997). In industrialized countries about 10% of women and 17% of pregnant women, are clinically anaemic, with twice as many being *affected* by iron deficiency. The formation of red blood cells, and hence, haemoglobin, is heavily reliant on the availability of certain substances within the body. Proteins,

folic acid, vitamin B12, and traces of copper must be present in adequate amounts for normal erythropoiesis (formation of red blood cells) to take place, as well as iron. The majority of iron absorbed from the diet is contained in complex compounds known as *haem* groups. Dietary iron is divided into two categories: haem (haem) iron and non-haem iron. Haem iron is present in meat, fish and poultry. Non-haem iron is present in plant foods. A diet lacking in sufficient haem iron is not only a cause of iron deficiency, but also reduces the amount of iron absorbed from other sources - the proteins in meat, poultry and fish assist the uptake of iron from other foods, and this is especially important if the diet consists of one or two staples. Daily requirements average 10-14 milligrams per day, but losses may be high in females. For example, the *average* loss in a menstrual period is about 15-20 milligrams: women suffering from gynaecological bleeding can feasibly lose much more on a regular basis. Iron is generally poorly absorbed from the intestine, a problem which is compounded in disorders of the alimentary tract. In a normal state of health an average of about 10% of dietary iron is absorbed. Haem iron (animal sources) may be absorbed in amounts up to 15-25% in the best scenario, but only about 8% of non-haem iron in plants is absorbed. This is because plant iron is largely bound to phytates which inhibit its uptake by the intestinal mucosae. Alternatively, Vitamin C enhances iron uptake. Sugars and fruit acids acidify the alkaline intestinal environment and keep iron in solution, allowing easier absorption (Coultate & Davies, 1994:86).

Copper and zinc depletion is already associated with some adverse birth outcomes (Allen, 1986; Bell *et al.*, 1975; Sato *et al.*, 1985), and uptake of these elements may be impaired by excess Vitamin C, zinc and iron (Prohaska & Failla, 1993:

311). Zinc deficiency is common in the elderly, the pregnant, the lactating, PEM cases, and trauma cases with major fractures (Kuvibidila *et al.*, 1993:140), and is strongly associated with populations who rely on a cereal staple to provide their protein. The effects of deficiency on the immune response are many and include reduction of blood lymphocyte numbers, lymphocyte proliferation, primary and secondary antibody response, and wound healing. Susceptibility to infection is raised (Kuvibidila *et al.*, 1993: 142).

Deficiencies of iodine in the early stages of growth (*in utero*) can result in cretinism, when the mother is suffering from deficiency immediately before or during gestation (Coultate & Davies, 1994:88). Selenium deficiency produces reductions in phagocytosis, bactericidal capability, and lymphocyte proliferation. Susceptibility to infection is raised (Kuvibidila *et al.*, 1993: 142). Even simple suboptimal intakes have been associated with impairment of the immune system (Stabel & Spears, 1993:333). Copper deficiency is probably most commonly seen today in premature infants, and is associated with increased infections in them (Prohaska & Failla, 1993:312). Deficiency in adults and children is associated with recurrent infections, but the identification of copper deficiency as the cause is not always possible, due to concurrent problems such as PEM or other deficiency (Prohaska & Failla, 1993:312). Human responses are unclear, but in laboratory animals responses have included reduction in T-cells and the microbicidal activity of phagocytes (Kuvibidila *et al.*, 1993:145).

*Fats* play a vital role in human health, enabling the synthesis of essential hormones, the repair of damaged tissues, the efficient maintenance of blood-

clotting capability and the stimulation of uterine contractions (Coultrate & Davies, 1994:37). Arachidonic acid (a fatty acid) was previously known as Vitamin F, and is found in meat, or it may be synthesised from linoleic acid. It is implicated in the inflammatory process as well as those processes outlined above. Arachidonic acid is found in trace amounts in most animal products, but not at all in plant products (Coultrate & Davies, 1994: 37). Linoleic acid cannot be manufactured by animals, but can be synthesised into arachidonic acid. It is a vital component of our cell membranes. The requirement today is for approximately 3g/day, but healthy modern diets usually supply more than this. In the past, of course, this may not have been the case. In addition, a reduction in dietary fat intake can lead to a reduction in the amount of stored body fat, meaning fewer reserves of energy in times of illness or reduced food intake. This would place pre-pregnant and pregnant women, and their growing infants *in utero*, at greater risk of nutritionally-mediated disorders and adverse birth outcomes. Fatty acids are known to have a modifying effect on some immune responses in laboratory tests. For example, high levels of dietary fat have been associated with increased T-cell proliferation (Erickson & Hubbard, 1993:58). The B-cell function of immunoglobulin production also appears to be modulated by dietary fat levels. Neonate animals have been found to have altered IgG levels in response to changes in dietary fat (Erickson & Hubbard, 1993:59). Erickson and Hubbard stated: "We believe that immune and inflammatory responses may be modified depending on FA [fatty acids] in the diet..." (1993:59). The conclusion seems to be that dietary fat has a selective modulatory effect on immune functions. Cholesterol, a fat-like substance, is also associated with immunomodulation. It appears that hypercholesterolaemia has a suppressive effect on the immune

response (Klurfeld, 1993:87). Whether *hypochloesterolaemia* is associated with enhanced immune response is still the subject of discussion.

*Sugars and starches* form the carbohydrate group, and are the principal dietary source of energy. All carbohydrates are eventually broken down into the simple sugar glucose, which is then used in metabolic energy-producing processes.

Lactose is a major source of energy in all milk (Coultate & Davies, 1994:55).

Polysaccharides in plants perform two principal roles. They form the “skeleton” of the plant’s tissues, and provide a reserve of energy for the future (Coultate & Davies, 1994:50). They perform similar roles in the human body. Starches, for example, are a major energy source. Plant starches make up a significant part of our diet, especially in grains and tubers. Fine milling causes much damage to starch granules, which allows them to be degraded by flour enzymes. Fibre, or non-starch polysaccharides, is found in many foods, particularly those which have undergone the least processing. In this respect, diets in the past must have generally been much higher in “fibre”. The attrition of tooth enamel in past populations bears this out.

#### **4.3.v Non-nutrients in the diet which may affect reproductive health**

There are a number of components in the diet which serve little or no nutritional purpose, and some which are positively harmful to health, many of which may have been implicated in health in the past, when legal controls over food preparation and storage were not in place. These tend to be found in the following categories, as outlined by Coultate & Davies (1994:105):



- *raw material contaminants* - pigments, toxins and flavour compounds
- *contaminants secreted by micro-organisms in food* - botulinum, staphylococcus toxins, aflatoxins from moulds
- *agricultural practice contaminants* - pesticide/herbicide residues (in the modern context); sewage- and midden-spreading on fields in the past
- *contaminants from processing etc.* - heavy metals in processing water, dirt or residues left in processing equipment
- *additives* - synthetic or natural additives to enhance colour, flavour, etc. and also illegal additives e.g. contaminated water added to milk and beer or ale.
- *cooking procedure contaminants* - flavours, toxins, brown pigments from heated carbohydrates and fats, heavy metals in cooking water, hand-spread organisms.

#### **4.3.v.a Toxins in foods which may affect reproductive health**

*Smoking* (as a preservation technique) is now known to cause toxicity in food. For example, smoke contains over two hundred compounds that include formaldehyde, formic acid and benzpyrene, a known carcinogen. These enter the surfaces of foods as gases, at the same time as the surface is “disinfected” of microbes. Smoking was a common preservation process in the past. Nowadays steps are taken to remove the harmful elements in the process, but in the past, smoked foods would have contained quantities of toxic compounds, which, if taken over a long period, could have caused health problems. In addition, the inhalation of all smoke inside houses (domestic, or specially-constructed smoke-houses) must have had an effect on maternal, and possibly foetal health and

development. Upper respiratory tract infections such as maxillary sinusitis have already been shown to affect urban populations more than rural ones (Lewis *et al.*, 1995).

*Salting* was a common preservation method in the past. This process worked by reducing the water content that favoured bacterial growth in food. Saltpetre (potassium nitrate) was used in the past to preserve meat and to give an attractive pink colour. This would have raised the intake of potassium to seasonally high levels.

*Toxic metal residues* enter foods from soil, sludge, sewage, cooking or preparation water, sea or fresh water, dirty equipment, utensils, dishes and so on (Coultate & Davies, 1994; Craun, 1995). Lead is the most commonly encountered heavy metal. Originating from pipes or from water tanks, or from lead-rich local geology affecting natural water supplies, it has been a problem in the past, and still is in some areas today. Lead tanks in breweries and cider factories have been found to have had adverse health effects (Coultate & Davies, 1994:124). Lead shot in game birds consumed unintentionally has also been a hazard in the past. The symptoms of lead poisoning include neuropsychological disorders, low IQ in affected infants and children (whose mothers had high lead levels in the blood when pregnant, or who have had high lead intakes themselves while in infancy and childhood), and poor social and learning skills. Methylmercury poisoning is associated with neurological disturbances affecting sight, sensation, and impaired motor activity such as reduced fine co-ordination or severe ataxia (Weiss & Doherty, 1975). Maternal and/or foetal intoxication may impair development and is occasionally

associated with brain damage. Low birthweight has been associated with mercury poisoning, in an inverse relationship with maternal mercury burden (Chen *et al.*, 1979). Although congenital malformations are “rare” (Weiss & Doherty, 1975:311), this does not rule them out. They include cerebral palsy, mental retardation, blindness, deafness, and severe motor handicaps (Weiss & Doherty, 1975:312). Mercury compounds pass freely between mother and foetus and also occur in breast milk. Females working in the hat-making industry in the past (18<sup>th</sup> century onwards; pers. comm. Marion Kite) could have passed on problems to their children, as their chronic exposure would have ensured the presence of gastro-intestinal disturbances, kidney problems, mouth ulceration and anaemia (Martin, 1994).

*Pottery glaze* has also been a hazard in the past. Earthenware pots glazed at less than 1200° centigrade have not had the lead content rendered sufficiently insoluble. Thus leaching of lead occurs into foods cooked or stored in such vessels, especially acidic foods such as pickles, fruit juices, cider, wine and vinegar.

Over one hundred and fifty types of mould spore have been shown to produce toxins on foodstuffs when left to grow. The most popular targets are cereals, and in the past, storage conditions must have been a major source of the problem. Not all moulds are harmful, however. Those used in cheese making, for example, and those found in many jams are usually harmless.

Some foods contain toxins in their natural state. For example, green potatoes contain a poisonous alkaloid, *solanine*. This develops following exposure of the potato to sunlight and the toxin is concentrated just under the potato skin. Peak incidences of anencephaly and *spina bifida* (ASB) have pointed to a relationship with potato quality, the suggestion being that the May-June poor-quality potato period coincides with the prevalence of ASB among affected babies conceived in late April. Alternatively, it has been suggested that metabolites of infected potatoes may reach a peak following winter storage, in May or June, the likely time of teratogenesis (Renwick, 1973:321), although the seasonal effect does not always occur in “affected” countries (Emanuel & Sever, 1973b: 326).

Geographical concordance between ASB and potato-blight severity has also been noted (Renwick, 1973:321), although other authorities questioned this (Emanuel & Sever, 1973:326). Renwick went as far as to suggest that “to minimize the risk of any malformation, not just ASB, no unnecessary products from infected plant (or animal) foods should be taken” by “women of child-bearing age in whom conception is likely” (Renwick, 1973:323). Other vegetables also contain toxic substances. Raw peas and beans, for example, contain *protease inhibitors*, which inhibit the uptake of proteins in the small intestine by restricting the protease enzymes. Prolonged eating of uncooked vegetables produces symptoms of protein deficiency. *Lectins* are also found in legumes - these bind red blood cells into clumps if they enter the bloodstream and cause clotting. Kidney beans are especially associated with this. Damage to the gut wall may result, further inhibiting nutritional uptake. *Alcohol*, when taken in excess or in large quantities regularly, is associated with increased rates of birth defects (Little & Sing, 1987). Ethanol levels in the foetus are directly related to maternal alcohol intake, and

may result in defects of the CNS, morphological defects, growth retardation and spontaneous abortion (Little & Sing, 1987:59). *Myristicin* is found in nutmeg, black pepper, carrots and celery. If taken in large quantities it may produce symptoms similar to alcohol poisoning, including hallucinations. *Comfrey*, often used as a herbal tea, is known to contain the alkaloid *symphetine*, a carcinogen. Crustaceans are frequently associated with food poisoning. This is usually seasonal, with peaks at certain times of the year. Plankton (on which crustaceans feed) are known in warm waters to turn the sea pinkish-red, as a huge number of red-tinted dinoflagellates (plankton type) proliferate. These are associated with toxic symptoms in those eating the shellfish that have consumed the plankton. Similar toxins are known in the North Sea, causing related symptoms. In the worst cases these can result in paralysis and death. (Coultate & Davies, 1994:114-117).

#### **4.3.v.b Micro-organisms in food which may affect reproductive health**

As humans and micro-organisms share many of the same nutritional requirements, human food is easily contaminated if poorly stored, cooked or prepared. The potential for acute food poisoning is well recognised today, and may be imagined in the less hygiene-conscious methods of the past. In the absence of cold storage, and of strict regulation of butchers, farmers and traders in food, it is likely that food contamination was a daily occurrence for most of the population in the past. This is likely to have been a greater problem in the urban environment, where people consumed food and drink that was often prepared elsewhere, and open to adulteration (White, 2000; Wilson, 1973). In rural areas, most food processing and cooking was probably done on a domestic scale (Hagen, 1999) with householders feeding themselves and having an interest in doing this as safely as

possible. However, as there was probably little or no understanding in the Medieval period of basic personal hygiene, such as the need to wash hands before eating or preparing food, there must have always been high rates of contamination of food by the faecal-oral route.

A complicating factor in human infection is that bacterial contamination of food is usually invisible, whereas moulds are readily seen, and may be avoided. Bacterial contaminants do not usually “appear” until sickness occurs, by which time the cycle has likely spread to others. The harmful organisms belong to the genera *Fusarium*, *Penicillium* and *Aspergillus*. For example, the spores of *Aspergillus* secrete aflatoxins, which vary widely in their strength. Experimental animals have been shown to be susceptible to minute traces of these - 15 parts per billion. They are particularly related to problems with the liver, including cancer and hepatitis (Coultate & Davies, 1994:118). Contaminated feed for animals can lead to milk contaminated with aflatoxins. This may have been a problem in the past with domestic dependence on the “family” cow and her untreated milk.

Risks from pathogenic organisms in foods are far higher than those from toxic agents, and as well as inadequate hygiene practices in food preparation, many organisms pass into food via the faecal-oral route following excretion in faeces and urine (Southgate, 1993:349). Infection occurs when harmful bacteria are ingested and cause illness. In the gut they proliferate and cause the symptoms of a specific disease, salmonella, for example. Intoxication occurs by the ingestion of bacterial agents, whose secretions then cause illness (Coultate & Davies, 1994:119-120). *Staphylococcus aureus* is the most common intoxication

agent known to humans. The organism is present naturally on human skin and in the mucous membranes of the nose and throat in large quantities. The potential for spread between individuals is therefore great, occurring by shed skin, touch, sneezing, coughing and so on. The organism grows on food and then secretes a toxin. Cooked meat and foods containing cream are the easiest targets, but any unconsumed food left unrefrigerated after cooking is vulnerable. One microgram ingested by an adult will produce symptoms within one hour. Vomiting, diarrhoea, fever, sweating, headache, muscle cramps and so on follow. The organism is kept in check today by strict regulation of food handlers in the trade, but in the past, it must have been very common, if not the norm. A foetus *in utero*, especially undergoing organogenesis, may be adversely affected by such an infectious episode in ways which are not yet fully understood.

#### **4.3.v.c Intestinal parasites, nutritional and immune status**

As the gastro-intestinal tract is a principal site of exposure to the environment, human and animal populations are at constant risk of ingesting not only pathogenic substances such as micro-organisms, but also parasites, through it into the body (Kagnoff, 1984:240-1). The commonest transmission route to humans is the through consumption of undercooked pork and beef, or of contaminated vegetable matter (Southgate, 1993:352). Numerous types of parasite afflict human populations. These include single-celled protozoa, platyhelminths (flatworms), nematoda (roundworms), arthropoda (insects, mites, ticks), and others (Markell *et al.*, 1992:11). Parasites are mostly descended from ancient lineages, with their origins in the distant past. Schistosomes, for example, are derived from the blood flukes of the Permian era, and have thus had 250 million years in which to perfect

their survival in host tissues (Capron & Dessaint, 1985: 455). They commonly affect millions today in tropical climes. Worms and protozoa live in the gastro-intestinal tract and sometimes its associated structures. They deprive the host of numerous essential substances required for normal health. Hookworms for example, suck blood and therefore deplete the host's iron complement, which may initiate or exacerbate anaemia; others can damage the intestinal wall or invade the bile duct or other structures, and inhibit their action (Kent & Stuart-Macadam, 2000; Markell *et al.*, 1992:9). Parasites of the intestines generally provoke an intense humoral response with production of antibodies in great numbers, particularly IgE (Capron & Dessaint, 1985:457, 471). Not only is the parasitic organism present in the host, but also its metabolic products. Such colonisation and production of by-products can lead to diarrhoea, dysentery and hepatic (liver) abscess (Markell *et al.*, 1992: 380), all of which may have an adverse effect on the foetus if a woman is affected, either during or before pregnancy. Damage to the lining of the gastro-intestinal tract may involve the destruction or reduction of Peyer's patches, the areas of lymphoid tissue in the small bowel vital to the efficiency of the immune system (Kagnoff, 1984; Reeves & Todd, 1996), which would result in impaired lymphocyte activity. Intestinal parasites are also implicated in malabsorption of micronutrients, but Knight believes that their role in this is overstated by many (Knight, 1982:171). As well as colonisation of the intestinal tract, parasites also infect bone and joints (Knight, 1982:158). Hydatid cysts in the long bones, vertebral column, skull base and ilium have been recorded (Knight, 1982). Bone is eroded as colonisation proceeds and spontaneous fractures may occur. Spontaneous union never occurs and today, surgical repair is necessary. The knee and ankle joints are often infected by dracunculus worms and



the resulting pain, immobility and effusion can lead to fibrous ankylosis (Knight, 1982:158). Knight noted that this leads to economic hardship in agricultural communities today when left untreated, particularly when a secondary bacterial infection adds to the primary parasitic lesion. Maternal infections may result in infected foetuses presenting with microcephaly, microphthalmia, blindness and mental retardation (Knight, 1982:174-5; Warkany 1971). Toxoplasmosis (*Toxoplasma gondii*) is the commonest congenital parasitic infection seen today, but only if the mother is suffering a primary acute infection can the infection pass to the foetus (Knight, 1982:174). Larsen (1977) found that multiple organ damage and/or dysfunction resulted from this protozoan infection. Hydrocephalus, microphthalmia, retinal lesions, fever, convulsions and skin rashes occur in infants. This infection is found in undercooked meat, contaminated soil and direct contact, especially with infected cats or their excrement. Wild rodents also carry the protozoa and their excrement is as infectious as that of cats (Larsen, 1977:214). The opportunities for this to spread in the Medieval period are obvious.

Environment is a significant factor in parasite infection - parasites may be carried between human-human and animal-human by arthropod vectors such as flies, which rest on infected material and contaminate their next contacts (Markell *et al.*, 1992:11). Any environment that is characterised by unsanitary conditions, with a population unaware of basic hygiene practices, is liable to parasitic infection. Hygiene has a significant effect on the quality of nutrition, and Markell *et al.* (1992:373) state that malnutrition may be an important factor in determining the severity of parasitic infection. They cite work by Chandra which found that T-

cells are reduced to less than 50% efficiency in protein-energy malnutrition (see above) and suggest that a parasitic infection on top of such a condition could be devastating (1992:373). Sher (1992) found that T cells are probably pre-eminent in the body's immune response to helminths (1992:392). Infection and gastrointestinal parasites are closely related, and may even be each the result of the other. Chronic fungal infections occur in individuals with iron-deficiency anaemia and impaired cellular immunity (Chandra, 1980:76; Scrimshaw, 2000). Chandra stated that "nutrition appears to be a critical determinant of susceptibility to infection" (1980:77), and that both acute and chronic infections can produce losses of body weight and muscle, contributing to malnutrition. In addition, infection tends to lead to reduced appetite, and food intake is reduced (1980:77). Body constituents are lost in vomit, sweat, urine, and stools (1980:77) and in Medieval populations suffering from chronic diarrhoea, with or without gastrointestinal parasites, the malnutrition-infection cycle must have been well-established. Therefore, much infection actually reduces the body's ability to fight it. Reeves and Todd (1996) noted that the impact of disease on the body, protein-loss disorders and malnutrition, poor diet, chronic inflammatory diseases (e.g. parasitic infection) persistent infections and skin damage such as burns or eczema all contributed to impaired immune response. The frequency of these problems in the Medieval period needs little clarification.

Lymphoid tissue in the gut (GALT - gut-associated lymphoid tissue or MALT - mucosa associated lymphoid tissue) makes up approximately 25% of the intestinal mucosa. Any agent, for example worms, or gastro-intestinal inflammation, which causes damage to such tissue may therefore have a severe effect on the

immunocompetence of the host, particularly bearing in mind that the gut is a prime site of interaction between the host and environmental pathogens. Kagnoff states that Peyer's Patches "appear to hold the key to the initiation of the mucosal immune response to many enterically encountered antigens" (1984:240). Other GALTs include intraepithelial lymphocytes and *lamina propria* lymphocytes (both found in the lining of the small bowel). These contribute T cells to the immune response, the former producing T cells of the cytotoxic-suppressor lineage and the latter, T cells of the helper-inducer lineage (Kagnoff, 1984:241-2). Any impairment to this function severely reduces the immunocompetence of the host, as found by Chandra (see above).

#### **4.3.vi Infection and immunosuppression**

Chandra (1978 and since) has looked at depressed immunocompetence amongst offspring of immunocompromised mothers, but has not analysed the rates of birth defects associated with this phenomenon. Chandra, and Scrimshaw (2000) also noted the relationship between reduced cell-mediated immunity and the frequency of infections. Micro-organisms including the measles, infectious mononucleosis, lepromatous leprosy, miliary tuberculosis, spirochaetal and bacterial infectious agents are all known to "markedly" suppress cell-mediated immunity (Chandra, 1980:77). Cell-mediated immunity involves T-cells, whose regulatory roles include defence against intracellular bacterial and protozoal pathogens, fungi and viruses (Ferguson, 1993). Trauma is also implicated in the nutrition-immunity relationship. Individuals who suffer trauma, and often subsequent sepsis, are subject to metabolic changes which have an effect on the whole body. These include hyperglycaemia, generalised fluid and electrolyte disturbances and loss of

nitrogen in urine. Responses to sepsis include reduced oxygen uptake, hyperglycaemia, and reduced metabolism of fatty acids and glucose (Broom, 1993:457-8). Both trauma and sepsis result in an effective loss of proteins from body tissues and this results in amino acids being moved to the immune system, which can actually result in tissue damage and unwanted host responses (Broom, 1993:463).

#### **4.3.vii Summary of teratology, immunity and nutritional status:**

These sections have brought together the elements of teratology, which considers the effects of insults to the foetus, particularly during organogenesis, nutritional status, and the immune response. The close relationship between nutritional status and the strength of the immune response is suggested by the author to be a possible mechanism in the aetiology of some birth defects of the axial skeleton. Reduced availability of nutrients, and/or raised infection rates within the mother (perhaps even when she herself was *in utero*) may be expressed by the foetus as developmental anomalies resulting from the imbalance between her ability to fight infection and the onslaught of infectious agents. These effects may be summarised as follows:

- the immune system is affected by compromised nutritional status, and *vice versa*
- pregnant women with impaired nutritional and immune status are likely to pass on these deficiencies to the foetus as imperfections in development due to lack of nutrients/ability to fight infection (e.g. folate deficiency and *spina bifida occulta*)

- recurrent infection rather than specific disease *per se* may be a possible causative factor behind the rate of defects - this is the author's hypothesis and is related to population density and its health implications
- this appears to be backed up by various sources in the medical literature which acknowledge the relationship between nutrition, immunodeficiency and infection rates, although there does not appear to have been any attempt to bring the arguments about infection together with reproductive health *in terms of birth defect rates*.
- active disease may have a teratological effect on the foetus simply because the presence of pathological processes within the mother's body renders the mother more susceptible to further opportunistic infections
- if we can see that urban populations suffered higher overall rates of disease and infection (as observed in skeletal material and through documentary evidence) than rural populations, we may be able to suggest, by referring to the medical literature, that birth defects rates must have been correspondingly higher amongst urban populations.

#### **4.4 EPIDEMIOLOGICAL INVESTIGATION AND BIOLOGICAL ANTHROPOLOGY**

This study considers certain aspects of the reproductive health of the populations in rural and urban environments. By investigating the possible differences in prevalence of axial skeletal birth defects between people from these two types of environment, the author is conducting a type of epidemiological study. This section considers the field of environmental epidemiological study, relating environmental factors to health issues, and shows how such investigation mirrors the biocultural approach taken by

biological anthropologists. It is the suggestion of the author that, having undertaken the study of archaeologically-derived human material by such methods, similar studies may be devised focussing on modern populations undergoing demographic transition.

#### **4.4.i Types of epidemiological study**

Epidemiological investigation may either *describe* the pattern of occurrence of a disease, or *analyse* the influences that determine the frequency of that disease (Barker & Hall, 1991). Some studies do both. In medical terms, studies may look at specific diseases, or syndromes, for example, Kelsey and White (1980) investigated the epidemiology of back pain. Analysis reveals the distribution of *attributes* (e.g. age, sex) or *variables* (e.g. time or place in which the disease developed) which may be affecting frequency rates.

*Descriptive studies* are usually cross-sectional, that is, they take a “slice” of a population at one point in time. They may determine the frequency of disease, define the population suffering from it, and show where and when disease occurs. Longitudinal studies, in which observations are repeated in a community over a prolonged period are becoming increasingly popular (Barker & Hall, 1991). Investigation of archaeological cemetery samples fits midway into this theoretical framework (Waldron, 1994). Excavated graves represent the population of the cemetery at one point in time (during the cemetery’s period of use) but also represent a kind of longitudinal study in

that they portray the annual dead of many years (unless the bodies are from a battlefield, for example).

*Analytical studies* in living populations generally set out to test a hypothesis about the influences determining the occurrence of a disease. They seek a cause-and-effect relationship between A and B. Such studies follow one of two methods:

- a comparison of diseased people with unaffected people, showing that the determinant occurs more frequently among those affected than those not affected
- a comparison of people exposed to the determinant with those not exposed, showing that a greater proportion of people develop the disease among the exposed than the non-exposed (Barker & Hall, 1991:8).

This biocultural study of archaeologically-derived human skeletal material equates to the second method. Exposure to an urban environment and its raised infection rates is the hypothetical determinant to which the populations are either exposed or not exposed. It embodies features of a cohort study but also shares some of the characteristics of a case-control study. Cohort studies address a group of people who are exposed to a suspected determinant - they are a group with a shared experience.

Archaeological samples cannot fit this definition exactly, as in living populations cohort members are healthy when assigned to the cohort, and then assessed over time for the development, or not, of the disease. Case-

control studies address the identification of a group of people with a disease (Barker & Hall, 1991; Siemiatycki, 1995) and show the proportion of cases of a disease that may be caused by a determinant (Barker and Hall, 1991:10). A cohort study gives results of an association between environment and disease as an *attributable risk*. For example: two cohorts, a rural and an urban group, are compared. If the urban group has a higher frequency of birth defects (A) than the rural group (B), then A-B is the *attributable risk*. The magnitude of the effect (the strength) of a cause can be expressed as  $a/b$ , which is the *relative risk*. The analysis of a cohort study shows the “proportion of people in whom exposure to the determinant results in development of the disease” (Barker & Hall, 1991:10).

Disease frequency may be expressed simply as the number of individuals affected as a percentage of the whole population (Barker & Hall, 1991). In archaeological contexts this will rarely be possible, as the entire population at any given time will be unknown. However, by expressing a percentage figure for disease frequency, we may at least define the “burden” on the excavated sample(s) (Waldron, 1994). In modern aetiological studies the *incidence* - the number of new cases occurring in a population within a specific time - is of more use than the *prevalence* - the number of cases within a population at a given point in time (Barker & Hall, 1991:25; Waldron, 1994:42). Unfortunately, archaeological samples are restricted to the calculation of prevalence. It is impossible to know the incidence unless, for example, the graves are documented with the date of death. However, this should not be considered too great a problem. For example, field survey



studies carried out in the developing world, based in outlying clinics, also have to work with somewhat imprecise identification data (Barker & Hall, 1991). For example, clinic patients may have to be vaguely identified as “coming from the catchment area” of the clinic, perhaps identified by some geographical or political feature such as regional boundaries (Barker & Hall, 1991). Likewise, biological anthropologists may identify their “patients” from excavated cemeteries in the same way. Catchment areas and access to clinics (or cemeteries) within them are affected by geographical, political and economic constraints today as much as they were in the past. Patients may originate elsewhere but come for treatment at a specific clinic anyway, in much the same way that the occasional “alien” burial may occur in an urban or a rural graveyard. In the same way, some patients may choose to be treated at a clinic outside of their own catchment area (or in this case be buried in a cemetery outside their own catchment area). We do not know that all of our urban/rural burials are life-long urban/rural inhabitants, but neither do all epidemiologists know the exact origin of all of the study-subjects in remote places in the developing world. Epidemiologists must reach conclusions based on such imprecise identification data, or risk no results at all. Biological anthropologists are compelled to do the same when working with incomplete materials as a data-source.

#### **4.4.ii Types of information sought and gained (see fig.4.2)**

Attributes and variables that are considered by epidemiologists and biological anthropologists may be summarised in the table below. It will be seen from the table that the biocultural approach enables the biological anthropologist to

come to more significant conclusions than may be initially thought.

Epidemiologists consider local and general environmental factors such as geographical environment, climate, altitude, soil composition, parasites, insect vectors, and pollution. Residential environment may be assessed for exposures or conditions which affect individuals in the home and work-place, such as smokey atmospheres, over-crowding in the household, relative lack of exposure to sunlight, diet, amount of outdoor activity, and so on. By doing so, epidemiologists are looking for the *time* of exposure as well as the determinant. In this study, the time of exposure is already identified as the nine months spent *in utero*, particularly the embryological stage in the first trimester. Archaeological and historical sources may identify very broad, generalized environmental conditions in which women lived before and during pregnancy, for example, in terms of housing type and quality, location of industrial processes, social and kinship arrangements, diet, sanitation and water supply. Some archaeological evidence is available to illustrate these conditions, particularly relating to York (for example, Dawes & Magilton, 1980; Magilton, 1980; Richards, 1983; Hall *et al.*, 1988; Bond & O'Connor, 1999), but the transient nature of the population passing through Aldwark, for example, means that the burials in St Helen's cemetery would not all have been life-long inhabitants in the vicinity. Occupants of the Chichester hospital, on the other hand, were probably Chichester residents prior to their admission as this was usually a pre-condition of entry to such institutions (Clay, 1966), but specific living conditions are still impossible to define. Archaeological environmental evidence for the observed rural settlements is available in broad terms also, with the remains of plant, animal and

technological materials from the local surroundings being documented (for example, Andrews *et al.*, 1979). However, the aim of this study is to *broadly* identify environmental conditions, and whilst it is possible to report *some* environmental evidence from archaeological sources, there is no possibility of providing a definitive environmental reconstruction in which the populations under examination lived. Archaeologically-derived environmental evidence has been described earlier, in Chapter 2.

**Table 4.1** Attributes and variables recorded by investigators

Attribute/variable	Epidemiologist	Biological anthropologist
age	yes	subadults, yes; adults to broad age categories
sex	yes	yes, if skull and/or especially pelvis present
ethnic group	yes	Occasionally (race by skull; documentation)
marital status	yes	only with documentary evidence
family structure	yes	with doc. Evidence; familial traits
occupation	yes	<i>possibly</i> by grave goods/ doc. evid. (difficult)
socioeconomic status	yes	doc. evid; gen.health/size (pop.level); archaeological environmental evidence
place of origin	yes	doc. Evidence; isotope analysis of teeth etc.

One aspect of health that is frequently studied by epidemiologists is the effect of the movement of migrants, both on their own health and that of the host population among which they settle. In the absence of documentary or obvious physical evidence, the identification of migrants within an archaeological cemetery is difficult, if not impossible. The obvious exceptions to this in the archaeological context are burial grounds specifically for socially controlled groups, for example, the Jewish cemetery at Jewbury,

York. Unfortunately, study of such remains is subject to religious restrictions and is often not allowed, such as in the Jewbury case (Lilley, 1994). Recent work by Budd and others (1997, 1998, 1999, 2000) addresses identification of origin by isotope analysis, although this simply identifies broad areas of origin in which the individual possibly grew up, and does not explain when the individual moved into the “new” area. Comparison of metrical data and analysis of non-metric traits may help to identify incomers in cemeteries.

#### **4.4.iii Cause and effect in environmental epidemiology: the role of archaeology**

The establishment of causal relationships in environmental epidemiology is a vexed one. Much research today is undertaken for regulatory agencies, whose job it is to decide on safe exposure limits in the home and workplace. Other research defines disease-control measures and the prediction of epidemics (Traven *et al.*, 1995). This work all relies on the living subjects, accurate medical records, adequate opportunity for suitable subject identification and follow-up, the recognition of relevant variables, and appropriate questioning to draw out the required information. All of this takes time, and even when reliable, does not always produce answers to the questions asked. However, “careful evaluation of the combined evidence from epidemiologic and *other research* [italics mine] helps to gauge the likelihood of an association or a causal relationship between a specific risk factor and a specific disease” (Traven *et al.*, 1995: 39). Archaeological inquiry surely has a role amongst the “other research”, though the authors of this statement may never have considered the possibility. A substantial quantity of human material, excavated from cemeteries and other burial sites now lying in many

collections around the world, offers a window on the health of past populations (although documentary and archaeological evidence may also offer insights into health risks in the past). Even so, it seems only a matter of common sense to apply appropriate environmental epidemiological methodology to this material in an attempt to answer some of the difficult questions regarding causality that we ask today. For example, one of the aims of this study is to associate skeletal disease markers with possible soft-tissue anomalies by learning from the clinical literature. By applying that knowledge to our interpretations of observed skeletons it may therefore be possible to “see” more than the bones alone can tell us.

#### **4.4.iv Human Health And The Environment In The Past**

*“Over the course of their short stay on earth, humans have helped to create their plagues, poxes and pestilence by unwittingly fashioning the kinds of circumstances that brought them forth and then, at times, almost compulsively improving those circumstances so that the diseases flourished...”* (Kiple, 1997:6)

Skeletal markers of disease are representative of many ailments which afflicted human groups in the past, and which may still be observed amongst modern populations. The differences that are most obvious to the researcher are the changes in the *patterns* and *frequency* of disease across time and space (Roberts & Manchester, 1995, 10). For example, hunter-gatherer groups appear to have enjoyed relatively good health compared to later agricultural societies, a phenomenon that may be at least partially explained by their largely non-sedentary way of life. By practising a nomadic or semi-nomadic lifestyle, they were able to avoid some of the problems associated with sedentism, such as the accumulation of domestic rubbish, vermin infestation

of dwellings, the need to use permanent cess-pits and so on (Cohen & Armelagos, 1984; McKeown, 1988; Bond & O'Connor, 1999).

#### **.4.iv.a** *Health implications of agriculture*

The agricultural lifestyle originating in the Neolithic period resulted in a large increase in the number of infectious diseases suffered by humans (Cohen & Armelagos, 1984; Roberts & Manchester, 1995:11). This rise in infection rates is believed to be associated with the efforts farmers made to control the environment to their own advantage. Farming involved ploughing and close contact with soil, animals and refuse, as well as a sedentary lifestyle that involved living in houses, with their attendant dark, smokey and probably damp interiors. Infectious agents such as the tetanus bacillus, released from the soil, must have killed many individuals. Likewise, close proximity to animals, often in the same house, exposed humans to the infectious agents that live within those animals, as well as increasing the risk of personal injury by traumatic accidents with large animals. Many human diseases are now believed to be derived from animal sources (zoonoses): tuberculosis from cattle, and possibly measles from canine distemper, for example (Kiple, 1997). Today, we share sixty-five diseases with dogs, fifty with cattle, forty-six with sheep and goats, forty-two with pigs, thirty five with horses and twenty six with poultry (Kiple, 1997:24). Most of these diseases were unwittingly admitted into the human experience due to the increased contact between humans and animals that occurred on a large scale only with the onset of the agricultural lifestyle. Parasitic infestation, another human health problem associated with food storage and inadequate cooking, is also

associated with animals and humans living in close proximity (Knight, 1982).

Such infestation may have affected those hunter-gatherers who practised some degree of food storage, but evidence for this is not available.

Practices such as spreading waste matter onto fields, neglecting latrines, and not keeping drinking water free from waste, all contribute to the spread of disease from animal to human, and human to human (Craun, 1995). In addition, increased susceptibility to infection is strongly-associated with dietary status (Golden, 1993). Hunter-gatherers ate a wide-ranging diet of animal and plant products, high in fibre and relatively low in fat. Food was consumed quickly while it remained fresh - storage played little or no part in most hunter-gatherer societies. The nomadic lifestyle also ensured regular and effective exercise. By comparison, agriculturalists ate a poorer diet and were more sedentary, taking less healthy exercise - the bending and lifting associated with farming, whilst still being hard work, did not confer the same health benefits as semi-permanent mobility, being less aerobic. Food types available to farming groups were less varied, typically based around one or two staples. This, of course, meant that any crop disaster could result in starvation or at least severely reduced food availability. Less meat was likely to be consumed (animals were valued for their secondary products such as wool and milk) and fibre content was likely to be reduced. Diseases such as scrofula, a tuberculosis of the lymph glands in the neck, were associated with use of dairy products from infected cattle and other animals. Ergotism is believed to be associated with a toxic fungus that forms on poorly stored damp rye (Kiple, 1997, 25). Deficiencies in vitamins and iron would have become commonplace due to an over-reliance on cereals in the diet (Stuart-

Macadam, 1989a; Kent & Stuart-Macadam, 2000). The consumption of the milk of farm animals also led to the transmission to juveniles and adults of disease agents present in untreated animal milk. Traumatic injuries also increased with agriculturally-related sedentism. Hunter-gatherers suffered relatively fewer traumatic injuries, and it is believed that the rise seen in farming communities is related to the competition for food due to a rising population. Combined with underlying poor nutrition, any wounds or fractures could have been subject to delayed healing. Farming is associated with water and soil pollution, increased animal contact and exchange, decreased dietary variety, decreased immune status, raised anaemia rates, raised birth rates, and raised parasite infection rates (Kiple, 1997: 8-9).

#### **4.4.iv.b** *Rural and urban comparative health*

Prior to the industrialization of the 18<sup>th</sup> century, 80% of people in Britain lived in a rural environment (McKeown, 1988:44). However, urban conditions in later Medieval towns were not directly comparable to modern industrialized towns and cities. The principal differences between town and country in the pre-industrial age were food availability, population density, increased long-distance contacts through trade and travel, and the special conditions that surrounded the craft specializations (Bogin, 1988; McKeown, 1988:46). Urban pollution as it is understood today was a feature of later industrial cities. The differences between rural and urban living conditions were most evident in housing, hygiene, quality of food and water supply, work and social circumstances and population density (Roberts & Manchester, 1995).



It is suggested in this study that variations in these domestic and occupational factors lead to an increase and change in nature of the pathogen pool that was experienced by both newcomers to towns and inhabitants of longstanding. Exposure to an inadequate or substandard diet and a larger variety of infectious agents can therefore be proposed as a challenge to health that requires some form of adaptation. It is well-recognised that nutritional status has a significant effect on the course of infectious diseases in populations (Golden, 1993): the dynamic interaction between nutrition and infection in humans can result in differing degrees of severity of an illness depending on the long-term nutritional status of the host (Newberne & Williams, 1970, cited in McKeown, 1988:53). The effects of pathogen load and immune status on the body, particularly with regard to groups exposed to new pathogens, are well-documented (Little & Leslie, 1993). It is suggested by the author that the necessary migration from rural to urban environments in the past may have had a similar effect on both incoming and host populations, particularly as rural-incomers would not have had an effective immunity to new diseases that they encountered in towns.

Rural populations lived from the land, being physically active and in close contact with domesticated animals in their everyday life, with their diet composed chiefly of unrefined foods. Their dietary deficiency as a result of poverty was the most serious problem to their health (McKeown, 1988:45). Agricultural diets in the past predisposed populations to deficiencies in protein, vitamins and minerals (Roberts & Manchester, 1995). Infectious diseases were present in rural populations, but these were most active when

the efficiency of transmission was very high, as in sexually transmitted diseases. Relatively small populations with restricted geographical contacts are believed to have been subject to only low levels of infectious disease unless either the human population or the pathogen levels rose significantly. Rural populations were also arguably exposed to fewer human contacts than urban ones, thereby reducing the potential for spread of infection (McKeown, 1988:49). Rural lifestyles differed within themselves depending on the nature of the agricultural practices followed. Pastoral agriculture, relying heavily on animals husbandry as well as crop cultivation, was a less seasonally dependent form than arable farming. The different workloads required during the year lead to distinct patterns of labour amongst the populations, imposing variable stresses and occupations on men and women (Goldberg, 1992).

Many early urban-dwellers in the Medieval period had access to open spaces in towns, as residential, civic, and religious buildings were all constructed amongst orchards, gardens and vineyards, from which produce was grown for the inhabitants (Bogin, 1988:91). Much of these open spaces was largely lost to building in the expansion of urban industry and crafts up to the late fourteenth century (Postan, 1973:52; Holt & Rosser, 1990; Schofield & Vince, 1994). Urban populations tended therefore to suffer from the effects of population density, with endemic infectious and parasitic diseases having a substantial effect on health (McKeown, 1988:62). For example, water supplies had to cater for much higher numbers of people, with water-borne infections more easily spread because of this (Karlen, 1995:129). Poor hygiene and lack of sanitation resulted in sewage disposal and animal refuse

existing in close proximity to both large numbers of people and their water supplies. Low-quality housing, typically timber-framed with wattle and daub panels and thatched roofs, offered unlimited scope for damp, rot and infestation by vermin in the domestic environment. Infectious agents were able to thrive under crowded human conditions, and low host immunity would contribute to the spread of disease. Poor air quality resulting from the relatively crowded, industrial and unhygienic urban setting would also have encouraged respiratory tract infections, as evidenced by Lewis *et al.* (1995), who found higher levels of sinusitis in the St Helen-on-the-Walls, York, population than in the Wharram Percy, North Yorkshire, population. Whilst market and trade centres, especially ports, like Chichester, in Sussex, were arguably able to obtain a variety of foods all year around, reducing the potential for dietary deficiencies; ports and major centres also allowed access to new infections from far away (Karlen, 1995:94). Urban areas, being “catchments”, also suffered increased exposure to infections from the large numbers of people passing through on a regular basis. In addition, the comparative wealth of urban populations compared to rural ones also lead to an increase in the amounts of refined foods consumed, which has been associated with an increase in metabolic disease (McKeown, 1988).

#### **4.4.iv.c** *Population density, urbanization and health*

Along with the agriculturally-associated increase in the number of diseases suffered by humans, the population itself rose sharply with the onset of agriculture (Cohen & Armelagos, 1984; Roberts & Manchester, 1995).

Modern studies show that as population density increases, so does

susceptibility to infection, and diseases spread more quickly (Swelund & Armelagos, 1990). Population density refers to the increased closeness of human to human contact within defined areas as numbers rise (Jackson *et al.*, 1989). Thus, the onset of urbanization and its concurrent association with raised population density is associated with even higher rates of infection than seen previously (Manchester, 1992; Roberts & Manchester, 1995:12). Whilst it is true that farming settlements in the Medieval period could consist of poor-quality homes, with the inhabitants often living in close proximity to animals, the author suggests that residents could probably adapt immunologically to their surroundings and survive them in reasonable health as community numbers were relatively low compared to the population of towns. However, similar living conditions in towns were usually associated with overcrowding and an increased population density - more people packed into the available living space (Jackson *et al.*, 1989). This is known to increase the spread of infectious disease dramatically. Timber housing, thatched roofing, latrines, contaminated wells, refuse disposal close to water supplies, butchery waste in the streets and so on, all brought about conditions in which disease agents flourish (Goldberg, 1992; Hall *et al.*, 1988; Bond & O'Connor, 1999). As population density increased and public health measures failed to develop in time, the spread of disease was, in effect, encouraged (Manchester, 1992:12). Insect and rat infestation was the norm, and people suffered from intestinal parasites from drinking contaminated water and eating undercooked or old meat (Knight, 1982; Markell *et al.*, 1992). Quality of food suffered as the mass-production in towns of ale and bread, for example, became open to adulteration for economic profit, a fact illustrated

by the increasing numbers of prosecutions mentioned in court rolls (Jackson *et al.*, 1989:6-7; Goldberg, 1992). Living conditions favoured the presence of rats (Bond & O'Connor, 1999), and it is through the black rat and its fleas that the plague was initially spread throughout Europe (Ziegler, 1969). Urban centres with densely packed populations were prime candidates for epidemics (Jackson *et al.*, 1989). Combined with a softer, higher-calorie diet with less fibre, dietary deficiencies, particularly of micronutrients, became widespread. Immunocompetence must have suffered in consequence (Ferguson, 1993; Jackson *et al.*, 1989).

Certain infectious diseases begin to appear more frequently in the skeletal record along with urbanization (Manchester, 1992). Plague, for instance, leaves no mark on the skeleton, as it kills its victims before any bone response takes place, but tuberculosis and leprosy have time to act on the bones as they do not always result in a swift death. Both of these diseases thrive in overcrowded environments and are passed on easily by droplet transmission in which the bacilli enter new hosts. The treponematoses yaws, endemic and venereal syphilis also leave markers and the latter is heavily associated with urbanization (Manchester, 1992). Metabolic diseases such as anaemia are represented by orbital *cribra orbitalia* (termed porotic hyperostosis of the orbits and cranial vault in North American groups). This type of anaemia is believed to be due to iron deficiency, which may be traced to a poor diet, or to inhibited uptake of iron due to gastrointestinal parasites (Kent & Stuart-Macadam, 2000; Kiple, 1997:10; Stuart-Macadam, 1989a and b). Scurvy, or Vitamin C deficiency, appears as new bone formation around the jaw and

other areas and is associated with a diet low in ascorbic acid, plus a reduced immune response (Siegel, 1993). Rickets, or Vitamin D deficiency, produces poorly mineralized bones and results in bow-legged individuals (Smith, 1993). This is associated with poor quality housing and little exposure to light (Stuart-Macadam, 1989b). Osteomalacia is an adult equivalent, affecting females through multiple pregnancies, long-term lactation and short birth intervals, resulting in a deformation of the spinal column and pelvis (Roberts & Manchester, 1995). A further dietary consideration is that of the availability of sugar. From the late 12<sup>th</sup> century onwards, it became widely available in Europe and an associated rise in caries and other dental diseases appear in the record (Hillson, 1979). Persistent infection from dental disease could result in recurrent low-grade infection in affected individuals, compromising their general health (pers. comm. K. Manchester).

#### **4.4.iv.d** *Urbanization today*

Urbanization has, then, for centuries, been associated with poorer health than that enjoyed by non-urban populations. This is not to say that rural populations did not suffer significant ill health - they did. Urban centres simply produced the better conditions for disease agents to flourish, and aided by increased population density, resulted in poorer health. Similar effects are seen today in refugee camps, where large numbers of people are thrown together in ill-prepared conditions with overcrowding, poor sanitation, reduced dietary intake, lack of warmth and light, and poor drinking water supply. The greatest fear is usually that of the onset of cholera, and past

experience has shown the devastating effects of such infectious diseases on stressed populations.

The developing world, whilst adopting useful “western” technology in its own period of rapid urbanisation, is also taking on the harmful aspects of industrialised living. The 1997 World Health Report stated that chronic conditions are a major problem in both developed and developing countries. “Increased longevity without quality of life is an empty prize. Health expectancy is more important than life expectancy.” (WHO,1997: v). People in poorer countries are now adopting many of the “bad” habits of the industrialized world - sedentary occupation, reduced physical activity, poor diet, and increased consumption of tobacco and alcohol and drugs. The WHO noted that the majority of chronic conditions are preventable but not curable and that the emphasis today has to be on prevention. There is now a “double burden” of infectious and chronic disease affecting the world’s populations (WHO, 1997). The report also noted that musculoskeletal disorders are common in developed and developing countries - 10%-30% of the workforce in industrialized countries and 50%-70% in developing countries may be exposed to a heavy workload, often exacerbated by unergonomic lifting practices. The principal consequences of this are cardiorespiratory and musculoskeletal disorders. Ecological changes associated with “progress” serve to produce favourable environments in which parasites and the vectors of infection are able to thrive. Nutritional changes resulting from new lifestyles associated with this progress result in altered nutritional states, frequently mal- or undernutrition. This in turn leads to an increase in disease susceptibility due

to impairment of the immune system. For example, iron deficiency is a common side-effect of dependence on one cereal, especially when associated with a low protein intake, as the presence of amino acids in the stomach aids the absorption of dietary iron (Kiple, 1997). Iron-deficiency is believed to adversely affect host immunocompetence, and iron-deficient individuals are often concurrently infected with bacterial or viral organisms (Kagnoff, 1984:253).

Until effective public health measures, and legislation to support these, were implemented in the post-Medieval period, urban living in England continued to be a serious threat to health. Even with such measures in place, problems still arose due to the lack of understanding of such issues as transmission of micro-organisms. Scientific knowledge has enabled effective preventative measures to be taken in the developed world, and the finances to implement such measures have been forthcoming over the last century in particular. The fight for public health elsewhere in the world, however, remains a priority, and until issues such as water filtration, sewage treatment and removal, and education regarding food storage and preparation are adequately addressed, the health of urban(izing) populations will continue to be a drain on the social and economic strength of their communities.

#### **4.4.iv.e** *Human health: the cultural perspective*

Culture comprises technology (the way in which energy and resources are extracted from an environment), social organization (how the society maintains and reproduces itself), and ideology (ideas, attitudes and beliefs) (Dennet &



Connell, 1988). The cultural system often acts as an effective barrier, buffering the population from environmental insults, but it may also be a source of insults as the technology, social organisation and ideology practised may produce the very insults that affect the health of the population. Swedlund and Armelagos (1990) argue that the ecological model (see Chapter 5) can serve as a general construct for organizing the major elements in the disease process. Much more important are the questions asked when the host, insult and environment are categorised. The questions asked and the approaches taken are where the real utility of a descriptive model is tested (Swedlund & Armelagos, 1990). There is nothing in the ecological model that prevents a consideration of political and economic spheres of influence. The biocultural model is virtually identical to the ecological model, and is used as the theoretical framework within which this study operates.

#### **4.4.iv.f** *Human health: the perspective of sex*

Although there has long been a realisation that the health of women plays an important role in the health of their offspring (Emanuel & Sever, 1973a), it is only in the last decade or so that this knowledge has been extended to include the earliest stages of the mother's own life. Barker (1994) stated that his theme was the improvement of nutrition and health of girls and young women, and of mothers during pregnancy and lactation, to improve the health of their children (Barker, 1994:vii). For example, numerous studies have shown links between mothers' own birthweights and the birthweights of their babies (Barker, 1994:122, 124). It is appreciated today that the mother's body and capability to produce healthy children are the result of factors acting on

her from her own earliest development, not simply how healthy she was prior to and during pregnancy. Emanuel and Sever (1973b), in a modern epidemiological study, noted that interference with maternal growth and development was a possible aetiological factor in the frequency of birth defects. They listed a variety of factors which could affect maternal growth, development and health, including nutritional deficiencies, severe illness, poor sanitation, and emotional and socio-cultural deprivation (1973b:329). These factors must have been commonly experienced in the past, particularly in urban environments as well as in rural ones during times of stress. Women are acknowledged to have a disproportionate burden of ill health that cannot be explained by biological differences alone. Their social, economic and political disadvantages already have a detrimental effect on their health (WHO, 1997:82). They also experience differential exposure to risk, access to the benefits of technology, information and services, and ability to protect themselves from ill-health (WHO, 1997:83). Their health is therefore of vital importance to that of subsequent generations, and to overlook this is to store up health challenges for the future.

Stinson notes that there is a commonly held belief within the physical anthropological literature that males appear to have a greater sensitivity to environmental stresses than females (Stinson,1985:123). This is often supported by the suggestion that female resistance to similar stresses is mediated by the female capacity for reproduction, with its own inherent stresses incurred by pregnancy and lactation. In addition, various studies on childhood growth patterns have been carried out in the developing world,

which may add to our knowledge of sex-based differences in response to stress. For example, Bogin and MacVean (1981) found in a Guatemalan study that boys and girls reacted differently to the stresses of the same socio-economic conditions, resulting in differential fat storage and muscle size between the sexes.

The possibility of sex-related differences in response to environmental stress is an important consideration in this study. Data from Raunds and Chichester (Sture, 1997) showed sex-related differences in the frequency of certain congenital defects, although this was simply a picture of those with defects who survived into adulthood, not the numbers who were actually born with them. Clinical data also show sex-related differences (Levene, 1991; Warkany, 1971), with some defects being more likely in one sex than the other (for example, cleft palate is twice as common among males than females). Is environment therefore a factor not only in the frequency of defects, but also in the sex-related frequency? Such observations could be the result of environmental stresses acting on the foetus *in utero*, or on the mother's health, indirectly acting on the foetus's development. Alternatively, could environment be acting on the foetal development of primordial germ cells (the precursors of the gametes) *within each foetus*, thereby damaging its own reproductive health in the future? In this scenario, the foetus itself may be unaffected, but its own gamete-production may be impaired, resulting in defects appearing in the second generation, rather than in the exposed parental generation. This may be supported by observations in Vietnam (Constable & Hatch, 1985) which show that subsequent generations are

affected by exposure of parents and grandparents to chemicals used during the Vietnam war (e.g. by exposure to Agent Orange), even when exposure is no longer a factor for the affected offspring.

Differences in sexual dimorphism have also been investigated. Stinson noted the suggestion that environmental stress causes growth retardation in males, and that therefore in populations undergoing longterm stress, the degree of sexual dimorphism should be markedly reduced. She pointed out that this does not always appear to be the case (1985:124).

#### **4.4.iv.g** *Reproductive influences according to sex*

Aside from the obvious factor of carrying the pregnancy, mothers seem to have more of an input to the growth and development of the foetus than do fathers (Barker, 1994), which may suggest that there may be a greater maternal role in the formation of birth defects. For example, Barker cited studies that compared the birthweights of half siblings and of cousins. These studies showed that relations through the maternal line had relatively similar birthweights whilst those related through the paternal line did not (Barker, 1994:121). Whether other aspects of foetal growth and development are directly related to solely maternal influences rather than to the input of both parents is uncertain, but the “conflict” between maternal and paternal genes in the foetus, making demands of the mother’s tissues during pregnancy, is well-documented (Barker, 1994:122). Genetic input, along with the nutrition and oxygen levels available to the foetus, appear to be prime influencing factors in infant development (Barker, 1994:121). For example, Little and Sing

(1987) found a significant relationship between the drinking habits of the father and infants' birthweight that was independent of maternal drinking. They stated that there was no increase in congenital defects noted due to paternal drinking, but based on the premise that low birthweight is strongly associated with increased birth defects (Levene & Tudehope, 1993 and others), there may well be some unidentified aetiological factor at work.

The question of parental environment as the causative factor in defect aetiology relates to research into defects that are already strongly associated with environment, for instance, radiation. Whilst the rubella virus is known to affect the foetus adversely at a specific period of gestation, and is therefore directly related to the mother's environment, the effects of radiation are not directly attributed to the mother *or* the father of the foetus. Are the mother's ova *or* the father's sperm affected by the radiation prior to conception to the detriment of the foetus? If the father's sperm quality is in question, then it may be the result of a continuous or semi-continuous process of exposure, as sperm production is an on-going process during a man's lifetime. If the quality of the mother's ova is in question, then this may be the result of some event during the mother's own gestational period, as females are *born with* their full complement of ova. On the other hand, if the foetus itself is the source of origin of the defect, then it follows that the foetus has been exposed to some environmental agent via the mother, during gestation. This study's hypothesis of detrimental environmental effects on human foetuses could be related to males, in that their environment could interfere with spermatogenesis, or females, in that their environment

could affect their pre-pregnancy and pregnant health status. Both of these scenarios could possibly result in skeletal defects present at birth.

#### 4.4.iv.h *Human health: occupational risks and socio-economic factors*

The World Health Organisation Report 1997 (WHO, 1997: 64-66) outlined the following as current issues in the effects of occupation on health exposures:

- hazards of work environment include exposure to *chemical or biological agents*, and adverse factors such as *ergonomic, psychological or psychosocial* problems (Knoblauch *et al.*, 1996; Richards, 1993; Ursin, 1994)
- workers in the chemical industry may not show ill-effects for many years (Garcia & Fletcher, 1998; Knoblauch *et al.*, 1996; Richards, 1993)
- *chemicals* may affect the *immune system*, leading to dermal or respiratory allergies/effects, and *increased susceptibility to infections, especially in the gut*, and the genito-urinary systems, in both males and females (Chandra, 1978, 1979, 1980; Palmer & Coggan, 1997).
- *metal poisoning*, eg. lead, mercury (Aswathi *et al.*, 1996; Chen *et al.*, 1979; Kolev *et al.*, 1996; Weiss & Doherty, 1975).
- reproductive disorders [unspecified] (Aswathi *et al.*, 1996; Farrow *et al.*, 1998; Garcia & Fletcher, 1998)
- biological agents including *viruses, bacteria, fungi, moulds* and *organic dusts*
- *parasitic diseases*, asthma, allergies, tuberculosis and hepatitis infections are the commonest responses to these (Knight, 1982; Markell *et al.*, 1992).

- *allergic dermatoses, respiratory tract and skin surface problems* are the commonest routes by which agents enter the body (Kagnoff, 1984; Lessoff *et al.*, 1987).

(The italicised factors in the above list may apply to Medieval environments as well as modern ones).

Studies of women at risk at work tend to concentrate on carcinogens and mutagenic agents affecting reproductive health. Organic solvents and toxic metals are the worst culprits for reproductive problems, but many biological agents, and heavy lifting (Xu *et al.*, 1997), are also associated with reproductive disorders (WHO, 1997:64-65). In addition, it is recognised that women have a double load of family rearing and work outside the home, with all of the stress that this involves (WHO, 1997:82).

Farrow *et al.* (1998) studied 14,000 pregnant women, considering their jobs since the age of 16, and the socio-demographic factors affecting them. They found a significant discrepancy in birthweight between the nine major job-groups identified. A difference of 148g showed between the mean birthweights of the babies of professional and plant/machine-operation women, with the non-professional mothers having the lighter babies. However, after adjustment for smoking, caffeine intake, height and ethnic origin, the job was no longer a significant factor in determining birthweight. Even so, the lowest birthweights occurred amongst the mothers working in metal forming and welding, and the textile trade (1998:23), that may be attributed to greater physical stress.

Emanuel and Sever (1973b) reviewed the evidence for social and economic factors in the aetiology of some birth defects, and rates of foetal mortality and low-birth-weight. They noted that there was an inverse relationship between foetal mortality and low-birth-weight, and the social status of the baby's *father* and *maternal grandfather* (1973b:327), suggesting that environmental factors continue to have a significant effect down several generations. It was also apparent in their study that short women within all social classes and age categories produced more defect-affected babies than the tall women in the same groups. They suggested that there is some mechanism at work that relates to growth disturbances in the mother (1973:327). This work appears to support that of Barker (1994).

Knox and Lancashire (1991) considered maternal age and birth rank as part of their study. They found that low birthweight was a common factor amongst first babies and explained this as the result of immature uterine vascularity and "other factors" in the mother-foetus relationship. Subsequent babies were usually bigger. The highest rates of defects were found amongst the teenage mothers under sixteen years of age (1991:47). This rate decreased in the next age band (16-19 years) and thereafter showed a slow increase in rates again as age increased. The high rates amongst the youngest mothers was suggested to be partly due to the effects of immunisation for infection (e.g. rubella vaccines at 12 years or thereabouts). Social class showed variation in the rate of birth defects, with the professional classes showing lower rates than the intermediate and unskilled classes.



Skjaerven *et al.* (1997) found a relationship between the birthweight of the mother herself and the survival of her offspring. In cases where the mother herself had weighed less than 2000g at birth (2500g is the cut-off point for low birthweight - LBW), the infant's chances of survival were severely reduced. Infants born to such mothers had a high risk of dying in the perinatal period. This suggests some mechanism acting in the mother-foetus relationship, possibly at an early stage. LBW is significantly related to birth defect frequency (Allen, 1986; Levene & Tudehope, 1993) and, although Skjaerven *et al.*, (1997), did not specify cause of death, it is to be assumed that a significant number could have been related to birth defects. Davey-Smith *et al.* (1997) found that socio-economic factors acting over a lifetime affect both health and the risk of premature death. Adults who have suffered protein-energy malnutrition resulting in stunted growth are at a disadvantage regarding their physical capabilities. They have a lower capacity for physical work related to their maximum oxygen uptake capacity (Golden, 1993:444). Golden believes that this impairs work ability over long periods, for example in the ability to labour for long hours in fields. This impairment not only reduces labour output from those affected, but may also be reflected in reduced rewards gained for such work. "The productivity of land workers is less in short, lightweight workers with a lower lean body mass" (Golden, 1993:444). This has obvious repercussions on a population-wide scale, as well as on the individual level, and would also have been a significant factor in the working abilities of Medieval populations.

#### 4.4.iv.i *Human health: the geographical and seasonal perspective*

*Spina bifida* and cleft defects are both associated with seasonal variation in incidence today (see Chapter 2). Among soft-tissue anomalies, congenital heart disease is, in some forms, seasonally variable. Ventricular septal defect (hole-in-the-heart) has been shown to have a strong seasonal peak in the spring, and does not always bear any relation to birthweight, maternal age, sex or birth order (Rothman & Fyler, 1976:31ff). Pulmonary atresia (absence or narrowing of the pulmonary artery from the heart to the lungs) and tricuspid atresia (narrowing of the valve between the right atrium and ventricle) and other associated anomalies are often linked to VSD, and seem to have some link with population density (Rothman & Fyler, 1976:33).

The frequency of congenital defects as a cause of death varies significantly according to geographical location and socioeconomic status. Among the general population in the developed world the rate of congenital malformations in newborns is approximately 2-3%, but in developing countries congenital malformations vary from 7.4% to 14.9% as the cause of death (Mattos *et al.*, 1987:305). This may of course reflect social and religious attitudes to deformity, medical and surgical expertise and availability and the willingness of parents to allow non-perfect children to die for economic reasons. It could be concluded that geographical and seasonal influences are secondary to cultural and economic factors.

#### 4.4.iv.j *Human health: the migration perspective*

*"The movement from country to town...is motivated by the possibility, held out by the town, of considerable or even dazzling advancement; an opportunity held out to all, though in fact it is only a few of those who move will achieve it."* (Hicks, 1969:134-5).

*"The study of historic period human remains produces important new information about the history of the people of the local areas.....such research should allow an overview of the nature of human adaptation in the historic period and assessment of the biological correlates of immigration and population displacement."* [Emphasis mine]. (Ubelaker, 1995:47).

"Transitional" populations may be groups undertaking physical removal to another location, or groups who are subject to changing circumstances in their original places of residence, such as changes in the population itself, the original environment, or in the nature of the pathogens affecting it (Swedlund & Armelagos, 1990:5). Rural-to-urban movement tends to occur when people view urban centres as an attractive focus of economic improvement, for themselves and their families (Bogin, 1988), and leads to congested living conditions in a new socio-economic system as cultural changes of family-size, occupation, and housing conditions and availability take place (Coleman, 1995:117). Today, this results in changing standards of health in the developing world, often as a result of the intervention of developed countries through the introduction of industrial, agricultural and social/cultural institutions that have an effect on human health (Roberts, 1995; Swedlund & Armelagos, 1990:6). It seems reasonable to propose that this "modern" transition reflects, in parts, the urbanisation process seen in Europe during the Medieval period, particularly as towns relied on rural influx to maintain population levels. The ingredients of epidemiological transition were essentially the same then as those observed today in Africa, the Indian sub-continent and other parts of Asia, and South America today. It is therefore reasonable to say that the process of historical urbanization

constituted a similar epidemiological transition to that seen today.

Acculturation, or the assimilation of “outsiders” into a larger population, results in the incoming group adapting to the health patterns of the receiving population. Changes in cancer rates have been well documented to illustrate this point (Willett, 1990:9). The exceptions to this rule appear to be groups who remove to a new environment but maintain some separate cultural practice, such as diet, or cosmetic use, that help to preserve their original health status.

Health and epidemiology issues have been studied in relation to migration since the pioneering work of Boas in the early twentieth century (Boas, 1911, 1912, cited by Little & Leslie, 1993). Much of this type of study has been “large-scale”, concentrating on issues such as the transmission of infectious diseases between continents, for instance that of small-pox and measles to Native Americans at the time of the European invasion, and the question of pre-Colombian syphilis (Crosby 1972; Larsen & Milner, 1994). Little work has been done on a smaller scale. Variables such as quality of diet, hygiene, housing and sanitation are known to have an effect on human health, and are strongly associated with social status. By leaving familiar environments to which they are immunologically adapted, migrants expose themselves to new pathogens and increased risk of disease (Little & Leslie 1993:74).

Rural-born people who moved into towns to work would be exposed to new and varied pathogens in greater concentrations than ever before. In addition, it would be foolish to suggest that rural and urban populations did not have

significant and frequent contact apart from that experienced by rural-to-urban migrants. Common sense and historical sources tell us that there was a great deal of everyday and seasonal movement of individuals into and out of towns. For instance, regular markets, horse fairs, and religious festivals brought large numbers of rural inhabitants into towns on a regular basis. Obviously, at such times, individuals would have been exposed to pathogens and pollutants in the town that were unfamiliar to them. However, it is suggested that the short-term nature of such exposure, followed by the return to the familiar environment of "home" would have mitigated the effects of such agents on the people. It is the hypothesis of this study that it is the *long-term* changes in environment, particularly in diet and domestic environment, that affected migrants and their reproductive health.

#### **4.4.iv.k** *Human health - the ethnic perspective*

The study of modern minority ethnic groups in developed countries offers great potential in the field of birth defect research to increase our understanding of the aetiology of all types of defects, whether genetic, environmental or multifactorial in origin. Ethnic groups living in broadly the same environments offer a great opportunity to assess causative factors of defects, for instance between the Asian and white populations of Bradford, and within the Asian community, between the Indian and Pakistani populations. Young (1987) rightly pointed out that the study of individual malformations in different communities gives clues to the aetiology of those malformations, and that the overall population incidence of malformations

offers insights into the effects of obstetric care, social circumstances, consanguinity, and other cultural factors.

Young (1987:109) stated that 'any approach that may shed light on an area of such shameful medical ignorance [regarding congenital defects] is to be welcomed' and suggested that the study of ethnic differences is one such approach. Young did, however, refer to some malformations, such as polydactyly, as "relatively trivial", but failed to consider that affected individuals may well be stigmatized by such deformities and suffer prejudice as a result (Roberts, 2000). Young's definition of major malformations included those that had an adverse effect on the physical well being of individuals. Cultural and/or religious prejudices could well have this effect on an individual, thus rendering a "trivial" defect a serious defect for that person, even though it does not directly threaten their life. When superstition results in aggression towards affected individuals, then the defect could be said to be life-threatening. It must surely be important if the psychological well being of the individual is affected - this could be as bad for a polydactylous person as one with an oral cleft.

Young believed that ethnic studies could be compromised by cultural or religious attitudes to medical care. For example, in quantifying ethnic defect rates, the ascertainment of absolute data must involve the recording of still births, abortions and births in places *outside* hospital in order to obtain a true picture of the rates of defects within each group. Late presenting malformations must also be incorporated into the figures, which may be problematic.

Confounding variables may also be numerous, for example differences in antenatal care, diet, maternal age and parity, social class and occupation all have an effect on the groups and on individuals. A Leicestershire study (Clarke & Clayton, 1983, cited in Young, 1987) found that Asian (*sic*) mothers whose GPs were not on the obstetric list had a higher risk of congenital malformations than non-Asian mothers whose GPs were not on the list. This suggests that there are some cultural factors at work perhaps preventing access to the doctors' surgeries, but the study did not distinguish between the different Asian cultures. If such practices are found to have an association with raised malformation rates, then difficulties may arise with educating the community about preventive measures.

Genetically-mediated anomalies such as hydrocephalus syndrome are well documented and often associated with a founder effect, or clearly associated with genetically isolated populations (Young, 1987:109). Environmentally-mediated anomalies are often geographically and therefore culturally distinct, for example, Minamata disease (methylmercury poisoning) has been documented in Japan, where pregnant mothers ingested poisoned fish, and in Iraq, where mothers ate fungicide-spayed grain. Both incidents resulted in the birth of brain damaged infants. Multifactorial inheritance has been described as "a polygenic disposition plus an environmental insult" (Young, 1987:110). Neural tube defects and cleft lip fall into this category, both groups having been shown to have strong cultural and/or geographical links. Cleft lip often persists in ethnic groups even after migration, suggesting a strong genetic element (Mongoloid rates are high, white rates are intermediate, Negroid

rates are low). On the other hand, neural tube defects appear to be sensitive to environment, as there are differences within and between ethnic groups, suggesting a stronger environmental element.

The problem of consanguinity is on-going today among some cultures and associated with much anecdotal evidence, which carries no scientific authority. The practice is common in the Indian subcontinent, particularly among Muslims, and hence the rates are high in Pakistani communities. First cousin and uncle/niece marriages are common. It is suggested that such groups would eventually “breed out” their weaknesses, but modern healthcare and treatment of defects must surely allow affected individuals to live longer and pass on their own genetic inheritance to their own offspring. In addition, anecdotal evidence suggests that marriages are still arranged for affected individuals, thus ensuring the continuity of the family pattern of defects.

Young believes that studies of ethnic differences in malformations would be valuable (1987:110). Problems have already been noted in relation to the practice of consanguinity in Bradford and Birmingham, with increased perinatal mortality associated with the practice (pers.comm. K.Manchester).

Terry *et al.* (1983) reported on a Birmingham survey conducted over three years involving over eleven thousand mothers. The incidence of congenital abnormalities in births was related to the ethnic origin of the mothers, in an attempt to quantify the effects of cultural, communication, health education and late-booking factors on the outcomes of pregnancies. The results showed that there were significant differences between ethnic groups monitored by the



study, with the Pakistani and Indian mothers producing babies with different types and rates of congenital malformations. Consanguinity and increased maternal age had been thought to significantly influence the occurrence of particular defects, but the study showed that the Indian group, with the lowest rates of consanguinous marriages and increased maternal age, in fact produced the highest rate of affected babies. Malformations of the alimentary system were commonest in the group, and were tentatively attributed to some environmental factor such as diet, or even a genetic predisposition. The Pakistani mothers, with the highest rates of consanguinity and increased maternal age (14% were 35 years old or older) showed a slightly lower rate of malformations, of which the majority were multiple and/or chromosomal in origin. These defects were attributed to consanguinity and maternal age. The authors rightly concluded that the blanket term "Asian" was inappropriate when applied to mothers from groups originating from the Indian subcontinent, as there were clearly demonstrated differences between the Indian and Pakistani groups. Environment was considered to be a possible causative factor for this phenomenon, along with possible genetic predisposition.

Knox and Lancashire's survey (1991) covered twenty-one years between 1964-1984 and included births among the Caucasian, Afro-Caribbean and Asian communities. Out of 352, 867 live and still-births recorded, a total of 9,965 presented with congenital malformations, making a rate of 28.24/1000. Affected individuals presented over eleven thousand malformations.

Unfortunately for this author's research, Knox and Lancashire excluded those defects 'deemed entirely secondary to another major defect, such as *talipes* in

the presence of *spina bifida*' (1991: 41). However, the results may therefore be deemed to be under-reporting the frequency of many skeletal malformations. This is most likely the general practice in the medical literature as a skeletal deformity is likely to be considered of less importance to doctors and patients than a soft-tissue problem such as congenital heart disease.

The overall defect rates were similar in the Caucasian and Afro-Caribbean communities, being 28.0/1000 and 28.4/1000 respectively. The Asian community showed overall rates at 32.6/1000. The authors did not distinguish between Asians of Indian, Pakistani or Bangladeshi origin, but consanguinity is likely to have played a part in these rates. Afro-Caribbeans had a low prevalence of neural tube defects, at 0.9/1000. Caucasian rates for neural tube defects were 3.46/1000 and Asian rates 3.09/10000 (1991:41-43).

Oral clefts were least frequent amongst Afro-Caribbeans at 0.74/1000, with Caucasians at 1.68/1000 and Asians at 2.00/1000. Congenital heart disease rates were highest in the Asian community at 4.48/1000; 3.86 among Caucasians and 3.56 among Afro-Caribbeans. Polydactyly was highest amongst Afro-Caribbeans at 9.55/1000, with Asians at 2.13/1000 and Caucasians at 1.11/1000.

Peach *et al* (1998) found that ethnic origin was not an over-significant factor when they analysed age-standardised and proportional mortality figures between aborigines and non-aborigines in various areas of Australia. Non-aboriginal groups showed statistically significant rises in circulatory,

respiratory, digestive and traumatic diseases in rural areas compared to metropolitan areas. Aboriginal groups showed similar significant rises in the same classes of disease, with the additions of endocrine diseases and mental illness. Interestingly, the latter was found in males only, which may be related to psychological stresses as described by Ursin (1994) and others. The authors concluded that both groups' increased mortality rates in rural areas were due to all-cause standardised mortality ratios, and that 'something about living in remote areas must cause most diseases and/or induce death regardless of cause at an earlier age' (1998:40). They do not appear to have considered the difficulties in obtaining medical attention in remote areas, however. Nevertheless, these results may suggest that, over time, ethnic differences between groups living in the same environmental conditions seem to disappear. This echoes, to some extent, the work of Willett (1990) and others regarding migrant population health trends in which it is generally noted that incoming groups adapt to the host health patterns.

#### **4.4.iv.1** *Human health: the psychoimmunological perspective*

The science of psychoimmunology has developed significantly over the past two decades and offers a perspective on human health that combines issues of immunology, psychology and endocrinology (Udelman & Udelman, 1983; Kiernan, 1987; Pansky *et al.*, 1988; Egger, J, 1992; Haas & Schauenstein, 1997; Hiramoto *et al.*, 1999). Issues of psychological stress and individual perceptions of stress have been monitored and investigated with regard to their immunological effects on affected individuals. Immune suppression has been correlated with psychological stress. Clearly, the results showing an adverse

immunological reaction to stress at the individual level could be also applied at the community level. This has obvious implications for migrants and their host communities. It is an accepted fact that disease reproduction rates are often higher in urban rather than in rural settings and it may well be that not only is population density a factor in increased disease rates, but also the relative psychological stressors acting on populations in varying environments. Mausch points out that:

“health or disease depend on complex, psychophysiological mechanisms. Factors very important for health are: genetic factor, personality, stress and coping, economic and social situation, work, family, etc. The immunity unites with personality, thinking, attitudes, emotions, whose kind, strength, and adjustment depend on the sense of coherence.” (Mausch, 1999).

This aspect of human health is likely to become a major area of research in the future, and has obvious implications for the study of health in the past. For example, the difference between small-scale, family-orientated village life such as that at Wharram Percy, compared with the work-orientated and relatively anonymous lifestyle of young, single economic migrants in York, often living without family in tenements, must have had a significant effect on the psychological status of those individuals. According to psychoimmunologists, this would also inflict consequences on their immunological status, and combined with the co-existence of population density-mediated health factors, must have been an added burden on their immune systems.

#### **4.4.v Summary of the epidemiological and biocultural approaches**

- epidemiological studies include description and analysis of patterns of disease, measuring various attributes and variables; the biocultural

approach also does this by investigating archaeologically-derived population samples in the same way

- both types of study may investigate and compare groups that are exposed/not exposed to a certain factor, or groups that are diseased with those that are not diseased; this study equates to the former category
- studies of modern populations can calculate incidence, whereas studies of archaeologically-derived samples can only calculate prevalence of a disease
- epidemiologists work with a sample of the modern population that is often neither ideal nor totally representative; biological anthropologists have the same limitations with archaeologically-derived samples
- comparable attributes and variables may be observed by epidemiologists and biological anthropologists
- health issues that are known to affect modern populations may be considered to have equally affected past populations, especially environmental exposures that are known to cause birth defects
- relatively high rates of population density in urbanised areas are known to be associated with raised rates of infection and other disease processes associated with hazards of overcrowding and changes in cultural behaviour and surroundings such as removal from family networks
- archaeologically-derived samples may only be evaluated in terms of generalized exposures unless documentary evidence suggests otherwise; hence comparison of urban and rural samples is a viable method of investigation

- archaeological evidence can provide evidence of generalized exposures of past populations by defining environmental evidence of foods, housing conditions, water supply, craft and industrial processes and so on, that may be related to the health status of associated populations by reference to epidemiological studies
- the study of archaeologically-derived samples may provide health-related data that could be used to assist in the health and safety and medical provision for modern populations.

## CHAPTER FIVE

### Materials and Methods

#### 5.1 THE MATERIALS

The skeletal material from four excavated English Medieval sites (one Early Medieval, three Late Medieval) is being examined (see fig. 5.1). The Early Medieval site at Raunds, Northamptonshire, is of the Anglo-Saxon period, and represents a small agricultural settlement (as yet unidentified archaeologically); the other four sites are from the Later Medieval period and represent populations from three cities and a further agricultural rural settlement. Wharram Percy, the rural site, and St. Helen-on-the-Walls, a poor urban parish in York, are both found in the same geographical area of Northern England, and thus comprise two environmentally-influenced groups from the same regional population; the Chichester site also offers a view of an urban population as represented by its poorest members (who are arguably the most likely to be affected by disease), being drawn from the burial ground of a hospital and almshouse; and the Hull sample is derived from the burial ground of a monastic foundation which flourished in the town, and is thought to contain the remains of benefactors, brothers and possibly the sick from the monastic infirmary

**5.1.i The Anglo-Saxon cemetery of Raunds**, Northamptonshire, dates from the mid-tenth to the late twelfth centuries, covering the period between the granting of burial rights to the church until its conversion to secular use, when the cemetery appears to have been abandoned and forgotten (Boddington, 1996). The site itself, however, was occupied from the sixth to the fifteenth centuries, and is possibly that of the original Saxon manor referred to as "Burgred" in the Domesday Book

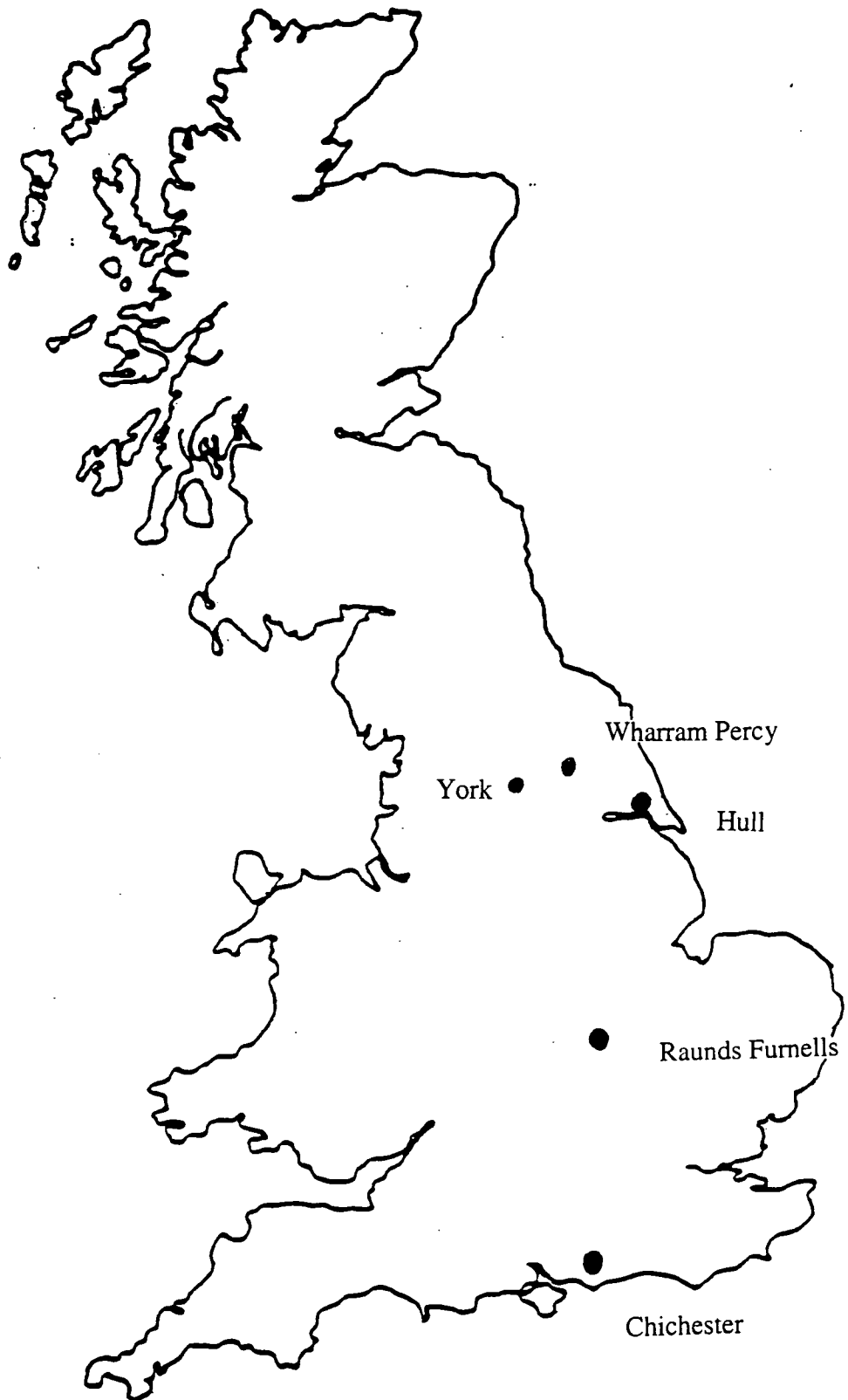


Figure 5.1: Map of Great Britain showing location of sites in the study



(Boddington, 1996). A total of 363 individuals were available for study from the churchyard cemetery, derived from an estimated living population averaging about forty people at any one time during the period of occupation. This sample, being from the Early Medieval period, and from a pre-urban population, may be considered as a notional "baseline" against which to assess the other three samples in the study, as the Raunds inhabitants lived in an essentially pre-urban environment and arguably would not have been exposed to the same degrees of population density-related hazards as rural dwellers in the later period, who had contact with towns and cities.

The grant of burial rights to the church at Raunds Furnells would have increased its importance, and enabled the burial of local people in a local churchyard, as previously the residents of the area would have had to seek burial further afield in a church with burial rights. The church itself underwent redevelopment during the two hundred years of its use, rising from the rank of "field church" to that of "small church with cemetery" and later being rebuilt in the late eleventh to mid-twelfth centuries (Boddington, 1996). The churchyard initially consisted of a ditch-outlined area measuring about 40 metres by 30 metres, and infilling by burials progressed along the enclosure in stages. Zone 1 was nearest to the church, with burials commencing only 2.5 metres from the walls. Zones 2 and 3, on the west and east sides, accommodated overspill burials, and Zones 4 and 5 made up the burial areas in the north-east and south-east corners (see fig. 5.2). During the late expansions, the space adjacent to the walls was brought into use, firstly with adult burials, within 1 metre of the walls, and then for infant burials, right up to the walls. The demographic structure of the population of the cemetery is typical of a pre-industrial group, suggesting that it represents a wide cross-section of the actual population (Boddington, 1996). Variation

in burial rites also suggests a cross-section of the social spectrum, from the so-called “founder’s plot” under an ornate stone slab to the simple shrouded burials without coffins but with stones for protection.

The conclusion of the Raunds Furnells report (Boddington, 1996:68) stated that the author believed there could have been another church serving the area at the same time, as the population numbers at Raunds Furnells are too great for the manorial household, but too few for a whole village settlement of the period. However, whatever the arrangements for church-provision and burial, the population was an agriculturally-based one, living in a pre-urban, pre-industrial period. As an associated village settlement has not been identified archaeologically (the population is too large to represent a manor house), it is not possible to recreate the specific environment in which these people lived, but it is possible by searching the relevant historical literature, and by considering other Anglo-Saxon sites and populations, to generalize about their lifestyle (e.g. Hagen, 1999; Malim & Hines, 1998).

**5.1.ii The cemetery of the leprosy hospital and almshouse of St. James and St. Mary Magdalene, Chichester, West Sussex,** is believed to have been in use from the hospital’s founding in the early twelfth century, through the period of use as a leprosy hospital up to the early sixteenth century, and throughout the subsequent almshouse period until the apparent closure of the institution in the early eighteenth century (Lee & Magilton, 1989). The Medieval city of Chichester was important as a market centre, port and religious focal point. Dell Quay was situated two miles to the south-west, and by 1341 AD had become a major port, guaranteeing substantial and regular contacts between Chichester and overseas ports, populations and diseases.

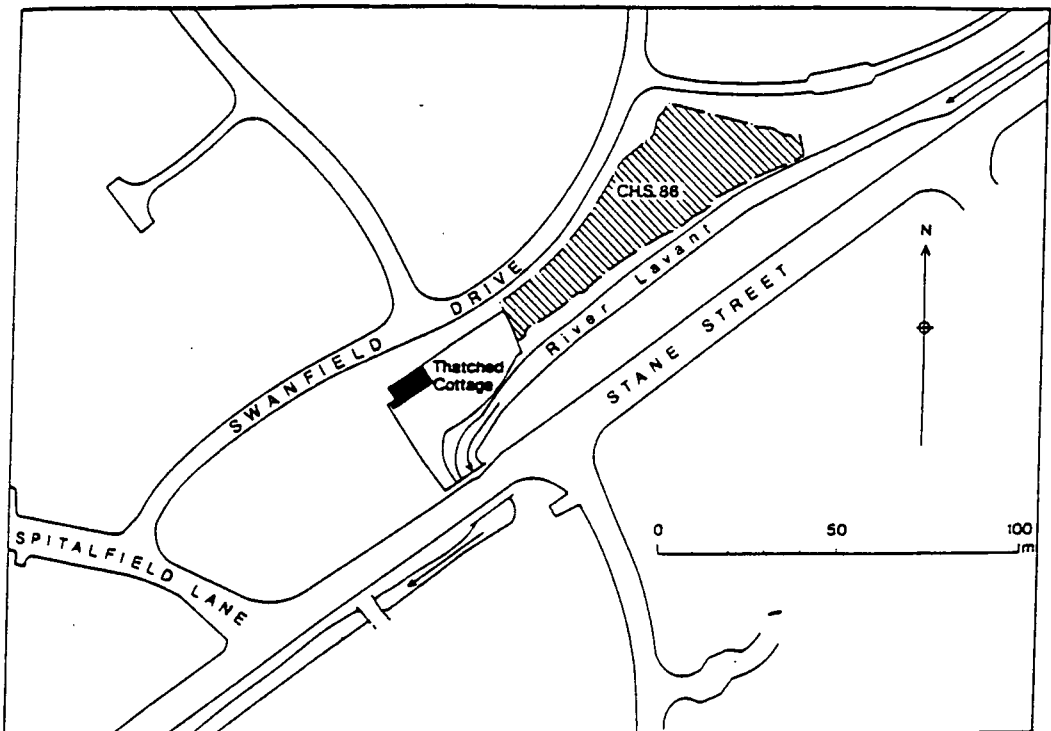
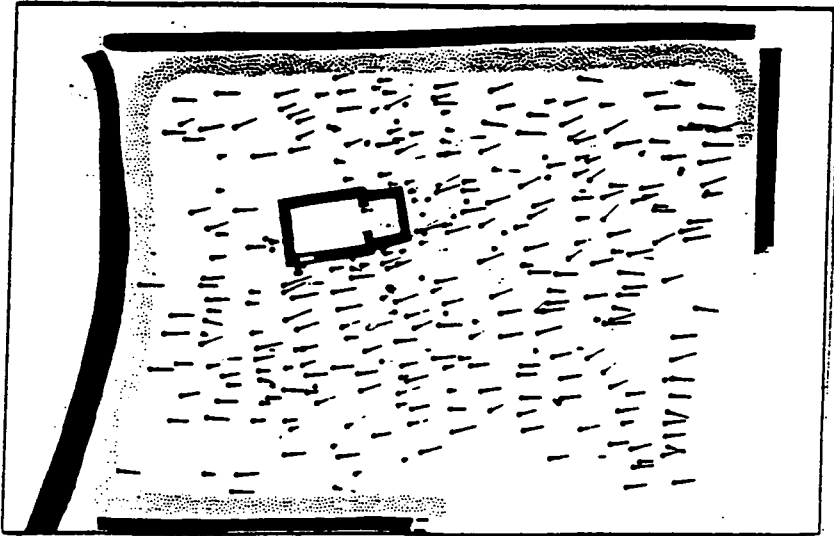


Figure 5.2: The cemetery at Raunds Furnells

Figure 5.3: The cemetery at St James and St Mary Magdalene, Chichester

The hospital of St James and St Mary Magdalene was one of five in Chichester during the Medieval period, and, like most leprosy houses, was situated outside the walls, in this case, on the north-east side of the city (see fig.5.3). Documentation suggests that the hospital was founded before 1118 AD and had become an almshouse for the sick and poor during the sixteenth century (Lee & Magilton, 1986, 1989). The cemetery was laid out in three zones, with the earliest, that of the leprosy hospital period, being to the west of the site close to the hospital walls (see fig. 5.2). Many graves were intercut, suggesting pressure of space during this period, and most of those buried in this zone are male. The central area was very disorganised and contained the burials of males, females and juveniles. The eastern zone was well-laid out with the graves in rows aligned east-west, and contains burials associated with the almshouse period, which exhibit a wider range of pathology (Lee & Magilton, 1986, 1989). Burials were in coffins with the nails surviving, but there was no evidence of grave markers. Two stone-lined tombs were found in the western area, and a charnel pit was partly excavated in the north-eastern area, dated between the earliest and latest phases of burial (Lee & Magilton, 1989).

The population of the cemetery is taken to represent the sick and poor of the city during the almshouse period, and leprosy sufferers from the earlier period. However, documents of 1589 refer to an inmate who had a house in the suburbs of the city, was married, and had two servants. This may be an illustration of the practice of taking in residents for money, which was apparently not uncommon from the fourteenth century onwards (Clay, 1966:39). The population of the cemetery may therefore represent a selective group of "sick and poor", rather than an unbiased group of those most in

need of the charity of the hospital. Nevertheless, to have been admitted to the leprosy hospital or the almshouse, an inmate would have had to be a resident of Chichester, therefore the population can be taken as representative of an urban population.

Unfortunately we do not know what exactly were the requirements for burial in the cemetery; for example, it is not known whether all inmates were buried there, or if others associated with the hospital were also buried there (Lee & Magilton, 1989).

Being a hospital cemetery, the sample is, of course, biased. Some of the apparently non-leprosy individuals in the early phase may have been sufferers, but died without the disease leaving any marks on them. The later burials of the almshouse period would presumably have been drawn from the poor (but probably selective) sick of the city who needed support and were admitted to the hospital on that basis.

A total of 349 individuals was excavated from the cemetery in 1986 and all were available for study. The earliest phase burials were of males only, as only male inmates were admitted to the hospital. From 1540 onwards, however, women and children were also admitted and allowed burial in the cemetery. A total of eighty three individuals exhibit skeletal changes associated with leprosy, although this does not necessarily indicate that this was the total number of leprosy sufferers buried in the cemetery. A further sixty individuals were excavated in 1993 and these have been included in the study.

**5.1.iii The cemetery of St. Helen-on-the-Walls, Aldwark, York,** was in use as a parish church graveyard from the twelfth to the sixteenth centuries (Dawes & Magilton, 1980). The church fell into disuse by 1549 and was demolished, and the

graveyard appears to have been forgotten, with later development on the site including that of the Victorian Ebor Brewery, whose demolition was the cause of its rediscovery. The cemetery and church contained the remains of at least 900+ individuals, which were all available for this study.

St Helen-on-the-Walls was, according to documentation of 1535, one of the poorest parishes in York (Magilton, 1980). This accords with the relatively high levels of stress indicators on bones and teeth from the site observed by other workers (Lewis *et al.*, 1995). The area had not always been very poor, however, as the tenements excavated in the 1980s had originally been built as a redevelopment scheme during the 1330s with relatively substantial rent rates (see fig. 5.4). Unfortunately, the arrival of the Black Death in the following decade caused severe economic recession in York, and that section of the street degenerated into a poor low-rent area as tenants became scarce. As well as being a poor area with crowded tenements, the street also housed a foundry (Richards, 1993), and the rich property known as The Bedern, which was the residence of the Vicars Choral of York Minster. At the south end of the street there were other rich buildings, including the Taylors' Guild and associated hospital (Magilton, 1980). Other recorded crafts and occupations sited in Aldwark and the neighbouring parish of Bedern included dyers, chandlers, brewers, tanners, masons, carpenters, apothecaries, and metalworkers (Grauer, 1989, cited in Lewis *et al.*, 1995). The street is thought to take its name from the "Old Werk" or ditch, which dates from the Scandinavian period of York, and which ran alongside the line of the Medieval street, being a focus for dirty water, refuse and associated pollution. As a result, the area tended largely to be inhabited by the poorer end of York society, particularly by single people working in the city's industries, who rented the poorer-quality

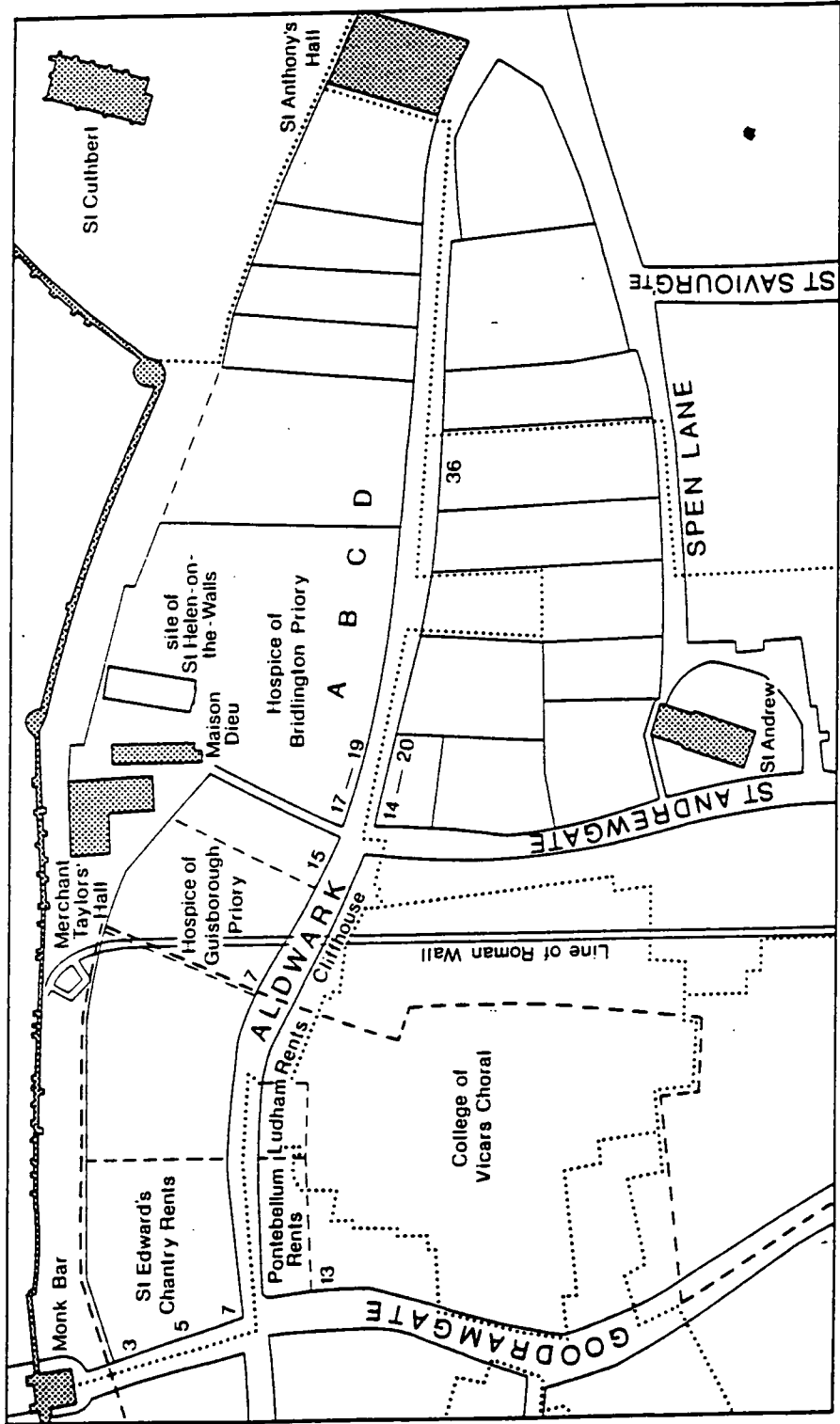


Figure 5.4: The cemetery location of St Helen-on-the-Walls, York

accommodation in the area (Dawes & Magilton, 1980; Goldberg, 1992). Full- and part-time prostitutes were also recorded as living on Aldwark in the fifteenth century (Hall *et al.*, 1988).

**5.1.iv The cemetery of the parish church of St. Martin, Wharram Percy,** North Yorkshire, dates from the thirteenth to the nineteenth centuries, with Medieval burials ending in the sixteenth century (Beresford & Hurst, 1990). Its excavation yielded the remains of approximately 600+ individuals, but only the Medieval burials (approx. 550) were considered for the current study. The Medieval village of Wharram Percy was occupied continuously between the twelfth and the fifteenth centuries, although there were previous settlements on the site dating from the Romano-British and Anglo-Saxon periods. The village was a typical agricultural settlement of the Medieval and post-Medieval periods, located on the chalklands of the Yorkshire Wolds, and was abandoned over a long period, although the church continued in use for three other nearby villages until the mid-twentieth century (see fig. 5.5). The village consisted of two Medieval manors (Beresford, 1979) and their associated peasant houses and tofts (gardens around the houses which could be used for vegetable/cereal growing). It is believed that there would have been about thirty families in the village during the twelfth century, and that this number increased during the following two hundred years (Hurst *et al.*, 1979). Timber-framed wattle and daub, and later, stone, longhouses have been excavated, varying in length between forty and eighty feet, with central doorways and packed earth floors of chalk (Hurst *et al.*, 1979). Hearths appear to have been central, and were probably vented through thatched roofs (Milne, 1979c). The excavators believed that the concave nature of the floors, indicating regular sweeping, would have kept debris harbouring parasites to a



Figure 5.5: Situation of Wharram Percy in the Yorkshire Wolds

minimum (Hurts *et al.*, 1979) but the very nature of longhouses implies the keeping of animals at one end for at least part of the year, so this assumption may not be quite accurate. The winter build-up of manure and animal bedding would probably have been a health-mediating issue for at least part of every year. Water supplies were believed to have been taken from the local stream (Mays, pers.comm.) which may, of course, have been subject to pollution by micro-organisms detrimental to health, but not to the same large-scale pollution as seen in York in the “Old Werk” and open sewers. Environmental evidence from Wharram Percy is outlined in Chapter 2.

**5.1.v The cemetery of the Augustinian friary, Kingston-upon-Hull,** was excavated in 1994 and yielded the remains of c. 300+ individuals. The friary is the best documented of the several religious foundations in Hull and functioned as a monastery from the second decade of the fourteenth century until its dissolution in 1539 (Evans, 1994). The house is known to have helped the sick and needy and it is possible that some of those in the cemetery belonged to this category. The remaining burials may, as well as members of the monastic community, have included wealthy donors or others associated with the foundation during its period of use. Males, females and juveniles are represented in the cemetery. Boylston *et al* (forthcoming) will provide new information on the site and its likely inhabitants.

## **5.2 METHODOLOGY**

### **5.2.i Introduction to methodological approach**

This study focuses on the prevalence of certain birth defects of the axial skeleton, both within and between the sample populations under observation. The defects observed are: border shifting (e.g.lumbarisation/sacralisation), block or fused vertebrae,

cervical/lumbar ribs, numerical variations in vertebrae, spondylolysis and spondylolisthesis, *spina bifida occulta*, cleft neural arch, and oro-facial defects including cleft defects of the maxilla. Stress indicators (*cribra orbitalia*, enamel hypoplasias and tibial periostitis) will also be observed in adults (Goodman *et al.*, 1988), as they are the actively reproducing component of the populations observed, and adult stature is being recorded as an indicator of population-wide general health (Gray & Wolfe, 1980; Trotter, 1970).

The prevalence of birth defects of the axial skeleton will be compared between the sites and statistical analysis carried out to arrive at meaningful conclusions. The observed defects are diagnosed in accordance with current clinical understanding and classification, which enables the suggestion of some implications for the affected individual's lifestyle. In the medical literature a certain number of soft-tissue defects are associated with skeletal anomalies (Grieve, 1981) and the author hopes to give an insight into some conditions that would otherwise remain invisible to the palaeopathologist by linking skeletal defects with modern frequencies of specific soft-tissue involvement. For example, some neural conditions relating to fused vertebrae have already been noted (Pizzutillo, 1983; Smith & Micheli, 1995) and the supposed irrelevance of transitional vertebrae to health is being reconsidered in the light of research with healthcare professionals.

Alongside the medical review of the skeletal material, general historical sources (Boissonade, 1964; Chambers, 1972; Hodgett, 1972; Postan, 1972, 1973; Razi, 1980; Reynolds, 1977, 1994; Holt & Rosser, 1990; Schofield & Vince, 1994), and in the case of York, specific sources (Dawes & Magilton, 1980; Swanson, 1983; Goldberg, 1992)

are being compared with the archaeological evidence available about the sites from which the skeletal material originates. The considerable historical Medieval literature, considered alongside specific documentation for each site, gives much detail about lifestyle during the period to which the skeletal material belongs. This provides a wide-ranging representation of the conditions under which people were living in both town and country during the period of use of the cemeteries (for instance, agricultural lifestyle, industrial conditions, urbanization, domestic conditions, water supply, diet, population size and density and so on).

Research is also being undertaken into the field of embryology, which indicates possible sources of insults to foetal development (teratology) resulting in birth defects. Factors such as folate deficiency in the mother are already documented (Smithells *et al.*, 1981; Smithells 1983, 1992) as having a damaging effect on the foetus; others become apparent during the review of the archaeological, historical, biological and medical evidence. This is the strength of the biocultural approach - all types of evidence are considered before making a final interpretation of the biological material.

### **5.2.ii Research philosophy**

By assessing the biological evidence of a genetically homogenous population (in this case, the Medieval English) over time and space, it is possible to enhance our understanding of human adaptability (Ubelaker, 1995) by observing the biological changes associated with particular environments. Archaeological populations offer a ready-made pool of data from which to gather such information. This study employs the biocultural approach (see below), and also incorporates an epidemiologically-focused methodology which draws on the study of disease related to environment (see

fig.5.5). In this way, the analysis of archaeologically-derived populations may be assessed in a similar manner to the free-living populations under epidemiological study today (Barker & Hall, 1991).

### 5.2.ii.a *The biocultural approach*

This theoretical framework allows the consideration of *biological* evidence in its *cultural* setting (Roberts and Manchester, 1995). It involves assessment of the past environment (including all identifiable cultural factors such as diet, housing, etc.) using contemporary archaeological and/or documentary evidence, considering all the relevant factors in the interpretation of the biological evidence. The study of palaeopathology today is founded upon a clinical background, with biological anthropologists making diagnoses based on current clinical knowledge (Auderheide & Rodriguez-Martin, 1998; Larsen, 1997; Ortner & Putschar, 1981; Roberts & Manchester, 1995). This study uses the same approach, diagnosing congenital defects of the skeleton using recognised diagnostic criteria which may be easily referenced by other researchers in the clinical literature (e.g. Barnes, 1994; Resnick & Niwayama, 1988). It is also helpful to utilize current epidemiological knowledge as a means of understanding firstly, what the skeletal evidence tells us and, secondly, what the archaeological evidence of the associated environment should lead us to expect in terms of human health patterns and changes. For example, socio-economic status and thus living conditions are closely related to health (Davey-Smith *et al.*, 1997) and constitute an overall environment which impact upon reproductive health over the long term (Skjaerven *et al.*, 1997). Poor quality living conditions have been shown to lead to rising infection rates in modern urban settings (Jackson *et al.*, 1989), and the same living conditions, with poor quality lighting, ventilation and sanitation may

apply equally to rural housing. In the urban environment however, they are almost always associated with overcrowding, and this appears to be a critical factor leading to major health problems in modern urban populations (Jackson *et al.*, 1989), especially in the developing world. The populations under observation in this study experienced environments that differed in terms of housing, sanitation, nutritional quality, risk of infection and over-crowding. For example, housing in the parish of St. Helen-on-the-Walls, York, consisted largely of brick and timber tenements on narrow ill-drained streets, some in close association with a foundry, and the street of Aldwark was itself named after the "Old Werk" or ditch which ran parallel to it which was recorded as being filled with rubbish and waste (Hall *et al.*, 1988). Housing at Wharram Percy on the other hand consisted of widely-spaced longhouses of wattle and daub construction with thatched roofs, set in individual crofts, all sited in a valley among fields. It is suggested by the author that these, and the other factors associated with over-crowding and living conditions (mentioned elsewhere in this chapter), produce different rates of birth defects which may be observed in these environmentally-identified populations. Applying this epidemiologically-derived knowledge to biological evidence is essentially a further development of the biocultural approach in action.

#### **5.2.ii.b** *The epidemiological approach*

This study makes use of environmental epidemiological techniques as a means to understand the health of observed past populations. Within this framework, certain human variables and attributes are important in epidemiological research, and may be reasonably applied to the study of past populations by archaeological means. These attributes were defined by Barker and Hall (1991, 55ff) to include *host* determinants such as age, sex, ethnic group, marital status, family group, occupation and lifestyle.

They also defined *agent* determinants such as pathogenicity, dosage and infectivity, *environmental* determinants such as climate, vector ecology, residential conditions and availability of food, patterns of *population structure* which allow the assessment of age- and sex-distribution of cases, and *levels of ascertainment* and *diagnostic criteria* which allow the quantification of true levels of disease rather than simply a picture of those who had access to health care.

Although Barker and Hall's work focuses on modern epidemiology, their framework may also be applied to work focussing on the health of past populations. It is potentially possible, through archaeological and anthropological enterprise, to ascertain past variables and attributes such as age, climate, vector ecology, living and working conditions and availability of food, with some degree of certainty, although this is dependant on the site itself, and the quantity and quality of evidence available (e.g. Ziegler, 1969; Postan, 1972; Andrews *et al.*, 1979; Reynolds, 1994; Bond & O'Connor, 1999). Attempts to quantify other factors such as pathogenicity, dosage and infectivity may also be made, although with less certainty (Kiple, 1997).

Assessment of occupation from skeletal remains, for example, is a subject on which there is little agreement, although some advances are being made (Kennedy, 1998). Of course, archaeological population samples are inherently biased also (see below), but so are any populations involved in epidemiological research.

The anthropological approach to disease is another perspective that is closely related to epidemiology. Two models are commonly used in the anthropological approach to disease study, the *ethnomedical* model and the *ecological* model (Dennet & Connell, 1988; Sargent & Johnson, 1996). The former interprets the *cultural* response to

disease by considering that society's definition of disease. Infanticide of visibly deformed infants may be considered from this perspective, as well as the response of societies attempting to control or contain disease threat (Dennet & Connell, 1988:1-2), for example, in the provision of leprosy hospitals outside Medieval towns. The ecological model considers the *biological* response to the disease process, identifying and assessing the variables involved. It has been criticised for focussing too heavily on the pathogen as the cause, rather than the sociocultural factors that allow its spread (Dennet & Connell, 1988:4). It focuses now on population health rather than individual health, and assesses the roles of all possible insults to the health of studied populations, making it the epidemiological equivalent of the biocultural approach. In addition, the ecological model considers health and disease to be a continuum rather than a "present or absent" position. Clearly, both of these approaches encompass the study of past population health.

### **5.2.ii.c** *Considering migration – the geographical movement of people*

As English urban populations were more or less constantly renewed by the arrival of rural incomers during the Medieval period, a consideration of the effects of migration on human health is appropriate. Of course, rural migrants to towns would be affected on a permanent basis by their exposure to the hazards of urban population density once they had taken up residence in town, rather than to the short-term exposures they had experienced on day visits, for example, to the market. It is this permanent exposure which the author proposes to be the aetiological agency behind any higher prevalence of birth defects in towns. The effects of a changed environment on human biological processes have already been addressed extensively with regard to migration. Ravenstein (1885, 1889) and Boas (1911, 1912) pioneered such studies in



the nineteenth and early twentieth centuries and have been followed by others. Boas' work concentrated particularly on the changing health patterns of long-distance migrants. More recently, Crosby (1972), and Larsen and Milner (1994) have focused on post-conquest biological responses in native populations. As well as considering long-distance movement, Ravenstein (1885, 1889), Stone (1966) and Clark (1972) found that significant migration also took place across short as well as long distances, including both planned betterment (elective migration) and subsistence (forced migration for survival) relocation. This short-distance movement is the likeliest form of migration that occurred between York and Wharram Percy (two of the archaeological sites considered in this study), for example, with younger adults coming into the city for employment in the local industries (Goldberg, 1992; Stone, 1966). It is difficult to identify individual migrants within historical cemeteries in the absence of legible gravemarkers and other appropriate documentary evidence. The cemeteries of Wharram Percy and St. Helen-on-the-Walls are in a relatively close geographical relationship with each other (c. 20 miles), perhaps being directly linked through migration from the village to the city. In practice, the examination of human material in urban parish cemeteries is unlikely to reveal any individuals or groups as "different" in any meaningful way. The constant influx of newcomers into towns would mean that at any one time, a substantial proportion of the population would be first or second generation migrants. Third and subsequent generations descending from any "different" outside groups would probably be indistinguishable from longstanding urban dwellers due to intermarriage with local individuals. If the hypothesis of this study is correct, it may be that those who are "acclimatised" to the urban exposure levels associated with population density and a different lifestyle, may, after two or three generations, become "immune" to its effects. Similar effects

are noted in modern immigrant populations with regard to disease rates (Bogin, 1988; Coleman, 1995), in which incoming populations take on the disease incidence of the host population over time. It may be that the higher proportion of an urban population expressing greater rates of birth defects may actually be concentrated within the relative newcomers, with the more established rural families expressing prevalence rates more akin to the rural levels observed outside towns. Recent work on identifying the geographical area of origin of individuals by analysing isotopes in dental tissue (Barreiro *et al.*, 1997; Budd *et al.*, 1997; Budd *et al.*, 1998; Montgomery *et al.*, 1999; Montgomery *et al.*, 2000) may be applied on a larger scale in the future, but is beyond the scope of this study.

However, plentiful documentary evidence shows that much “economic” migration took place from country to town during the Medieval period (Bogin, 1988; Chambers, 1972; Clark, 1972; Coleman, 1995; Goldberg, 1992; Mays, 1997; Reynolds, 1994; Stone, 1966). Indeed, such migration was necessary for towns to maintain their population levels until at least 1750 (Chambers, 1972). Mays suggested (1997) that the high proportion of females in the St Helen-on-the-Walls cemetery may be due to female-led economic migration to the city, with poorer women immigrants living in and around Aldwark. This is a useful suggestion, and appears to be supported by the correspondingly low male ratio in the cemetery at Wharram Percy.

This study seeks evidence of change in health patterns over long periods of time, however, and does not therefore seek individual migrants. The working hypothesis of the study must be examined over a long time period in order to obtain statistical validity of the overall health of urban and rural populations. Thus, the observed

populations from which the data are drawn each covered periods of not less than two hundred and fifty years (Raunds Furnells: 10<sup>th</sup> - late 12<sup>th</sup> centuries; Chichester: 12<sup>th</sup> to possibly late 17<sup>th</sup> centuries; Wharram Percy Medieval burials: 13<sup>th</sup> -to 16<sup>th</sup> centuries; St. Helen-on-the-Walls, York: 12<sup>th</sup> - 16<sup>th</sup> centuries). In the case of the urban populations this long time period is expected to reduce the effects of individual environmental “outsiders” significantly skewing the data by introducing characteristics of a non-urban environment.

Barker and Hall (1991:77) state, in regard to migration and its role in disease patterns:

*“Two kinds of movement are of epidemiological importance. Firstly people may migrate from one area to another, taking diseases with them, removing themselves from exposure to diseases in the area they leave, or exposing themselves to new diseases in their area of settlement. Secondly, diseases may move through static populations leaving clusters of affected persons in their path.”*

They also say (1991:76) that changes in the methodology of identification, and in attitudes to disease, may also influence the detection of disease within populations under observation, and this may lead to apparent changes in disease frequency which do not in fact exist. This could be said to apply also to palaeopathological research, with improvements in diagnosis once the clinical base to study became established, for example in Barnes’ distinction between cleft neural arch and *spina bifida occulta* with neural tube defect. From a biological perspective, the effects of migration are simply to place populations *within different environments* (Bogin, 1988; Coleman, 1995). This is usually achieved by actual *movement* of populations, but similar effects may also result from a *change in the nature of the environment* in which the population resides (Larsen & Milner, 1994; Little & Leslie, 1993). Such a change in the environment can of course, as Crosby and Larsen and Milner showed, be due to the influx of a new population into an area, bringing with them new environmental

factors in the form of disease agents. It is this introduction of a wider range and concentration of pathogens which is the focus of the hypothesis of this study.

#### **5.2.ii.d** *Nutritional epidemiology*

Particular attention is paid in this study to nutritional epidemiology, which highlights health deficiencies associated with under- and malnutrition (e.g. Willett, 1990).

Studies have shown correlations between nutritional factors, immune status and growth (e.g. Chandra, 1978, 1979, 1980; Frisancho *et al.*, 1980; 1985; Gray *et al.* 1980; Jenkins, 1981).

#### **5.2.ii.e** *Occupational epidemiology*

Occupational epidemiology, which addresses hazards in the workplace such as lifting and heavy manual work, plus the dangers of toxic metals and other chemical agents, is also considered (e.g. Palmer & Coggan, 1997), as is sex-based epidemiology, with particular reference to women's health problems, as women's health is widely held to be directly or indirectly the primary source of birth defects in infants (e.g. Barker, 1994).

#### **5.2.ii.f** *Maternal infection and defect rates*

Recurrent maternal infection over prolonged periods could result in a generalised permanent state of low grade bacterial, viral and parasitic infection in association with malnutrition (Chandra, 1980). This situation would reduce immunocompetence still further and render the mother, and thus the foetus, open to greater pathogenic threats. Whether the pathogens themselves result directly in birth defects, or whether defects result from generalised maternal infection/malnutrition leading to inadequate nutrition

of the embryo during organogenesis is not known. However, it is suggested that as well as the more commonly recognised teratogens, infectious agents and malnutrition may also have a teratogenic effect, directly or indirectly, on the foetus. In the past, it is likely that the majority of a population living with over-crowding would have regularly suffered some sort of recurrent low-grade infection. This would equate to a long-term exposure. Chronic diarrhoea (Markell *et al.*, 1992: 380) is a good example of this, and is closely associated with parasitic infection of the gut (Markell *et al.*, 1992: 380). Periodontal disease would probably be the commonest source of such recurrent infection, with micro-organisms belonging, for example, to the staphylococcal and streptococcal families feasibly being present in the mouth permanently. As the immune and nutritional systems are key components of biological response mechanisms, any metabolic disease process that leaves a mark on the skeleton may be a useful indicator of population-wide stress levels if found in sufficient numbers (Roberts & Manchester, 1995:163ff).

#### **5.2.ii.g** *Immune response and health in the past*

It will be appreciated that the immune responses outlined in Chapter 4 may well be compromised in the absence of modern treatment such as antibiotics, or in the presence of concurrent/recurrent disease or impairment processes in the liver, blood or gut. The addition of further antigens before the immune response has adequately dealt with earlier antigens, as in cases of recurrent infection for example, would have been a frequent complicating factor in the past. Problems with recurrent or concurrent infections over prolonged periods of life may have therefore inhibited the overall effectiveness of the immune response as well as failing to combat such health issues as parasitic infestation of the gut, for example. Dental infection may feasibly be an on-

going process, as may chronic diarrhoea. Both of these have the potential to keep the immune system operating at a reduced level when confronted with further infections. Combined with helminth or other parasitic infection, the gut and the immune response would be over-stretched for much of the individual's lifetime, possibly diverting nutritional resources away from such tasks as normal reproduction. Of course, not all infection is related to specific infectious diseases such as tuberculosis, leprosy, or treponemal disease. There are many types of non-specific infection that may enter an individual by one route or another (as a result of trauma, sexually transmitted disease, or other means) or reduced immune status, and have an adverse effect. In such circumstances, the individual may be more susceptible to a less-virulent variety of the pathogen, to which he would have been immune had he been in a better state of health. Opportunistic infections are a common occurrence today following medical procedures such as surgery (Reeves & Todd, 1996:160). In the past, they must have been equally, if not more, pervasive. Opportunistic infections are favoured by conditions in which host defences may be by-passed, e.g. burns, trauma, excema, or other conditions in which the integrity of the skin is breached; primary immunodeficiency disorders (problems with the production and maintenance of elements of the immune system, such as lymphocytes, leucocytes etc.), and conditions in which other diseases have reduced the host's immune status, e.g. other infections, (acute, or particularly chronic ones); metabolic disorders such as *diabetes mellitus*; protein-losing states; malnutrition; prematurity (of babies) and old age (Reeves & Todd, 1996:161). Secondary deficiencies such as those due to disease-impact on the body, disorders which involve protein loss, impaired protein uptake (e.g. poor diet or gut deficiency), chronic inflammatory diseases (including parasitic infestation), malnutrition, persistent infections, congenital viral infections e.g. rubella, passed from

mother to infant at parturition, and reduced acidity e.g. in the vagina, leading to candidiasis, all favour increased systemic infection (Reeves & Todd, 1996:193-5).

#### 5.2.ii.h *Stress in the palaeopathological and epidemiological framework*

Another useful means of surveying biological responses and quality of life in different environments is the analysis of indicators of stress. Life tables and mortality profiles are considered the best indicators of overall adaptation, and illustrate both chronic and severe periods, and types, of stress (Goodman *et al.*, 1988). Skeletal indicators of stress are generally accepted as a gauge of general levels of *malnutrition* and *infection* in the past (Lewis & Roberts, 1997), even though the assessment of the nutritional status of past populations “can be particularly difficult” (Stuart-Macadam, 1989a: 201). These two factors are thought by the author to be influential in the production of birth defects, although the mechanism is uncertain at this stage (Sture, 1997). The frequency of three stress indicators was recorded on the individuals in the samples being studied: *cribra orbitalia*, enamel hypoplasias and tibial periostitis were scored as either present or absent in the adult remains which it was possible to reliably assess (dependant on survival of relevant skeletal elements). These stress indicators are being recorded in order to see if there is any association with the rates of birth defects observed. *Cribra orbitalia* is believed to be associated with iron-deficiency anaemia and is believed to have a “potential synergism with infection” (Cybulski, 1977; Goodman *et al.*, 1988: 179; Stuart-Macadam, 1989a, 1989b). The presence of enamel hypoplasias is associated with nutritional stress and/or childhood disease according to Goodman *et al.* (1988) and may be related to decreased longevity; in effect, it is associated with episodes of acute stress on top of underlying undernutrition. Tibial periostitis is associated with chronic stress, impairing the normal functions of the

healing process but it may be due to other factors, including trauma, localised infection, soft-tissue ulcer, or a feature of some specific infectious diseases such as leprosy (Roberts & Manchester, 1995). Other indicators of stress include adult stature, which has been recorded for this study by the method of Trotter (1970).

Anthropometrics not only provide a sensitive measure of stressful conditions but are also related to variables such as nutrition and immune status (Chandra, 1975). It is recognised that “nutrition is a critical factor in the dynamic interrelationship between a population and its environment” (Stuart-Macadam, 1989a:201). Thus, in living populations, stature assessment is regarded as a key method of nutritional status analysis (Goodman *et al.*, 1988). Only adults are being observed for stress indicators in this study, as they are the reproductive component of the populations under examination. Juveniles with stress indicators died before being able to reproduce and pass on any unhealthy characteristics to the next generation.

A further aspect of stress which is considered in the interpretation of the results of this study is that of psychological stress (Ursin, 1994). An increasing amount of research during the last decade has shown that adverse psychological stress has a measurable effect on the immune response in those affected, and the author suggests that such stresses and responses may be implicated in the prevalence rates of birth defects as observed in the Medieval material. For example, young people moving into towns to work in industry or service, or any disadvantaged persons in an urban setting, who are removed from the support of family kinship networks and exposed to the less personal *milieu* of a town, may find themselves adversely affected in terms of their ability to fight infection (Egger, 1992; Hiramoto *et al.*, 1999; Oakley *et al.*, 1982; Ursin, 1994).



The psychological aspects of stress have probably been over-looked in the assessment of archaeologically-derived populations to date.

### **5.2.iii Suggestions made in this study**

Certain environmentally-related conditions may be factors in the aetiology of axial skeletal anomalies. These may include:

- the absence, or effective absence, of certain nutrients
- a reduced ability to fight infection
- the presence of active infection/parasitic infestation itself
- recurrent infection
- the presence of disease processes or environmental conditions making opportunistic infections more likely
- psychological stress in the mother leading to neuroimmunomodulation, thus affecting the foetus.

These effects may be due to:

- long lasting health effects in the mother due to her own childhood illnesses or deprivations
- the direct or indirect action of micro-organisms/chemicals (naturally-occurring or deliberately produced) on the developing foetus
- direct action of the above on the mother during pregnancy
- reduced maternal health status prior to becoming pregnant
- raised maternal temperature at critical periods

- maternal perception of stress and her ability to “cope” with stressors, such as feelings of hopelessness/helplessness (e.g. as in migrants in a new environment)
- compromised spermatogenesis in affected males (either as fathers, or as offspring of affected males). (Female oogenesis may be unaffected as females are born with their full complement of ova; alternatively, affected females may have developed altered ova themselves while *in utero* and then pass on genetic changes to their offspring).

#### **5.2.iv Practical issues: why macroscopic examination of the axial skeleton?**

The axial skeleton is the site of many congenital defects resulting from developmental impediments at an early stage of gestation (Barnes, 1994). These elements have been chosen for examination in this study partly because they are easy to recognise and to excavate even by non-osteologists and are therefore relatively well represented in collections. The axial skeleton is a relatively robust part of the body (Nawrocki, 1995) and this influences both its survival and recovery rate. Although pathological bone is known to decay more rapidly than healthy bone, and the congenital defects are classed as diseases, with their manifestations known as lesions, bones which express a birth defect are actually composed of *normal, healthy* bone material. The problem of survival of weakened pathological bone is not therefore necessarily a major issue when addressing birth defects of the skeleton, although the actual burial environment itself also affects preservation, with animal interference, inter-cutting of graves, moisture levels and pH of the soil being particularly influential (Garland & Janaway, 1989). A prime consideration within this study is also the effect of defects on an individual's quality of life. The maxilla, being the site for the development of cleft palate and/or cleft lip, the commonest craniofacial congenital malformation (Hagberg

*et al.*, 1998) and the vertebral column, being affected by several potentially serious defects that can inhibit life quality, are therefore ideal subjects for appraisal.

The appendicular skeleton is, of course, also the site for several serious birth defects, such as *talipes* (club foot), which may be diagnosed using the lower leg and foot bones. Similarly, polydactyly may affect the hands, but is known to have a strong genetic element (Ortner & Putschar, 1981, 362) and congenital dislocation of the hip may be seen in the acetabulum and femoral head, but can be confused with traumatic injuries to the femoral head. Many other appendicular birth defects are known, of course, but are not considered here. The drawbacks of considering defects of the appendicular skeleton include issues of excavation and recognition as well as differential survival. For example, in considering clubfoot, a differential diagnosis of the condition could be that of a post-paralytic deformation (Ortner & Putschar, 1981, 364); and, in the field, problems with recovery of hand and foot bones by untrained excavators could be a serious confounding factor in the study. As a result of these considerations, the author chose to focus on the axial skeleton as a source of data.

Macroscopic examination is deemed a satisfactory method of diagnosing the conditions under investigation in this study, as the defects are readily visible to the naked eye. Radiological examination is not necessary to either aid or confirm diagnosis, as the conditions are not manifested either microscopically or simply internally within the bone matrix itself. Rothschild *et al.* (1997) found macroscopic observation methods adequate for the distinguishing of leukaemia from other forms of cancer for broadly similar reasons, although he found that radiography could give more information once diagnosis had been made. The only scenario in which the

author believes that radiography could help in diagnosis would be in that of spondylolysis, in which weaknesses or cracks in the *pars interarticularis* could be revealed (pers. comm. J. McConnell). This is beyond the scope of this study, but is mentioned later under recommendations for further research. The skeletal material is therefore being examined macroscopically for selected defects, which are assessed as either present or absent.

Inter-observer error (Waldron & Rogers, 1991) is eliminated by the use of a single observer, and intra-observer error by random re-assessment of material during the course of the data collection. The conditions themselves are being diagnosed according to the criteria expressed by Barnes (1994), Resnick and Niwayama (1988) and others in the medical literature, and are therefore readily observable as such by any other interested parties by referring to these sources. Recording of the defects, stress indicators and height is being done according to the standards laid out in Buikstra and Ubelaker (1994) and recording forms are included in this volume.

Some data obtained from other workers have been used in the study. These include age and sex analyses for all the populations (Lee, 1986; Powell, c.1986; Grauer, 1989; Mays, Isaac, 1999). The author completed an age and sex assessment on approximately 45 individuals from the Raunds and Chichester materials (see Appendix x) in order to test the reliability of the data. The results matched those of the original workers. Stature data were also taken from the original reports on the Raunds population and from data from another thesis (Ward, 1996).

### 5.2.v Diagnostic protocol and criteria

The concern with many pathological lesions is that misdiagnosis is a possibility, although improvements in the theoretical and methodological aspects of palaeopathology should reduce the risks of this occurrence (Ortner, 1991; Wood *et.al.*, 1992). Fortunately, the congenital defects are a class of lesions that may be diagnosed with relative certainty, by combining a knowledge of skeletal anatomy, growth physiology and embryology. There are few differential diagnoses possible for the defects studied in this research. For example, in the diagnosis of cleft palate it is relatively straightforward to rule out post-depositional damage to the palate by examination of the edges of the lesion (by microscope if necessary), which would show a clean break with little or no healing and a sharp edge in the case of post-mortem degradation. There may also be a colour difference at the edges, with the bone being lighter at the break than in other areas in a case of post-mortem damage. Likewise, lumbarisation of the first sacral element in a complete sacrum may feasibly be confused with incomplete union of the first and second sacral elements, with a space visible between the vertebral bodies of S1-S2. However, an understanding of the process of skeletal development and an assessment of other age indicators should rule out a misdiagnosis of lumbarisation if the affected individual is, for instance, an adolescent/young adult. The ability to diagnose congenital defects depends greatly on an understanding of the embryological processes through which the foetus passes during gestation, as many defects are simply the result of developmental delays causing missed "critical period" progression (Barnes, 1994; Scott, 1986). Thus, the congenital defects are not the result of other recognised pathological processes which may act on bone as are, for instance, the infectious diseases (Ortner, 1991). Congenital

malformations are solely the result of insults to the foetus whilst it is *in utero*, and are not of post-natal origin.

It has been suggested that any specialist examining material which he/she has not personally excavated, is at a serious disadvantage (Nawrocki, 1995). This is, of course, an important limiting factor in the assessment of skeletal material. However, it is not considered to be too great a limitation in this study, because the bones selected for examination are relatively easy to identify and excavate in a grave context, with a minimum of recognition training. The skull and the vertebrae, for instance, as well as being relatively robust elements of the skeleton, are not as likely to be lost in the soil as small phalanges or broken rib ends. Environmental factors which may have had an influence on the taphonomic processes (Nawrocki, 1995; Ubelaker, 1995) acting on the observed samples may be identified and accounted for by utilizing the information obtained from the relevant archaeological reports of the burial sites. All of the sample populations in the study are from discrete burial grounds, notionally protected from scavenging animals, for example, but some interference is bound to have occurred. Graves were purposely dug for the interment of remains, and apart from some intercutting, appear to have remained undisturbed and protected from the direct effects of weathering by wind and rain as all were taken from below the ground surface.

#### **5.2.vi Considering the interpretative challenges in palaeopathology**

Wood *et al.* (1992) identified three challenges to researchers which have been considered in the design of the project: “demographic non-stationarity; selective mortality, and unmeasured, individual-level heterogeneity in the risks of disease and death” (1992: 343). Demographic non-stationarity refers to a population’s move from

a stationary state (a population in which equilibrium of fertility and mortality leads to zero growth and which is closed to migration) to a non-stationary state. The Medieval settlements observed in this study include both states, with the town populations being supplemented at various rates and times by incoming rural migrants, and the rural groups maintaining fairly close-knit communities within themselves (Goldberg, 1992; Patten, 1973; Stone, 1966: 23). The problem of identifying rural migrants in town cemeteries is one which may be helped by the work of Budd and others (see above) which considers isotope analysis of dental enamel as an indicator of childhood environment. However, as the period of use of the observed town cemeteries covers centuries rather than decades, it is suggested that any recent arrivals within them will have little effect on the final results.

The problem of selective mortality, by which there is always an inherent bias in skeletal as well as clinical studies, means that age-at-death and specific lesions do not have to be directly related (Wood *et al.*, 1992). We do not have access to the whole population, either living or dead, and we thus rely on the data of a selective sample (Waldron, 1994). This study is no different from any other research in the field of palaeopathology with respect to selective mortality. It is only possible to show the congenital lesions observed, and offer figures such as age-at-death and divisions by sex as they are seen in the available material. Interpretation is another question entirely. For example, rural sites may be more likely to undergo total excavation than urban ones, as more restrictions apply to excavation of urban sites. This in turn limits the amount of material available and the interpretation possible. However, if the results show, for instance, a consistently lower age-at-death for certain defects, it may be possible to suggest a relationship between that defect and mortality risks.

Hidden heterogeneity is the problem of individual frailty or susceptibility to disease. As large skeletal samples such as those under observation in this study usually cover a long period of time, it is difficult to determine individual risks of death. Variables influencing risk of death include socio-economic factors, genetic predisposition, and environmental considerations (Wood *et al.*, 1992: 345) amongst others. Obviously, Medieval populations experienced such variations constantly, and it is impossible to make accurate interpretations about the risks to individuals from the skeletal samples examined. However, this study aims to record and analyse the prevalence of congenital defects within and between urban and rural *populations*. By applying current clinical and environmental knowledge to the results it is hoped that suggestions may be made as to the causes of any difference in defect prevalence rates. The results will only be applicable to the groups as a whole, however, and cannot be taken as representative of specific risks to any individual, unless there are, for instance, some outstandingly obvious figures related to certain defects which make death by a certain age highly likely.

#### **5.2.vii Diagnostic criteria**

This section explains the specific criteria used to diagnose each of the conditions quantified in the study.

*Block vertebrae* are diagnosed where two or more elements are deemed to be “fused” as the result of a developmental union rather than by trauma or sepsis;

*absent vertebrae* are diagnosed when a complete segment is present along with the adjacent element of the next segment;



*extra vertebrae* are diagnosed when a complete segment is present;

*cervical/lumbar ribs* are diagnosed when costal facets or bony extensions or tubercles are present on cervical or lumbar vertebrae;

*spina bifida occulta* is diagnosed when the neural arches of the lumbar or upper sacral elements (S1 – 3) are open and flared outwards, reflecting the growth of the arches over an extrusion of meninges or lipoma in life, i.e. the presence of a neural tube defect; occasionally this effect may be found in other segments of the column;

*cleft neural arch* is diagnosed when any adult vertebral arch is either fused off-centre, is open to any degree, or, in the case of cleft sacrum, is unformed (i.e. not the result of a neural tube defect); cleft neural arch is diagnosed in juveniles if it is apparent that all other vertebral arches have already fused and the anomalous arch has bony tips which are clearly “complete” rather than being the last to fuse as a function of maturation; apparent clefting of S5 is not included in this study as it is a feature of almost every sacrum observed. S1, however, is usually closed, so an opening at that level is included in the data as either a cleft neural arch or as a spina bifida occulta (if flared outwards);

*border shifting* is diagnosed when at least one complete segment is present along with the adjacent element of the next segment, e.g. the shift may be identified as lumbarisation or sacralisation, or as an unidentified border shift if all elements are not present. It is also the diagnosis for border shifts at the higher segmental junctions.

Identification of sacralisation or lumbarisation archaeologically may be problematic in the absence of complete segments. For example, identifying a border-shift in a sacrum with damage to the lower elements may be difficult. If all lumbar or sacral elements are present, the identification is more straightforward. However, familiarity with the normal anatomical variations of the lumbar and sacral elements can help. Where there is doubt about the direction of the segmentation anomaly, the variation can be simply termed a “border-shift”;

*spondylolysis* is diagnosed where there is an apparent break (well healed) at the *pars interarticularis* between the neural body and the lower articular facets;

*spondylolisthesis* is diagnosed where there is slippage of a vertebra affected by spondylolysis, over the vertebra below it, as a result of the spondylolysis. There is usually osteophytosis on the margins of the affected vertebrae as the elements attempt to hold together.

*cleft palate* is diagnosed if a developmental cleft is observable, either anteriorly, affecting the premaxilla and palatine process, or posteriorly, affecting the back of the hard palate close to the posterior nasal spine (an occult defect);

*cleft lip* is diagnosed by the presence of an uneven nasal aperture as described by Barnes (1994); and/or by the abnormal development or placement of the canines and/or incisors in the maxilla (Barnes, 1994);

*tibial periostitis* is diagnosed when present as either healing or active new bone formation on the tibiae (Buikstra & Ubelaker, 1994);

*enamel hypoplasias* are diagnosed when at least three teeth show signs of enamel defects such as discoloured lines, patches, or grooves (Buikstra & Ubelaker, 1994);

*cribra orbitalia* is diagnosed when porosity, pitting or coalescence of pits is present in either orbit (Buikstra & Ubelaker, 1994);

*age and sex* have been assigned according to data gathered by previous workers (Powell 1996; Lee, 1986; Grauer, 1989; Mays, unpublished; Isaac, 1999). Random checks done on the Chichester and Raunds Furnells material revealed no discrepancies between the age and sex assignments of the original observers and those of the author. Methods used by the author were, for ageing: pubic symphyseal changes (Brooks & Suchey, 1990), auricular surface changes (Lovejoy *et al.*, 1985), dental attrition (Brothwell, 1981,1989), sternal rib end changes (Iskan *et al.*, 1984; Iskan *et al.*, 1985), cranial suture obliteration (Masset, 1989; Meindl & Lovejoy, 1985); for sexing: skull and pelvic characteristics, based on work by Bass (1987), Phenice (1969), and White and Folkens (1991). Data were also taken from the work of the original observers as listed above for the St. Helen's, Raunds Furnells, Wharram Percy and Hull collections. The Chichester material was assessed for stature by Ward (1996). In these two latter collections, stress indicators were only observed in females, as they had formed part of a pilot study. Waldron found in a methodological test that the Trotter method resulted in considerable underestimation of male height, no matter which bone was used, but female heights did not show the same effect (Waldron, 1998). The test related to two English populations from Hertfordshire and Yorkshire; it may be that the male statures in the populations observed in this study are therefore

underestimated. However, as the method has been applied consistently across the populations, this should not significantly alter the conclusions drawn from the results.

#### **5.2.viii Statistical analysis**

Birth defect distributions will be shown for all populations in both adults and sub-adults. Male and female populations will also be compared for birth defect and stress indicator rates. Rates will be shown as percentages of observed populations on bar charts for ease of understanding. Age-at-death distributions for those affected will be compared to those unaffected. Age groups were assigned by the original workers (as above), and took the following categories: neonate (newborn – 1 month); infant (above 1 month-12 months); juvenile (above 12 months – 16.9 years); young adult (17-24.9 years); mid-adult (25 years – 34.9 years); mature adult (35-44.9 years); and 45 years and over. Comparison will be made of mean population stature in both males and females, and between those with and those without birth defects/stress indicators. Chi-squared distributions will be calculated for the rates of defects and stress indicators observed between populations and between males and females.

#### **5.2.ix Cultural responses to disease**

Finally, it must be noted that the absence of certain congenital defects in the archaeological record is not to be taken as proof of low incidence rates in life. Cultural factors play a part in the formation of the archaeological record, and these may have an effect on the material that we see entering the record. For instance, primary cleft palate (at the back of the mouth) appears to be a rare condition in British skeletal populations (Anderson, 1994a), although there are more recorded examples of Native American individuals living into adulthood (Barnes, 1994). In reality, many babies

born in the past with severe cleft defects affecting the facial appearance may well have been killed directly or indirectly (e.g. exposure or neglect), or may have died through undernutrition due to impaired ability to suckle (Bacher *et al.*, 1998; Kellum, 1974). Even babies with minor, “invisible” defects at the back of the mouth are likely to have developed severe pharyngeal ulceration which would have reduced their intake and possibly lead to an early death (Bacher *et al.*, 1996), although their cleft defects would have been occult and they may have been expected to have reached the cemetery at death. Babies with manifest cleft defects who were subject to infanticide would almost certainly have been disposed of illegally (Kellum, 1974) and would therefore not have reached the burial ground. Most other defects studied in this research, however, such as those affecting the vertebral column, were occult and may have affected the individual only in his/her ability to move and work free from pain and reduced mobility. As cultural attitudes and concepts of disease involve the *recognition* of disease, interpretation of burial treatment of individuals with occult defects may be problematic. Cultural factors are probably unlikely to have played a part in the way such individuals entered the archaeological record, as invisible defects would not have been recognised.

### **5.2.x Summary of materials and methods**

- three late Medieval sample populations from the parish churchyard of St. Helen-on-the-Walls, York, the churchyard at Wharram Percy, North Yorkshire and the burial ground of the Augustinian Friary, Hull, Humberside, are to be examined macroscopically for the presence/absence of certain birth defects (as outlined above)

- results from these sample populations will be compared with results obtained in a pilot study (Sture, 1997) which examined sample populations from the Anglo-Saxon churchyard at Raunds Furnells, Northamptonshire, and the burial ground of the hospital of St James and St Mary Magdalene, Chichester, West Sussex, using the same methodology
- data will be collected from adults and sub-adults
- data will include presence/absence of the birth defects outlined in 5.2.vii above, along with stature, and the stress indicators cribra orbitalia, enamel hypoplasias and tibial periostitis (all in adults only)
- statistical analysis will include distribution of birth defects and stress indicators as percentages of the observed populations, chi-squared distribution of defects and stress indicators between populations and between males and females, mean stature within and between populations and between males and females, Student's t-test on the mean stature between populations and males and females, and comparison of age-at-death statistics for those affected with birth defects compared to those unaffected
- the results will be assessed against the medical and related background as outlined in Chapter 4.

## CHAPTER SIX

## Results

## 6.0 Overall figures

From the five populations examined, a total of 1442 individuals were found to have the necessary elements present to provide data regarding defect presence or absence, and/or stature. Table 6.I shows the observed numbers of individuals from each site. All five population samples provided evidence of the presence of congenital skeletal anomalies affecting both sexes, and sub-adults (see Table 6.II).

Table 6.I: Number of observed individuals from each site.

Site	Male	Female	Unsexed adult	Sub-adult
Chichester	151	74	6	40
Raunds Furnells	88	67	1	57
Hull Mag. Ct.	119	46	10	19
St Helen's, York	141	140	39	65
Wharram Percy	172	117	9	79
<b>Total</b>	<b>671</b>	<b>444</b>	<b>65</b>	<b>260</b>

Table 6.II: Number of individuals with defects from each site (% of sexed/aged population)

Site	Male	Female	Unsexed adult	Sub-adult
Chichester	93 (61%)	34 (45%)	4 (66%)	12 (30%)
Raunds Furnells	40 (45%)	20 (29%)	0	10 (17%)
Hull Mag. Ct.	44 (36%)	8 (17%)	2 (20%)	7 (36%)
St Helen's, York	67 (47%)	57 (40%)	7 (17%)	18 (27%)
Wharram Percy	44 (25%)	38 (32%)	1 (11%)	19 (24%)
<b>Total</b>	<b>288</b>	<b>157</b>	<b>14</b>	<b>66</b>

The urban sites of St Helen-on-the-Walls, York, and the hospital cemetery of St. James and St Mary Magdalene, Chichester, had the highest defect prevalence overall in absolute terms. The rural sites of Raunds Furnells and Wharram Percy had lower

overall defect rates, and the Hull site, whilst an urban cemetery, appeared to mirror closely the rural pattern of defects. Unsexed adults were not included in the overall pattern of defect rates for each site, nor in the breakdown of defects by sex, age-at-death, or stress indicators. Juvenile defects are addressed in section 6.4.

The overall prevalence of defects among adults is shown in Fig. 6.1 below.

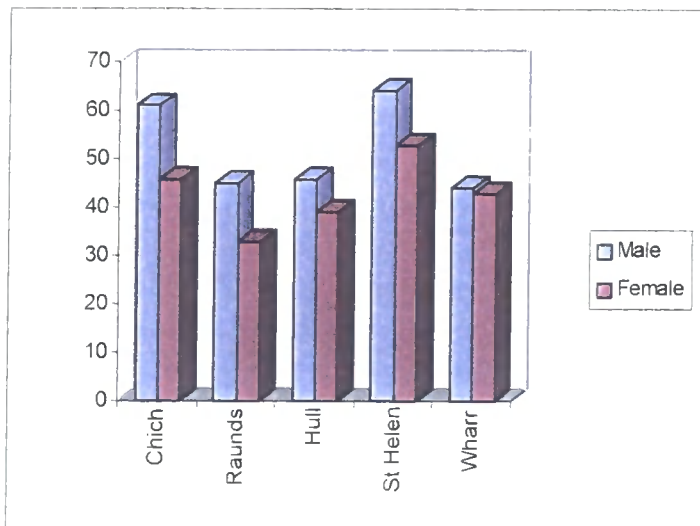


Fig. 6.1: percentage with defects per population (n=445)

## 6.1 Adult results by defect

### 6.1.a Sacralisation

Figure 6.2 shows the evidence for sacralisation.

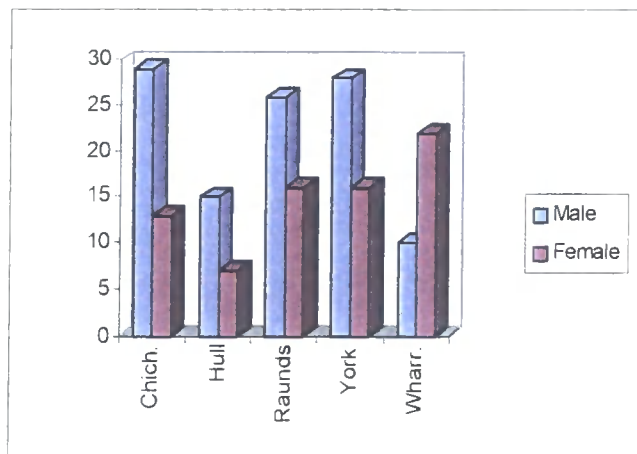


Fig. 6.2: percentage of individuals with sacralisation (n=147)



Males had higher rates of sacralisation than females in all sites except Wharram Percy. Chichester males had the highest prevalence, while Raunds and St Helen's had similar male and female prevalences. Wharram, whilst having the only low male prevalence, also had the highest female rate of all. Hull had the lowest female prevalence. Chichester expressed the highest rate of sexual dimorphism and Raunds the least. Sacralisation was significantly associated with other spinal anomalies, with 69% of those affected overall having at least one other spinal defect. The rates at Hull and Wharram were similar, with 50% and 45% respectively of affected individuals having associated spinal anomalies. The other three sites all had co-occurrence rates between 23-25%.

Chi-square tests showed significant differences between males in Raunds and Wharram at 95%; Wharram and York at 99%; Chichester and Hull at 99%; and York and Hull at 95%. Among females, chi-square tests showed significant differences between Raunds and Wharram at 95%; Wharram and Chichester at 99.5%, and Wharram and York at 95%. In terms of urban/rural comparison, there was no significant difference in sacralisation rates between rural and urban sites overall. However, there were differences between specific sites, which may suggest the involvement of some environmental factors.

#### **6.1.b** *Lumbarisation*

Figure 6.3 shows the evidence for lumbarisation. Males expressed more lumbarisation than females except at Chichester. Hull rates were the same for both sexes. York males had the highest prevalence, at twice the rate of Chichester and almost three times the rate of Raunds. Wharram showed the highest female proportion within a

population. Raunds showed the greatest sexual dimorphism, and Hull the least.

Lumbarisation was heavily associated with the presence of other defects, with 87% of those with lumbarisation overall also having at least one other spinal defect. The rates varied between sites, with all affected individuals at Hull and Raunds having other associated spinal anomalies. At Chichester, 94% had other anomalies present.

However, at Wharram, only 55% had other associated spinal anomalies.

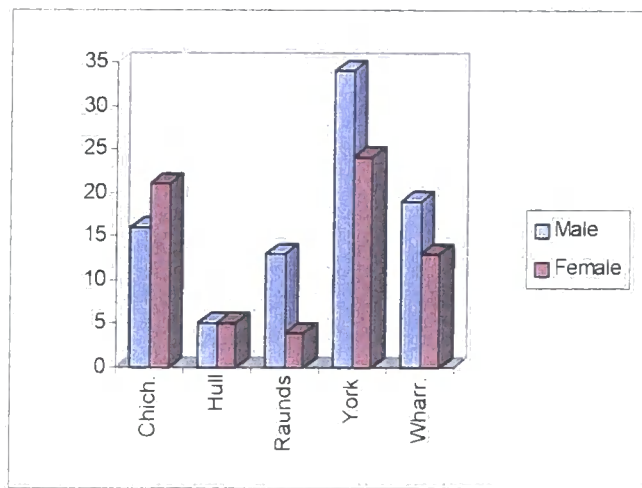


Fig. 6.3: percentage of individuals with lumbarisation (n=104)

Chi-square tests showed significant differences between males in Raunds and York at 99.5%; Wharram and Hull at 95%; Chichester and York at 99.5%; and York and Hull at 99.9%. Among females, there were significant differences between Raunds and Chichester at 95%; Raunds and York at 99.5%; Wharram and Chichester at 99.5%, and Wharram and York at 99.9%. A statistically significant difference was noted between the urban and the rural sites in lumbarisation prevalence at 97.5% on a chi-square test.

### 6.1.c *Spina bifida occulta*

Figure 6.4 shows the evidence for spina bifida occulta. The chart represents absolute numbers, not the percentage of the population.

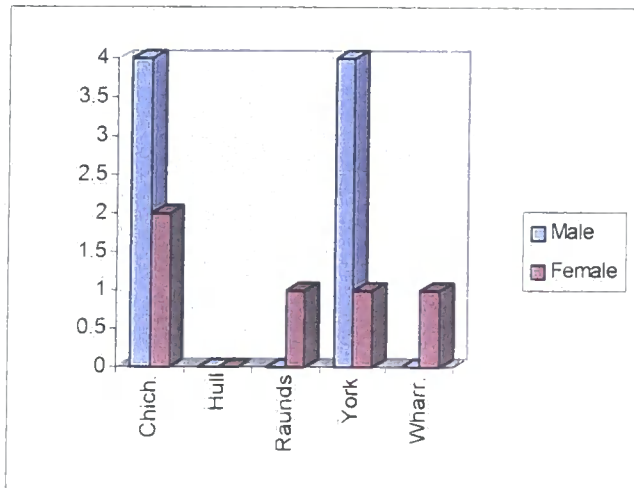


Fig. 6.4: individuals with spina bifida occulta (n=13)

These cases of spina bifida occulta, ie. individuals with probable neural involvement associated with open neural arches, were diagnosed by the flaring nature of the bony edges of the cleft (Barnes, 1994). Rates of spina bifida occulta were very low in all populations, therefore interpretation is not straightforward. Juveniles were also affected at Raunds and Wharram Percy, and are included in the section on juvenile results. No spina bifida occulta was observed at Hull. Of those affected with spina bifida occulta, 60% had other spinal anomalies also present, and all but one of the affected females also had stress indicators present.

Chi-square tests were not possible except between Chichester and York, and showed no significant difference in prevalence.

### 6.1.d *Cleft Neural arch*

Figure 6.5 shows the evidence for cleft neural arch.

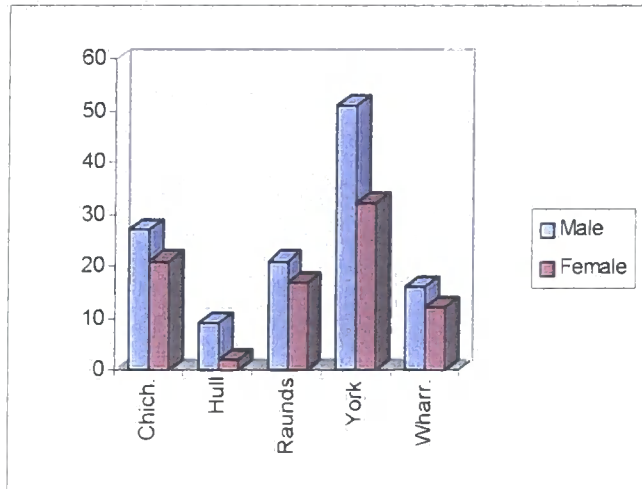


Figure 6.5: percentage of individuals with cleft neural arch (n=219)

Males expressed higher prevalences than females in all sites. York showed the highest male and female rates, with over 50% of males affected. Hull had the lowest rate. The female rate in Hull was very low. Wharram had the least sexual dimorphism and Hull has the greatest. Within the affected populations, Chichester, Wharram and Raunds have similar proportions of males and females. Of those affected with cleft neural arch, 58% also had other spinal anomalies present. Variations between sites were observed, with Chichester and York having the highest rates of co-occurrence with other spinal anomalies (65% and 68% respectively), whilst Hull and Wharram had the same rate of co-occurrence, at 35%.

Chi-square tests showed significant differences between rural and urban populations (chi-square = 27.12, at 99.9%), and also between Chichester and York.

### 6.1.e *Supernumerary vertebrae*

Figure 6.6 shows the evidence for supernumerary vertebrae. Most of these cases involved an extra lumbar element, although 3 cases at Raunds Furnells involved an extra cervical element. An extra thoracic element was observed at York, along with 3 cases of a sixth sacral element.

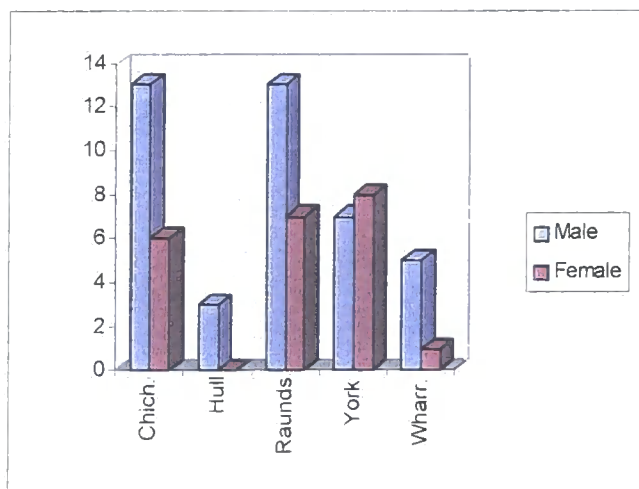


Fig. 6.6: percentage of individuals with supernumerary vertebrae (n=56)

Chichester and Raunds males had the highest prevalence of supernumerary vertebrae. Chichester, York and Raunds female rates were similar. Wharram had a very low female rate. Only in York did the female rate exceed the male prevalence. Hull did not have any affected females. There was an 81% rate of co-occurrence with other spinal anomalies overall. Chichester had the highest rate of co-occurrence with other spinal anomalies, with 91% of those with an extra vertebra also having at least one other anomaly present. Hull had the lowest rate of co-occurrence, with only 60% of those affected having another spinal anomaly also.

Chi-square tests showed significant differences between males in Raunds and Hull at 95%; Wharram and Chichester at 95% and Chichester and Hull at 99%. Among

females, significant differences were observed between Wharram and York at 95%.

No significant difference was noted between urban and rural populations overall.

#### 6.1.f *Absent vertebrae*

Figure 6.7 shows the evidence for absent vertebrae.

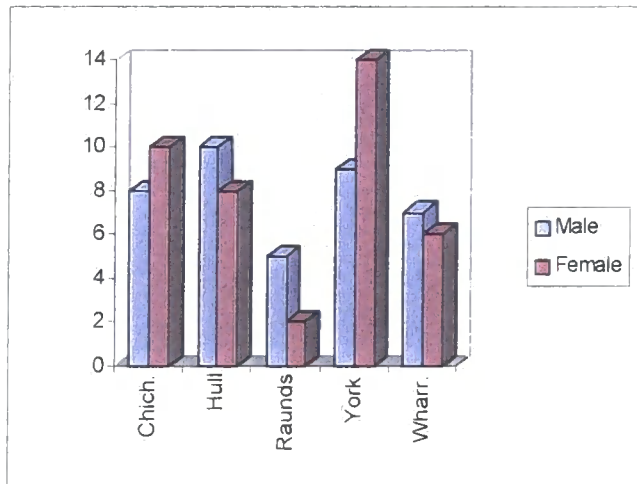


Fig. 6.7: percentage of individuals with absent vertebrae (n=69)

Absent vertebrae were noted in all segments of the vertebral column. York females had the highest prevalence of absent vertebrae. Raunds males and females had the lowest rates. Chichester, Wharram, York and Hull males had similar rates. Within the affected populations, Wharram has the least sexual dimorphism and Raunds the greatest. There was an overall rate of co-occurrence with other spinal anomalies of 52%. At Wharram, 94% of those with an absent vertebra had another spinal anomaly, but only 32% of those at Hull also had another anomaly.

Chi-square tests showed no significant differences between males in any sites. Among females, the only significant difference was noted between Raunds and Wharram, at 95%.

### 6.1.g *Fused vertebrae*

Fused or block vertebrae were observed at all sites except York, but in very low numbers. The distribution is shown in Table 6.III below. Females were more commonly affected than males, and one juvenile was affected at Raunds.

Table 6.III: fused vertebrae

Site	Male	Female	Juvenile
Chichester	0	3	0
Hull	1	0	0
Raunds	2	2	1
Wharram	0	1	0

Klippel-Feil syndrome was observed in three individuals. The Hull male and one of the Raunds males both had a fusion of C3-4, and the Hull male had an associated partial unilateral spondylolysis of C4 (type II); the Raunds male had sacralisation of L5 (type III). One Raunds female had a fusion of C4-6 (type II). Fused lumbar elements were noted in one of the Chichester females (L5/6) and the Raunds juvenile (L4/5). Fused mid-thoracic vertebrae were observed in two Chichester females, with fusions of 3 and 4 adjoining elements between T4-8. One Raunds female had fusion of T1-2 and a Raunds male had a fusion of T10-11. The Wharram female had a fusion of T2-3. None of these thoracic fusions were due to infection (such as tuberculosis, for example). All were clearly congenital "fusions" which were in fact solid units which had developed as a single entity during the embryonic period. Of the ten affected individuals observed, 5 had other spinal anomalies present.

### 6.1.h Spondylolysis

Figure 6.8 shows the evidence for spondylolysis.

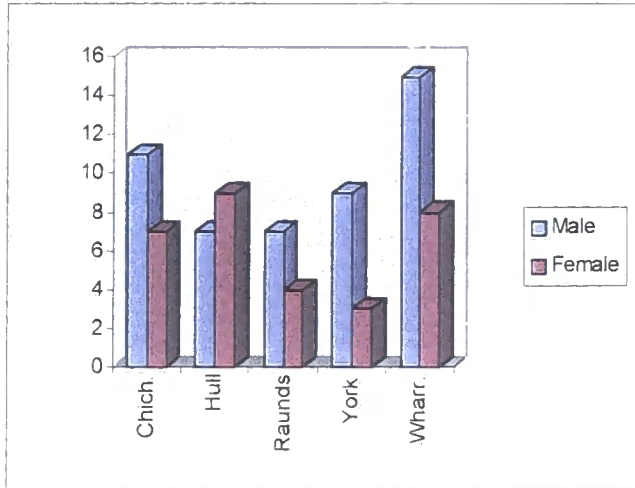


Fig. 6.8: percentage of individuals with spondylolysis (n=63)

More males than females had spondylolysis in all sites except Hull. Chichester and Wharram had the highest male prevalences, and Hull had the highest female prevalence. York had the lowest female prevalence. Within the affected populations, York had the greatest sexual dimorphism, and Hull the least. Co-occurrence with other spinal anomalies (including spondylolisthesis) varied between 56% at Wharram and 86% at Raunds.

Chi-square tests showed no significant differences between males or females at any sites.



### 6.1.i *Spondylolisthesis*

Figure 6.9 shows the evidence for spondylolisthesis. Rates were low, therefore interpretation is difficult.

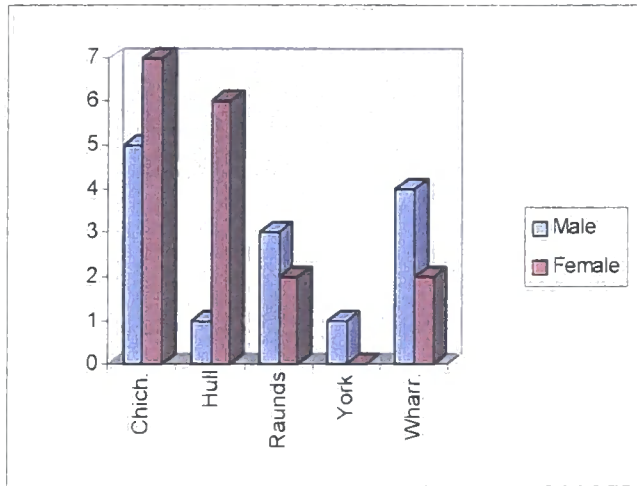


Fig. 6.9: percentage of individuals with spondylolisthesis (n=20)

Chichester had the highest rates among males and females. No females in York had the condition. Wharram males had a high prevalence, and York the lowest male rate. Within the small affected populations, after York, the greatest sexual dimorphism was seen in Hull. Raunds had the least sexual dimorphism. Chichester had the highest associated rate of spondylolisthesis at 45%, and York the lowest rate at 15%.

Chi-square tests showed no significant differences between males or females at any sites.

### 6.1.j *Cleft defects*

A total of 730 maxillae were sufficiently well preserved to enable an assessment of the presence or absence of anterior and/or posterior clefting. No definite cases of cleft palate affecting the palatine process or the palatine bones were diagnosed, but several cases of cleft lip/palate were noted, as outlined in Table 6.IV below.

Table 6.IV: cases of cleft lip/palate (absolute figures)

Site	Male	Female	Juvenile	Adult
<b>Chichester</b>	1	1	0	0
<b>Raunds</b>	0	1	1	1
<b>York</b>	1	3	0	0
<b>Hull</b>	0	1	0	0
<b>TOTAL</b>	<b>2</b>	<b>6</b>	<b>1</b>	<b>1</b>

An adolescent from Raunds (5311) had several oro-facial anomalies, including a midline cleft lip, with extremely protruding front teeth. The central maxillary incisors were missing, with signs of longstanding infection around the premaxilla, which was incomplete. Infection may have weakened the bone and led to post-mortem loss, but the remaining incisors were so protruding that some developmental problem was obviously present. It is possible that the central incisors were congenitally absent, a sign of midline cleft lip/palate. The nasal aperture was also uneven along the lower border, due to unilateral hypoplasia of the premaxilla, giving rise to a unilateral cleft lip. The presence of such significant levels of infection (the incisive suture was obliterated) is commensurate with oro-nasal anomalies and is common among cleft defect sufferers. In addition, the palatine process was bilaterally open, but this may have been due to post-mortem damage.

A mild expression of midline cleft lip was observed in an adult female from York (5378), consisting of a midline groove running through the alveolar bone of the maxilla, also affecting the midline of the mandible (Barnes, 1994:189). A diastema was formed between the central incisors of both jaws, with the groove extending several millimetres into the bones. The left upper canine was also congenitally absent.

A midline cleft lip with possible associated secondary cleft palate was noted in an adult from Raunds (5152). This consisted of an unusual facial configuration in which the two halves of the premaxilla joined in the midline, with the right half being hypoplastic. The lower margin of the nasal cavity was even, and the difference in depth of the premaxilla was apparent in the uneven line of the incisors (see fig.xx - drawing). The incisors and canines were confused and unevenly arranged. A possible cleft palatine process was observed, but there was also some post-mortem damage present to the roof of the mouth.

A mild expression of bilateral cleft lip was noted in a male adult from York (5264) consisting of congenital absence of the right and left maxillary lateral incisors. The mandibular lateral incisors were rotated by about 45 degrees. The very small size of the maxilla and mandible may be diagnostic of Pierre Robin syndrome, which involves a small jaw and is often associated with cleft defects. Similar mild bilateral cleft lip was noted in two adolescents from York (5240 and 5232) who both had congenital absence of the maxillary lateral incisors, with one (5240) also lacking the left maxillary first premolar.

A female from Hull (1027) had an uneven nasal aperture, which inclined severely to the left, indicating hypoplasia of the left blastemal frontonasal process. Had the right side not successfully joined the hypoplastic left side, a naso-ocular cleft would have resulted. The facial appearance would have been abnormal.

A male from Chichester (41) also had an uneven nasal aperture, with hypoplasia of the right premaxilla, giving a right-sided unilateral cleft lip. Six incisors were present in the maxilla, with the right third incisor clearly a canine-incisor hybrid. This indicates confusion at the maxillary -premaxillary junction.

A Chichester female (190) also had a unilateral cleft lip, with right premaxillary hypoplasia resulting in an uneven nasal aperture. The front incisors protruded greatly.

A Raunds female (5371) also had right premaxillary hypoplasia giving rise to a unilateral cleft lip, with confusion of the incisors and canines, all of which had characteristics of each other in shape and size. The deciduous incisors were retained.

A York female (5666) had an unusual facial configuration, with a twisted appearance to the nasal aperture, which inclined severely to the right, possibly with hypoplasia of the right nasal bone or the right blastemal frontonasal process. The orbits were asymmetrical, with the upper outer margin of the right orbit taking a low line across the eye, suggestive of some disturbance to normal skull shape development. The right maxillary canine was twisted and sat behind the lateral incisor and first premolar, suggesting a unilateral fusion problem between the maxilla and premaxilla. This is suggestive of a right sided cleft lip. The right eye would also have appeared abnormal.

A York female (5357) had an uneven nasal aperture, with a right-sided hypoplasia of the premaxilla giving rise to a unilateral cleft lip, but the nasal aperture was twisted across to the left, with the vomer noticeably deflected to the left. The teeth were normal. The facial appearance would have been abnormal.

## **6.2 Sex and prevalence of conditions**

Among the sample populations observed for this study, males had the highest prevalence of the majority of conditions. However, fused vertebrae were found more commonly in females than males in three populations (Chichester, York, and Wharram Percy). Absent vertebrae and supernumerary vertebrae were also more common in females in the York sample. Equal rates between males and females of cervical rib/C7 anomalies were found in Chichester and Raunds, and equal rates of fused vertebrae were found in Raunds. As the prevalence of these conditions is very low, interpretation of these results may be problematic.

Among stress indicators, males suffered the highest rates of all types among populations where they were assessed, except for the females of York and Wharram Percy, who had higher rates of cribra orbitalia.

## **6.3 Single and multiple conditions**

Females were more likely to have multiple defects than single defects in all populations except Wharram Percy, where four times more women have single defects than multiple defects. Males in Chichester, Raunds and York had more multiple defects, but in Hull and Wharram they had more single defects. Juveniles all

had more single than multiple defects. This appears to be the opposite pattern shown by most of the adults.

Figures 6.10 - 6.14 show the single versus multiple defect distributions, measured as percentages of observed populations.

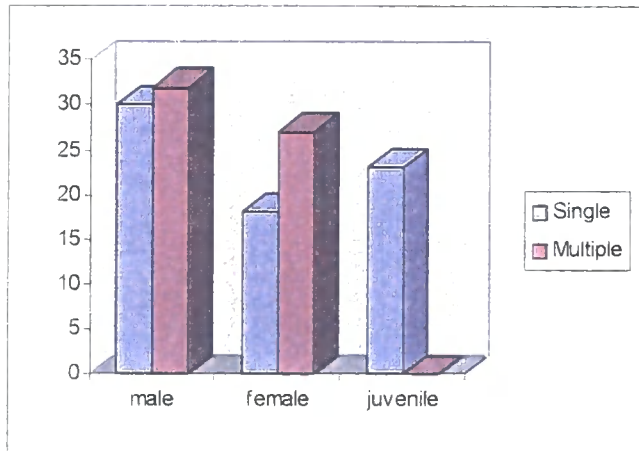


Fig. 6.10: Chichester single vs. multiple defect distribution

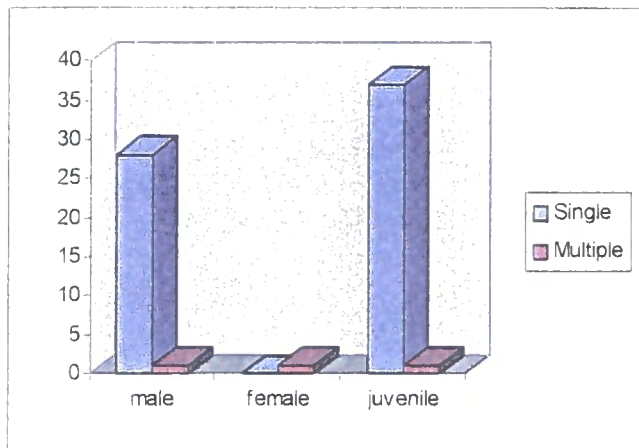


Fig. 6.11: Hull single vs. multiple defect distribution

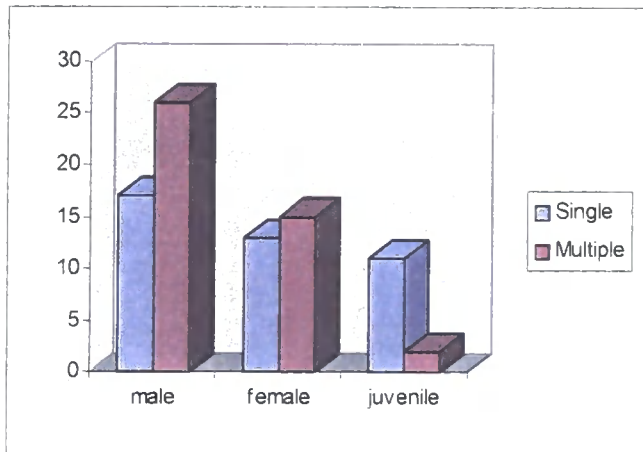


Fig. 6.12: Raunds Furnells single vs. multiple defect distribution

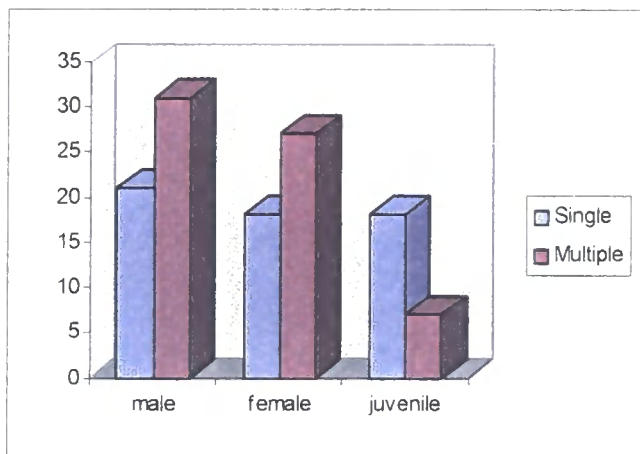


Fig. 6.13: York single vs. multiple defect distribution

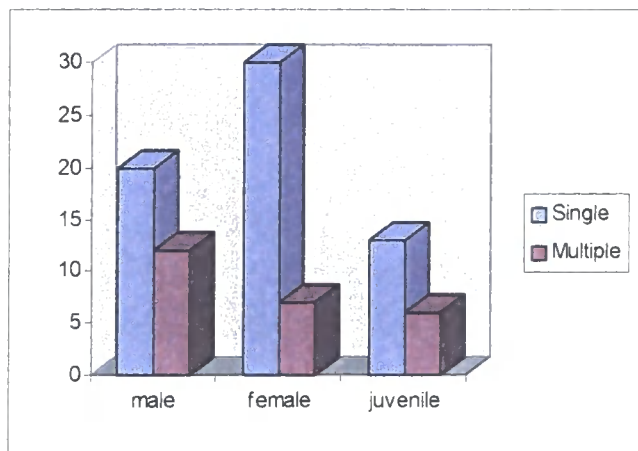


Fig. 6.14: Wharram Percy single vs. multiple defect distribution

## 6.4 Juvenile results

Juvenile defect prevalence is laid out below in Figure 6.15 according to condition, and Figure 6.16 according to site.

Table 6.V: juvenile defect distribution (percentage of juveniles examined)

Site	Juveniles affected
Chichester	30%
Raunds	17%
Hull	36%
York	27%
Wharram Percy	24%

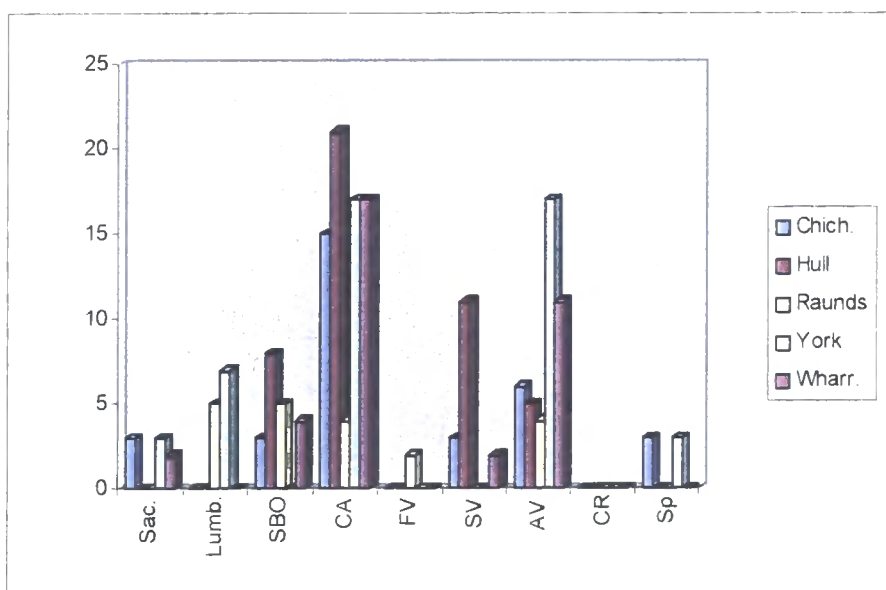


Fig. 6.15: juvenile defect prevalence by condition (% of juvenile population)

sac. = sacralisation  
 lumb. = lumbarisation  
 SBO = spina bifida occulta  
 CA = cleft neural arch  
 FV = fused vertebrae  
 SV = supernumerary vertebrae  
 AV = absent vertebrae  
 CR = cervical ribs/C7 anomalies  
 Sp = spondylolysis



The only conditions not represented in the juvenile populations were cervical ribs and C7 anomalies. The most commonly represented defects were cleft neural arch, supernumerary and absent vertebrae. Significant differences were observed only between the rates of juvenile defects between Hull and Raunds at 99%, Hull and Wharram at 95% and Raunds and York at 95%. A comparison between adult and juvenile defect rates is laid out in Table 6.VI.

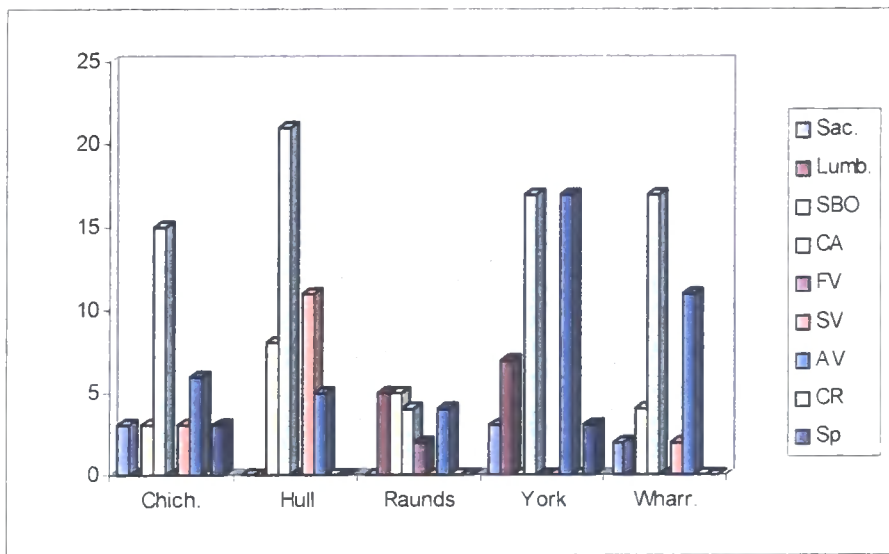


Fig. 6.16: juvenile defect prevalence by site (% of juvenile population)

Table 6.VI: comparative prevalence of defects among adults and juveniles (% of adult and juvenile populations).

	Chichester		Hull		Raunds		York		Wharram	
	<i>Adult</i>	<i>Juve.</i>	<i>Adult</i>	<i>Juve.</i>	<i>Adult</i>	<i>Juve.</i>	<i>Adult</i>	<i>Juve.</i>	<i>Adult</i>	<i>Juve.</i>
<b>sac</b>	21	<b>3</b>	11	<b>0</b>	16	<b>0</b>	17	<b>3</b>	12	<b>2</b>
<b>lumb</b>	13	<b>0</b>	14	<b>0</b>	6	<b>5</b>	28	<b>7</b>	17	<b>0</b>
<b>SBO</b>	3	<b>0</b>	0	<b>8</b>	2	<b>5</b>	5	<b>0</b>	1	<b>4</b>
<b>CA</b>	19	<b>15</b>	11	<b>21</b>	16	<b>4</b>	39	<b>17</b>	9	<b>17</b>
<b>FV</b>	1	<b>0</b>	1	<b>0</b>	3	<b>2</b>	1	<b>0</b>	0.3	<b>0</b>
<b>SV</b>	10	<b>3</b>	2	<b>11</b>	7	<b>0</b>	5	<b>0</b>	2.4	<b>2</b>
<b>AV</b>	8	<b>6</b>	8	<b>5</b>	2.5	<b>4</b>	11	<b>17</b>	5	<b>11</b>
<b>CR</b>	1	<b>0</b>	1	<b>0</b>	1	<b>0</b>	1	<b>0</b>	0	<b>0</b>
<b>Sp</b>	9	<b>3</b>	6	<b>0</b>	4	<b>0</b>	5	<b>3</b>	9	<b>0</b>

*Sacralisation* was not greatly represented among juveniles, only appearing in Chichester, York and Wharram Percy.

*Lumbarisation* only appeared in Raunds and York, and in slightly higher proportions. The York figure was particularly noticeable, with 7% of observed juveniles affected.

*Spina bifida occulta* was present in juveniles in all sites except York. Hull had the highest rate, with 8% of observed juveniles affected. Cleft neural arch was the most commonly observed juvenile defect. All sites had affected individuals, with Raunds having the lowest prevalence and Hull the highest among observed juveniles.

*Fused vertebrae* were observed only in Raunds, affecting a single individual.

*Supernumerary vertebrae* were observed at Chichester, Hull and Wharram Percy. Hull had the highest prevalence.

*Absent vertebrae* were observed in all sites, with York having the highest prevalence and Raunds the lowest.

*Spondylolysis* was observed among Chichester and York juveniles, with one individual affected at each site.

#### 6.4.a Juvenile age distribution

Figure 6.17 shows the age-at-death distributions among juveniles at the four sites with compatible age categories.

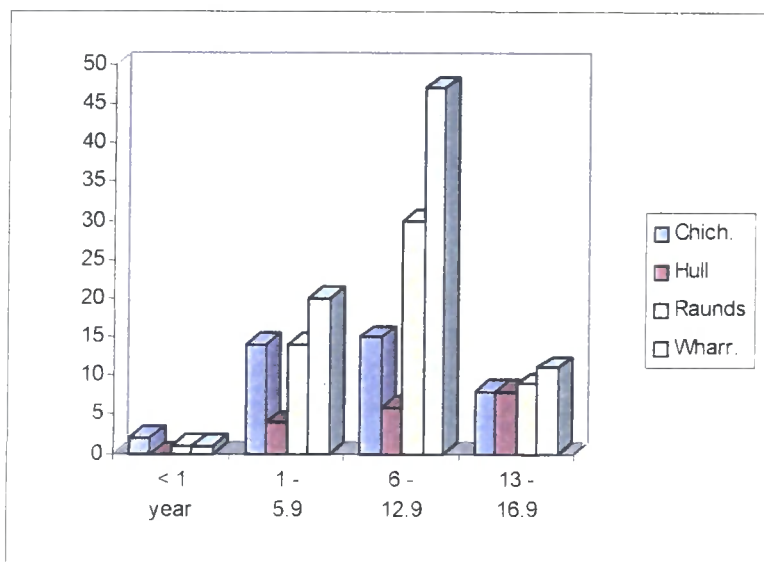


Fig. 6.17: age-at-death distribution for juveniles (percentage of juvenile population)

Figure 6.18 shows the distribution at St Helen-on-the-Walls:

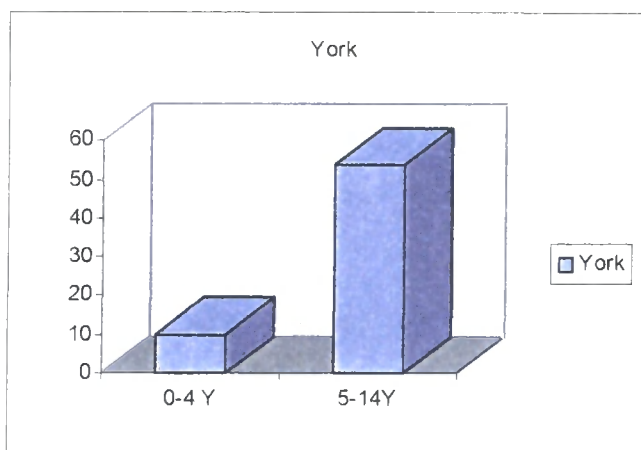


Fig. 6.18: age-at-death distribution for juveniles at York (percentage of juvenile population)

Age assignment among juveniles was taken from the original reports on the populations (Powell, 1996; Lee, 1987; Grauer, 1989; Mays, Isaacs, 1999). Age assignment was checked on all sub-adult individuals as work progressed. No

alterations were made to ages already assigned. Assigned age was generally restricted to categories such as "0-3 months", "1-2 years", "10-12 years" and so on. The exception was the work by Grauer (1989) on the St. Helen-on-the-Walls population, in which the juveniles were divided into two categories: 0-4 years and 5-14 years. As the other populations included sub-adult categories up to the age of 17, adolescents at the York site were particularly closely observed for apparent age.

The Chichester pattern has a similar rate of death among the second and third age groups (age 1 year to 12.9 years) which is not apparent elsewhere. In the Hull cemetery the greatest proportion of sub-adults represented is in the mid-teen category of 13 - 16.9 years. No infants were present at the Hull site. The Raunds profile shows a high risk of juvenile death in the age group 6-12.9 years, with a relatively high risk in the preceding group also. Wharram children appear to have had the greatest risk of death in the 6-12.9 year category also, with rates similar to those at Raunds. The Raunds and Wharram sub-adults show similar age-at-death patterns in a way which is not echoed at the other sites. The York sub-adults, divided into two categories, again show a higher proportion among the older age group, with 83% of juveniles falling into the second category.

### **6.5 Defects and stress indicators (SI)**

Stress indicators were observed in males *and* females at St Helen-on-the-Walls, Wharram Percy and Hull Magistrates Court. These observations were compared with the data taken from *females only* at Chichester and Raunds in the pilot study (Sture, 1997). The distributions are shown in Fig. 6.19 below. Chichester had the highest rates of stress indicators and Wharram the lowest. Hull's high rate is skewed by the

unusually high rate of tibial periostitis observed at the site (see Chapter 7) and may not reflect the underlying trend among that population. The high Raunds rate may indicate a harsher life and/or poorer-quality, perhaps discriminatory, diet in the early Medieval period than that which was enjoyed by the rural peasants of Wharram Percy in the later Medieval period.

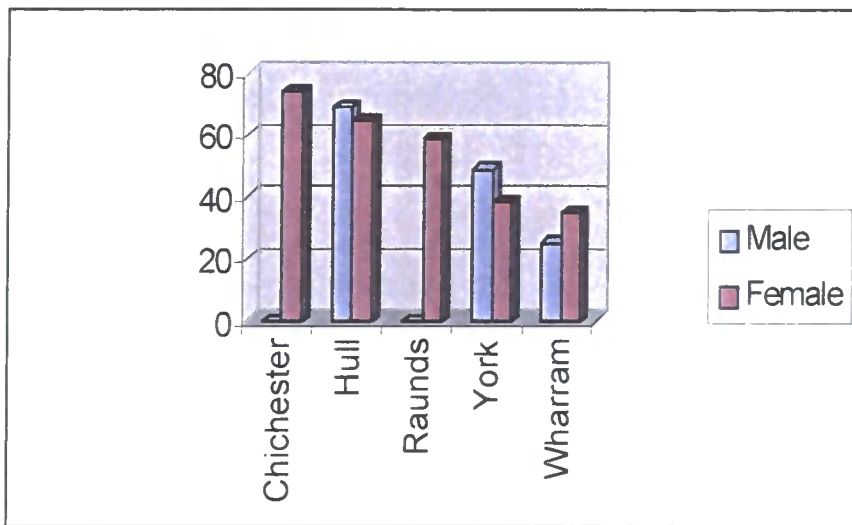


Fig.6.19 Percentage of observed population with at least one stress indicator

The likelihood of co-occurrence of stress indicators with defects among adults varied between the sites. Fig. 6.20 shows this distribution. Table 6. shows the percentage of defect sufferers who also had stress indicators present. The high rate of stress indicators associated with defects observed at Hull is a reflection of the unusually high rate of tibial periostitis observed at the site, which has been discussed elsewhere. At Chichester, although only 37% of the overall population had stress indicators *and* defects, of the population with defects, 81% had at least one stress indicator. This suggests that there may have been other health issues affecting these women, which may have been instrumental in their admission to the hospital/almshouse as residents.

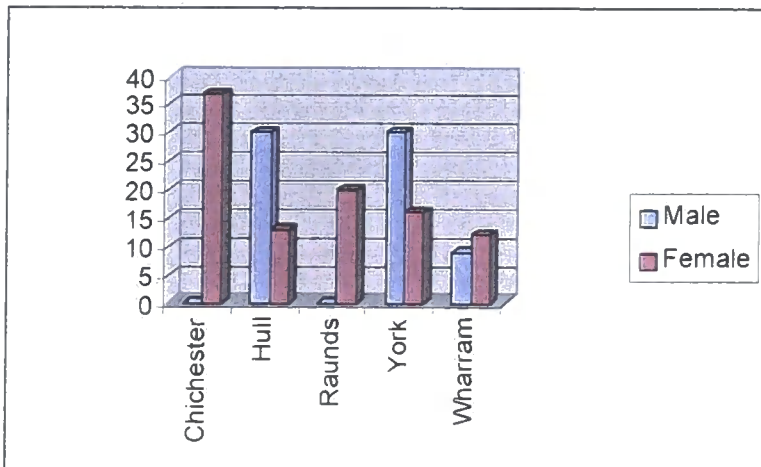


Fig. 6.20 Percentage of total population who have defects **and** stress indicators

Raunds females suffered high rates of stress indicators (59% overall) but 70% of those with defects also had stress indicators. York males with defects appear to have had a greater risk of stress than the York females with defects; 64% of male defect sufferers had stress indicators, and only 42% of female defect sufferers had stress indicators. York males with defects had a greater risk of stress than those without defects, but this was reversed among the York females. This may be due to some culturally-mediated factor such as exposure to occupation-related pollution, or perhaps to injury rates affecting overall male health. Alternatively, it may be simply a reflection of a generalized male sensitivity to environmental stressors in the city. Wharram males had a higher rate of stress than Wharram females (9% : 12% overall), but only 37% of males and 39% of females with defects had stress indicators, which constitutes the lowest proportions of defect sufferers with stress indicators in the study.

Table 6.VII Percentage of defect sufferers who also have at least one stress indicator

Site	Males	Females
Chichester	-	82%
Hull	81%	75%
Raunds	-	70%
St Helen-on-the-Walls	64%	40%
Wharram Percy	36%	39%

### 6.5 Age-at-death patterns among affected individuals

Age-at-death was correlated with specific conditions, sex, and stress indicators.

#### 6.5.a Sacralisation

Figures 6.21 and 6.22 show the evidence for age-at-death among sacralisation cases.

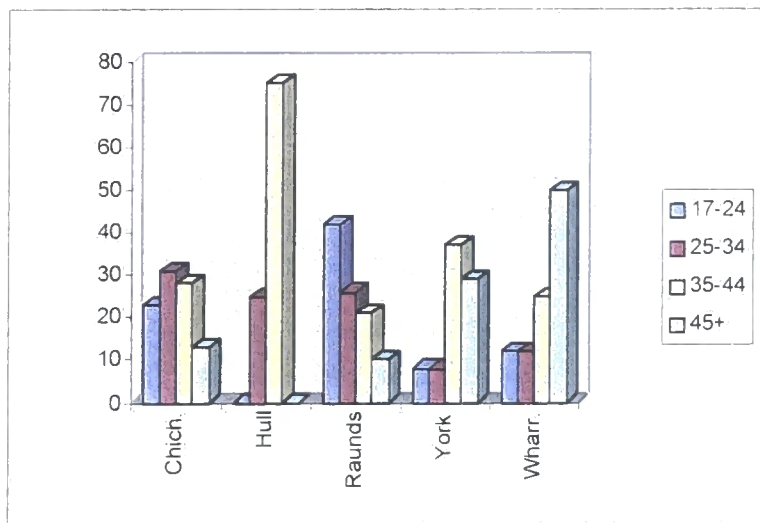


Fig. 6.21 Male sacralisation and age-at-death (% of affected population)

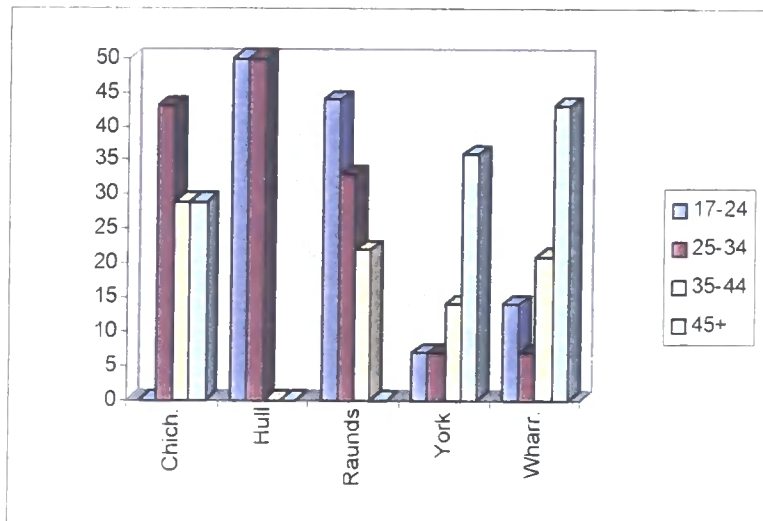


Fig. 6.22: female sacralisation and age-at-death (% of affected population) (n=47)

The age-at-death pattern of affected males at Chichester was similar to that of those without sacralisation. However, the female death rate among those affected was highest in the 25-34.9 year age group (43%), whereas age-at-death was more evenly spread in those without the condition. At Hull, males with sacralisation all died by 44.9 years, but of those without the condition, 15% survived to 45+ years. The female cases both died before the age of 34.9 years, whereas those without sacralisation were spread across all age groups, with 31% living beyond 34.9 years. Affected Raunds males expressed a different pattern of death to that of those unaffected: the age group proportions were reversed. Affected males died in greatest numbers in the 17-14.9 year age group, with proportions declining through to the 45+ group. Unaffected males on the other hand, died in increasing numbers as age increased. Raunds females with sacralisation died in the greatest proportion before the age of 24.9 years (44%), followed by a further 33% before the age of 34.9 years. Among affected York males, the age-at-death pattern was similar to that of the unaffected males. Female sacralisation cases survived in substantial proportions into older age. 36% died at 45+ years of age, with only 7% dying in each of the age groups



17-24.9 and 25-34.9 years. Unaffected females died in greater proportions in early adulthood (before 34 years old) than those with sacralisation, and unaffected females had a high death rate in age group 35-44.9 years (50%) which is mirrored only by the pattern of age group 35-44.9 among affected males. Wharram males with sacralisation appear to have had a similar low death rate in early adulthood to the affected York females. 50% of affected males in Wharram survived to 45+ years of age. Unaffected Wharram males died in substantial numbers in the early years, however, with 21% dying before age 24.9 years. Despite this high young adult death rate, however, 50% of unaffected males still reached 45+ years of age. Wharram females with sacralisation survived in substantial numbers into older age, with 43% dying at 45+ years of age. Unaffected females however, died in greater proportions before 34.9 years (40% as opposed to 21% of affected females).

### 6.5.b Lumbarisation

Figures 6.21 and 6.22 show the evidence for age-at-death among lumbarisation cases.

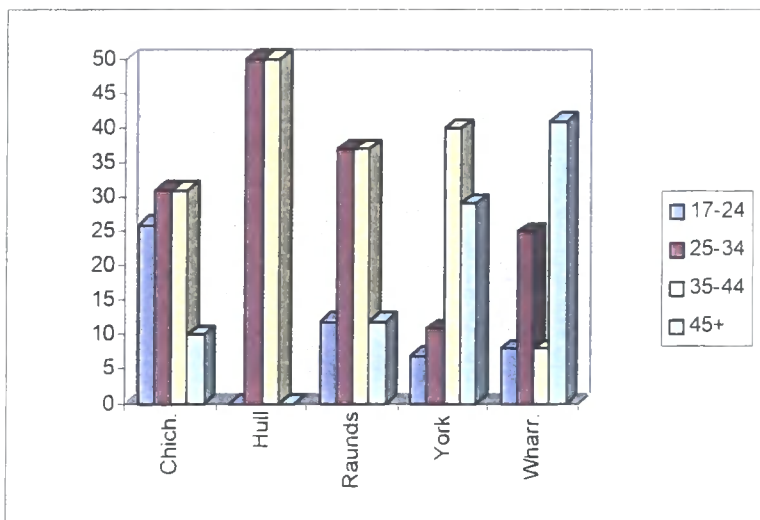


Fig. 6.21: male lumbarisation and age-at-death (n=78)

Lumbarisation does not appear to have significantly affected the death pattern of Chichester males, but there is a slight difference for females. Affected females show a lower death rate in the young adult age group than unaffected females, although the mid- and mature-adult age group rates are similar for affected and unaffected females. At Hull only two males had lumbarisation, and these died in the mid- and mature-adult age groups. Only one Hull female was affected, dying in the 25-34.9 year age group. Affected Raunds males seem to have survived young adulthood in greater proportions than those unaffected. 12% of affected males died before 24.9 years compared to 27% of unaffected males. The proportions were reversed in the age groups 25-34.9 and 35-44.9, however, with 37% of affected males dying in both groups, compared to 20% and 23% respectively, of the unaffected males. Only 12% of affected males survived to 45+ whereas 30% of unaffected males lived to 45+. Only two females in Raunds had lumbarisation, featuring in the young and mid-adult age groups.

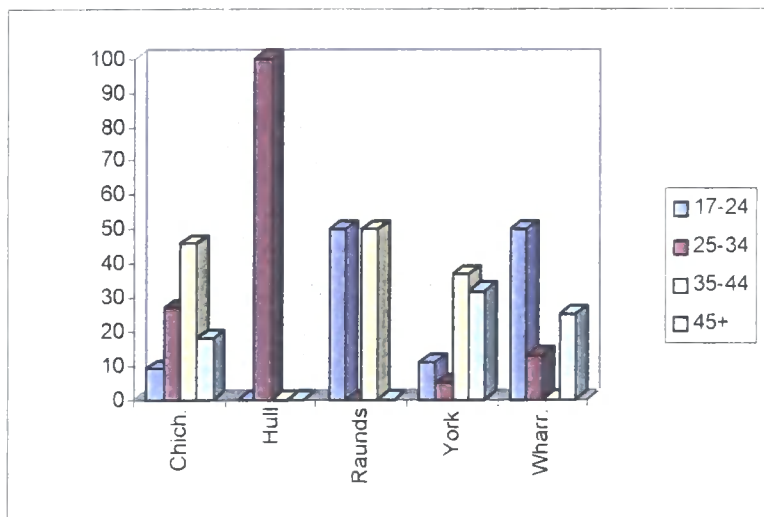


Fig. 6.22: female lumbarisation and age-at-death (n=35)

Affected York males had a lower death rate in the 17-24.9 year group compared to those unaffected, and the patterns of death in the age groups 35-44.9 and 45+ years

were reversed, with the affected males dying predominantly in the mature adult group, whereas unaffected males had a good survival rate into the 45+ group. York females with or without lumbarisation survived in greater proportions into older age than their female counterparts elsewhere. However, 16% of affected women died by age 24.9 years as opposed to 23% of those unaffected. A lower proportion of affected women also died in the 35-44.9 year group, but all women had a similar risk of death in the next age group. 32% of affected women survived into the 45+ group, with only 26% of the unaffected women doing so. Wharram males had similar risk of death in mid- and mature-adulthood, whether they had lumbarisation or not. In the age groups 17-24.9 years and 25-34.9 years, however, the risk of death before 24.9 years was greatest for those without lumbarisation (30% without lumbarisation died in the younger group, and only 8% of those affected). The proportions were reversed in age group 25-34.9 years. Among affected females at Wharram, there was a high risk of death by age 24.9 years.

### 6.5.c *Spina bifida occulta*

Figures 6.23 and 6.24 show the evidence for spina bifida age-at-death rates.

Chichester had the highest number of spina bifida occulta cases (14), of which 9 were male, 4 female and 1 juvenile. Males were represented across all the age groups, although four feature in the mature adult age group. The largest female proportion also features in this group. The age-at-death pattern was similar for affected and unaffected individuals. Hull only had two cases of spina bifida occulta, one male, and one juvenile. The male died after the age of 45 years. Raunds had six cases, two each of males, females and juveniles. Males feature in the young and mid-adult groups, but

both the females died between 17 -24.9 years. York had eleven cases, of which 7 were male. One male died in each of groups 17-24.9 and 25-34.9 years, two in the mature adult group and three in the 45+ group. Among the affected females (4), only three were assigned an age group, one in group 17-24.9 years, one in group 35-44.9 years and one in group 45+.

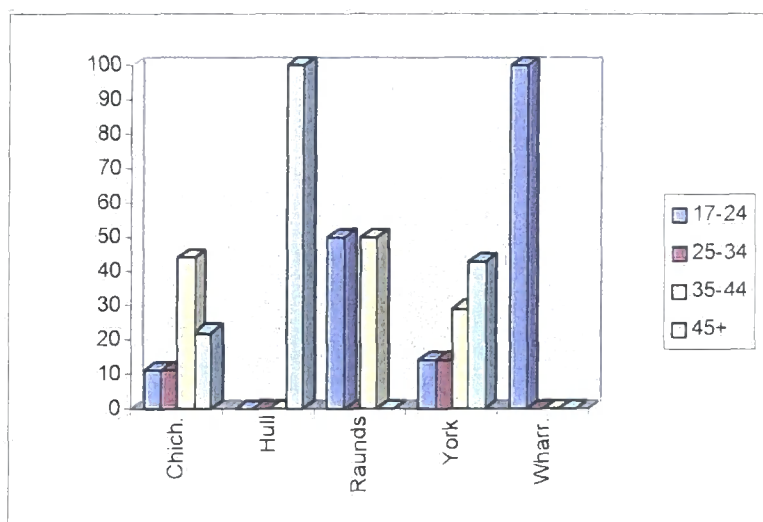


Fig. 6.23: male spina bifida occulta and age-at-death (n=20)

Affected York males were at greater risk of dying before 34.9 years of age than unaffected males (28%:20%), but a greater proportion of affected males survived to 45+ (43%:33%). For York females, comparison of the age-at-death patterns between affected and unaffected individuals is difficult. Wharram had only four cases of spina bifida occulta, comprising one male, one female, and two juveniles. The male features in group 17-24.9 years, the female in the 45+ group.

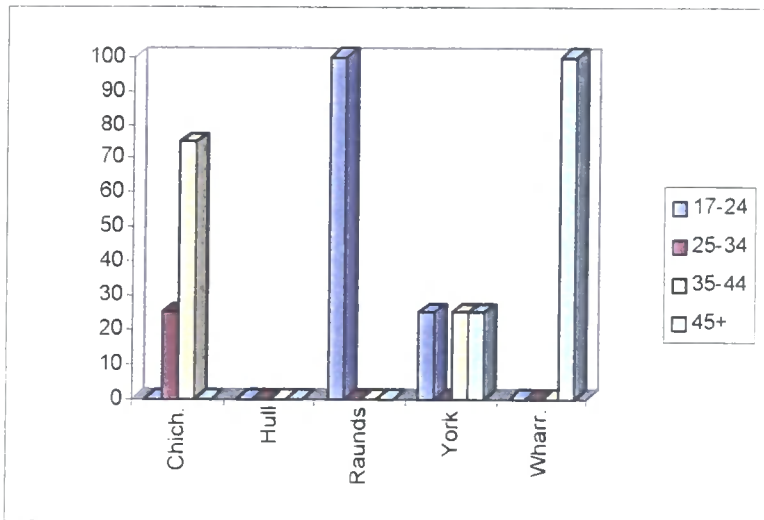


Fig. 6.24: female spina bifida occulta and age-at-death (n=11)

#### 6.5.d *Cleft neural arch*

Figures 6.25 and 6.26 show the evidence for cleft neural arch age-at-death rates. The condition appeared to make no difference to the age-at-death profiles of the Chichester male population, but did make some difference to females. Affected females had a higher risk of dying between the ages of 25 and 34.9 years, with 53% of those affected in the 25-24.9 age group, and only 22% of those unaffected. A similar proportion of affected and unaffected females survived to 45+ years. At Hull, male risk of death appears unaltered by the presence of the condition. Females however, all died before the age of 34.9 years, with three dying between 25 and 34.9 years. This may not be a significant departure from the unaffected female death pattern.

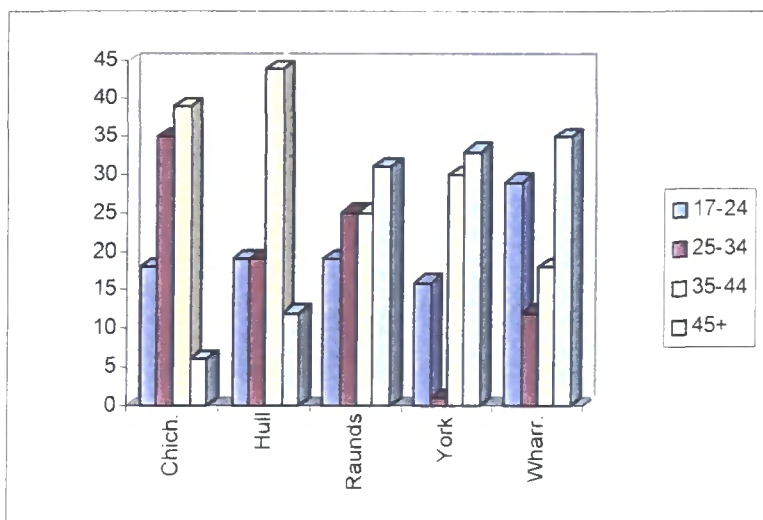


Fig. 6.25: male cleft neural arch and age-at-death (n=127)

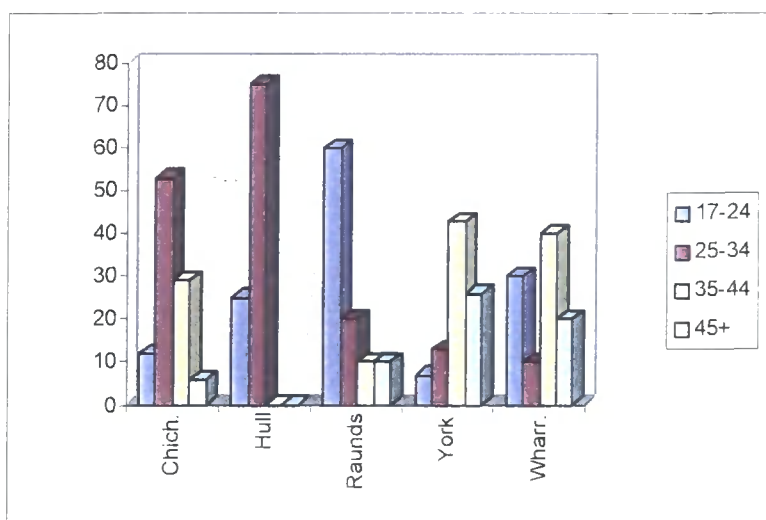


Fig. 6.26: female cleft neural arch and age-at-death (n=68)

As at other sites, male risk of death at Raunds does not appear to be affected by the presence of cleft neural arch. Affected females, however, had a greater risk of dying young, with 60% of affected females in the 17-24.9 year age group, as opposed to only 39% of unaffected females in this group. In York, affected males had a higher death rate between 17 and 24.9 years, with 16% of affected males dying at that age as opposed to only 1% among unaffected males. Those affected males who survived beyond 34.9 years appear to have then enjoyed a similar risk of death at any age to



### 6.5.f *Supernumerary vertebrae*

Figures 6.27 and 6.28 show the evidence for age-at-death of supernumerary vertebrae cases.

At Chichester, affected males died in fewer numbers between 17 and 24.9 years, but in later categories the condition does not appear to have any effect on the male death pattern. Affected females all died before 34.9 years, with one dying before 24.9 years. At Hull, affected males all died between 35 and 44.9 years. At Raunds affected males expressed an opposing pattern of death to that of unaffected males, with the highest death rate in the 17-24.9 year age group (44%), declining through the age groups to only 11% surviving into the 45+ category.

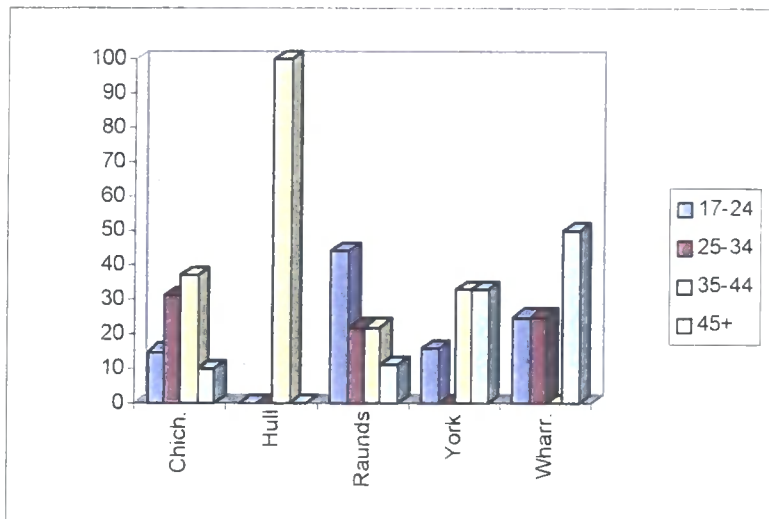


Fig. 6.27: male supernumerary vertebrae and age-at-death (n=43)

Of the unaffected males, only 42% had died before the age of 34.9 years, and 29% survived into the 45+ category. The four affected females all died before the age of 44.9 years, with the highest death rate in the 25-34.9 year age group. In York, affected males appear to have had a greater risk of death between 17 and 24.9 years, but equal



proportions of unaffected males were dead by the age of 34.9 years. None of the affected females died before the age of 25 years. However, 38% of affected females died in the 25-34.9 year age group, giving a higher risk of death than for unaffected females, of whom only 22% were dead by the age of 34.9 years. 38% of affected women survived into the 45+ category, as opposed to 23% of unaffected women.

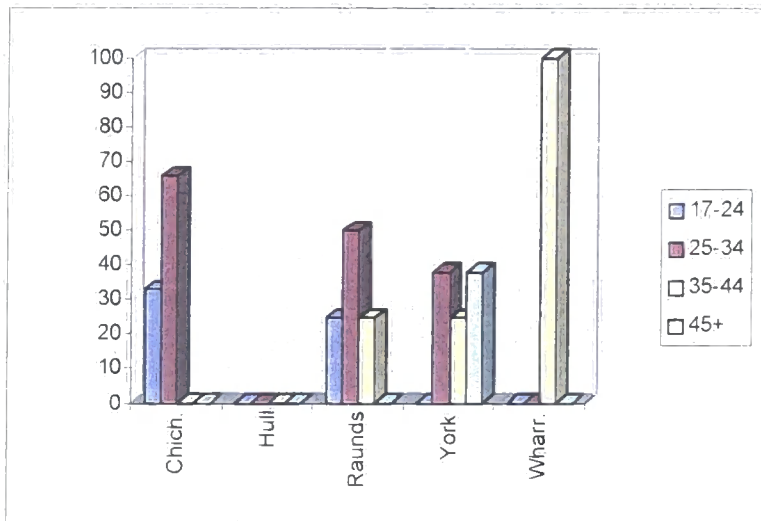


Fig. 6.28: female supernumerary vertebrae (n=17).

At Wharram, half of the affected males died by the age of 34.9 years, but the other half survived to 45+. This is not a dissimilar pattern to that of the unaffected males, of whom 50% survived to 45+ years. The affected female died between 35 and 44.9 years.

### 6.5.g *Absent vertebrae*

Figures 6.29 and 6.30 show the evidence for age-at-death distribution for absent vertebrae cases. At Chichester, affected males were all dead by the age of 44.9 years, with 63% dead by 34.9 years as opposed to 50% of unaffected males. The age-at-death pattern for affected females was similar to that of unaffected females, and the presence of the condition appears to have lead to a greater proportion of affected females surviving into the 45+ category (17%: 7%).

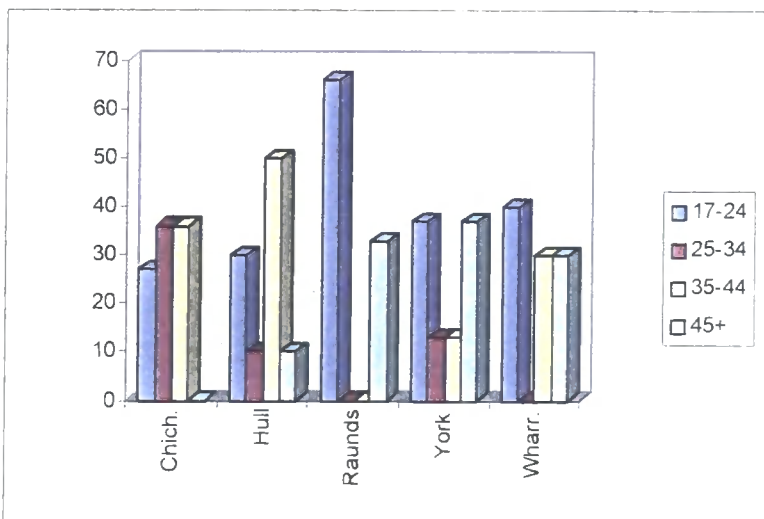


Fig. 6.29: male absent vertebrae and age-at-death (n=41)

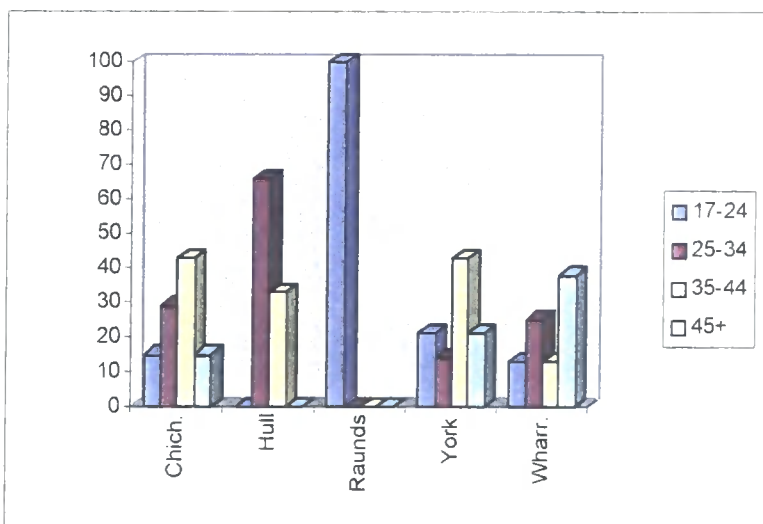


Fig. 6.30: female absent vertebrae and age-at-death (n=30)

At Hull, a higher proportion of affected males died between 17 and 24.9 years than unaffected males (30%:11%) but by the age of 34.9 years the figures are similar. At Raunds, two of the three affected males died between 17 and 24.9 years and one survived into the 45+ group, whereas unaffected males survived in substantial proportions into the older categories. The single affected female died in young adulthood, as was common for females at Raunds. As with supernumerary vertebrae rates, York is the only site with a higher female than male prevalence of the condition. Affected males had a greater chance of dying young than unaffected males, with 50% of those with the condition dying by the age of 34.9 years as opposed to only 15% of those unaffected. However, 37% of those affected survived into the 45+ group as opposed to 29% of the unaffected males. Affected females died in a greater proportion in the 17-24.9 year age group than unaffected females, but other age groups were similar. At Wharram, most affected males died at a younger age than unaffected males, with 70% dead by 44.9 years, and 40% dying between 17 and 24.9 years. 52% of unaffected males survived to 45+ years. Affected females appear to have a similar pattern of death to that of unaffected women. Although only 13% of affected women died between 17 and 24.9 years as opposed to 21% of unaffected women, the number of affected women was small, so the figures may be misleading. It is possible, however, that presence of the defect acted in favour of female survival in early adulthood.

### 6.5.h *Cervical ribs/C7 anomalies*

These cases occurred in small numbers and are presented in Table 6.VI.

Table 6.VI: prevalence of cervical vertebrae/C7 anomalies.

Site	17-24		25-34		35-44		45+		Adult
	M	F	M	F	M	F	M	F	
Chichester	1	0	0	0	0	1	0	0	1
Hull	0	0	0	0	1	0	0	0	1
Raunds	0	1	1	0	0	0	0	0	0
York	0	0	0	0	2	1	1	0	0
Wharram	0	0	0	0	0	0	0	0	0

Males were affected twice as often as females among those skeletons assigned a sex.

The majority died before the age of 44.9 years.

### 6.5.i *Spondylolysis*

Figures 6.33 and 6.34 show the evidence for age-at-death distributions for spondylolysis cases.

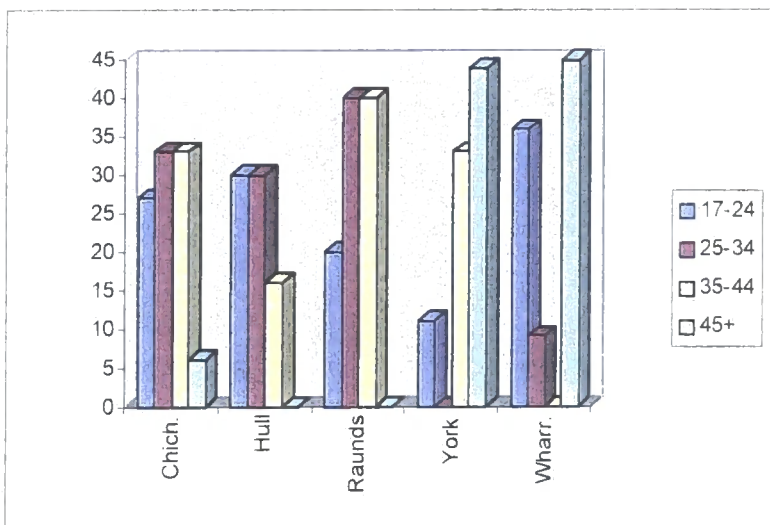


Fig. 6.33: male spondylolysis and age-at-death (n=45)

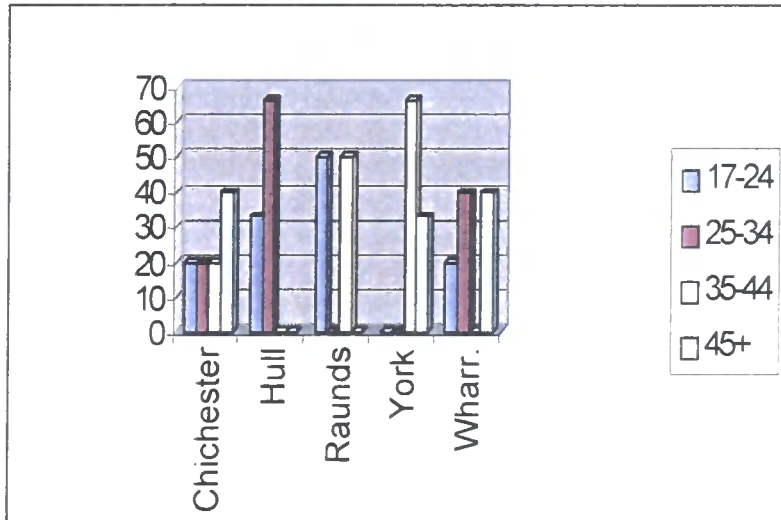


Fig.6.34: female spondylolysis and age-at-death (n=17)

At Chichester, more affected males died in the 17-24.9 year age group than unaffected males (27%:20%) and the age groups 25-34.9 years and 35-44.9 years shared equal prevalences. Only 6% of affected males survived to the 45+ category as opposed to 10% of unaffected males. One female died in each of the four age groups, but unaffected females died in greatest proportions in the 35-34.9 year age group. At Hull, four of the affected males died before 34.9 years and the remaining age-assigned male died before 44.9 years. Affected females died before 34.9 years.

#### 6.5.j *Spondylolisthesis*

Figures 6.35 and 6.36 show the evidence for age-at-death distribution of spondylolisthesis cases.

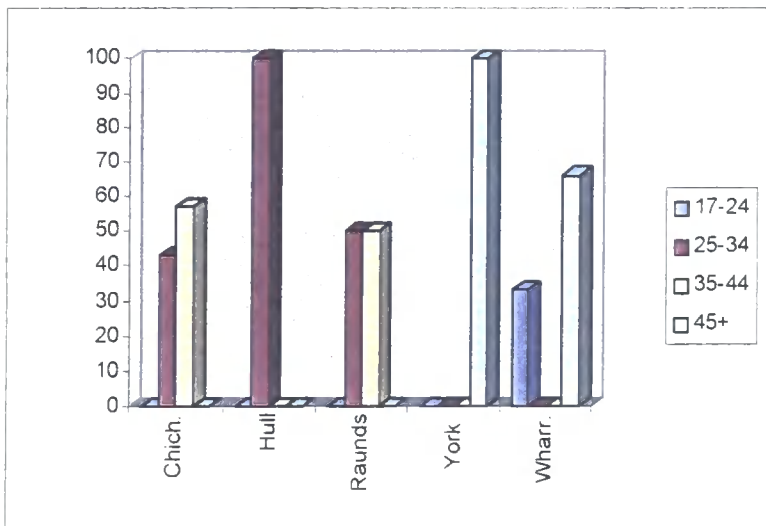


Fig. 6.35: male spondylolisthesis and age-at-death (n=14)

At Chichester, affected males all died in the 25-34.9 year and 35-44.9 year age groups, with a slightly higher proportion in the latter group. The two affected females also died in these age groups. At Hull, all those affected and with an assigned age died in the 25-34.9 year age group. At Raunds, all affected individuals died in the 25-34.9 year and 35-44.9 year age groups. In York, the affected male died in the latter group. At Wharram, one of the affected males died between 17 and 24.9 years but the other survived into old age.

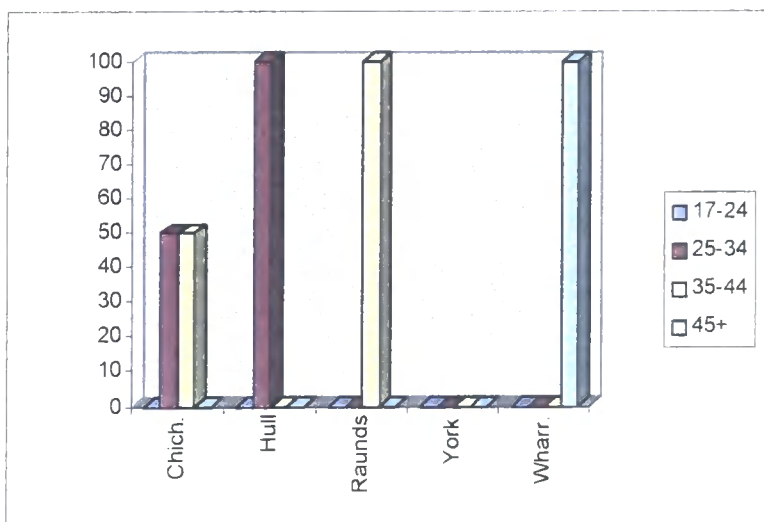


Fig. 6.36: female spondylolisthesis and age-at-death (n=17)

## 6.6 Age-at-death and stress indicators

### 6.6.a *Cribra orbitalia*

Figures 6.37 and 6.38 show the age-at-death distribution of cribra orbitalia cases.

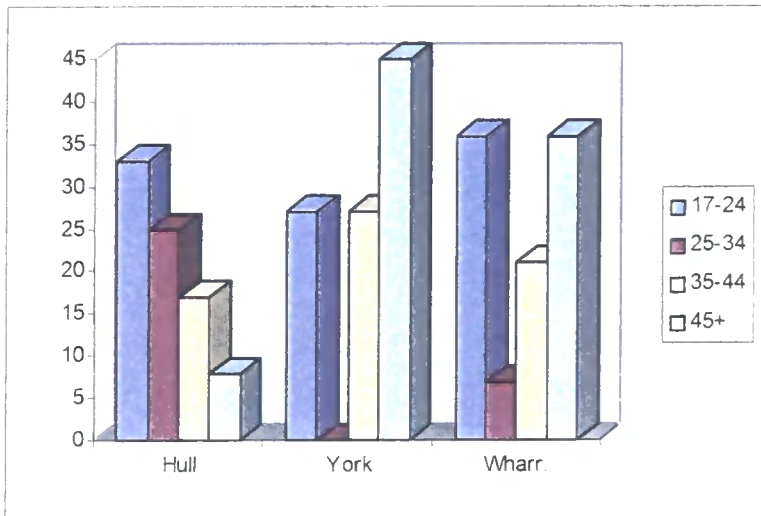


Fig. 6.37: male cribra orbitalia and age-at-death (n=44)

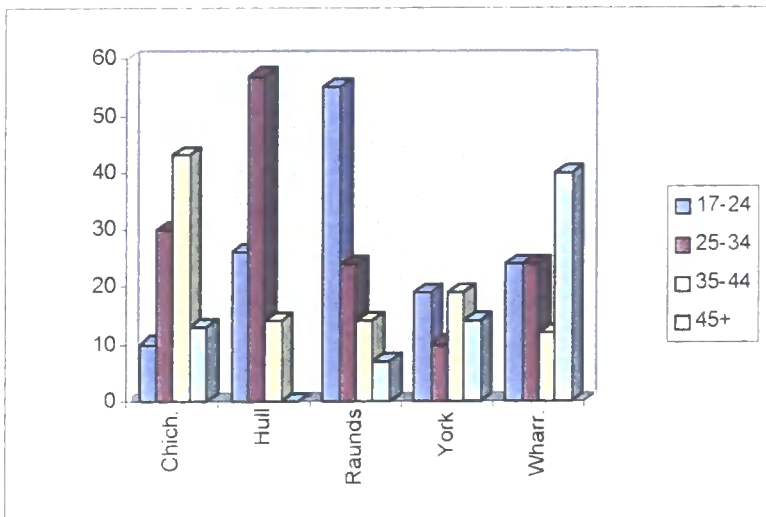


Fig. 6.38: female cribra orbitalia and age-at-death (n=103)

Chichester females with cribra orbitalia appear to have survived early adulthood in greater proportions than unaffected women (10%:17%). Subsequent age group proportions were similar but more affected females survived into old age than

unaffected women. Hull males with cribra orbitalia died in higher proportions in the 17-24.9 year age group than those unaffected (31%:8). Subsequent age groups were similar in proportion. Survival of affected females was significantly influenced by cribra orbitalia, with all affected women dead by the age of 44.9 years, as opposed to 78% unaffected women. The 25-34.9 year age group was particularly heavily represented by affected females, with 57% of women with cribra orbitalia dying in this category. Affected Raunds females also died in greater proportions in age group 1 (55%), and only 7% of affected women survived into the 45+ age group as opposed to 25% of unaffected women. York affected males had similar age-at-death patterns to those of unaffected males. York affected females had a higher risk of death than unaffected women in the 17-24.9 year age group (19%:0%) and in the 25-34.9 year group (10%:6%), resulting in 28% of affected women being dead by the age of 34.9 years. Subsequent age groups were similar for all women, however. Wharram affected males died in greater proportions in the 17-24.9 year age group than unaffected males (36%:8%). 64% of affected males were dead by the age of 44.9 years but only 45% of unaffected males died by that age. Wharram females with cribra orbitalia also died in greater proportions in early adulthood (24%:6%), and only 40% survived into the 45+ category as opposed to 52% of the unaffected females.

#### 6.6.b *Enamel hypoplasias*

Figures 6.39 and 6.40 show the age-at-death distributions for enamel hypoplasia cases.

Chichester females (n = 19) with enamel hypoplasias had a similar age-at-death pattern to that of those women without the defects. Fewer Hull males (n = 21) died in the 17-24.9 year group if they were affected, but other age groups had similar rates for all males. Hull affected females (n = 9) were all dead by the age of 44.9 years, with



56% dying in the 17-24.9 year age group ( $n = 5$ ), whereas unaffected females died in the greatest numbers in the 25-34.9 year age group.

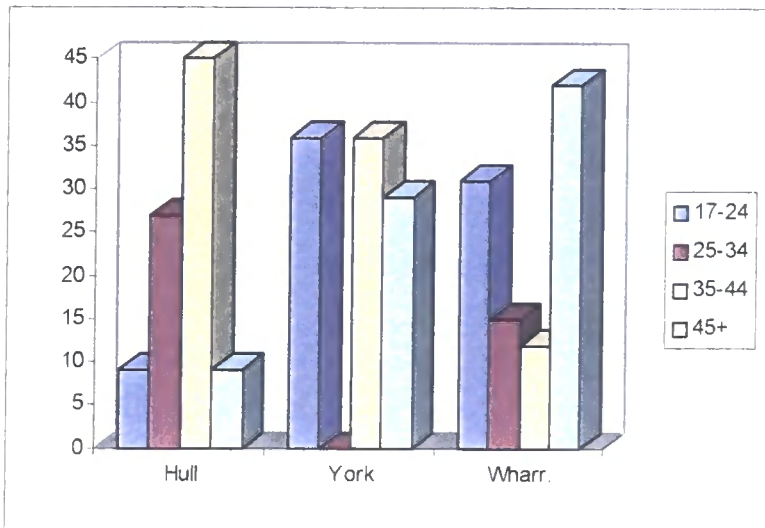


Fig. 6.39: male enamel hypoplasias and age-at-death ( $n=58$ )

Raunds affected females ( $n = 7$ ) also died most frequently in the 17-24.9 year age group, with all other age groups also represented. York affected males ( $n = 14$ ) died in the 17-24.9 year age group more than unaffected males (36%:5%), but no affected males died in the 25-34.9 year age group. York females with enamel hypoplasias had a better chance of surviving early adulthood than those without (15%:23%), but the risks evened out in the subsequent age groups. Wharram affected males died in the 17-24.9 year age group more often than unaffected males, but in all other age groups, affected males died in lesser proportions. 42% of affected males survived into the 45+ age group but only 29% of unaffected males did so. A lesser proportion of affected females at Wharram died in the 17-24.9 year age group, but this was compensated for by the higher death rate in the 25-34.9 year age group, so that the risk of death by the age of 34.9 years was approximately even for all women. More unaffected females survived into the 45+ age group than affected women (33%:24%).

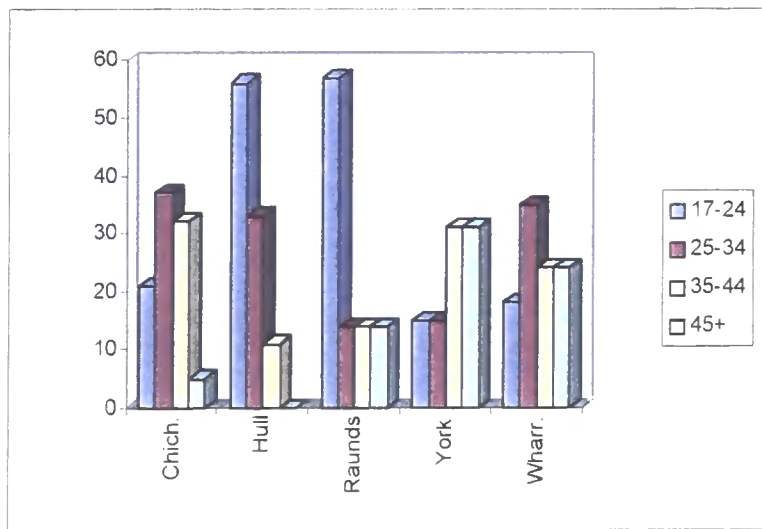


Fig. 6.40: female enamel hypoplasias and age-at-death (n=65)

### 6.6.c *Tibial periostitis*

Figures 6.41 and 6.42 show the age-at-death distributions for tibial periostitis cases. Chichester females with tibial periostitis (n = 25) died in greater proportions in the 25-34.9 year age group rather than in the 35-44.9 year age group, which is the case with unaffected females. None of the affected females survived beyond 44.9 years. Hull males with tibial periostitis (n = 69) survived into older age in greater numbers, with the majority dying in the 35-44.9 year age group and 12% surviving into the 45+ age group. Affected females (n = 26) had a lower risk of death in the 17-24.9 year and 25-34.9 year age groups than those unaffected, with 43% of affected women dead by 34.9 years as opposed to 66% of unaffected women. However, similar numbers of affected and unaffected females survived into the 45+ age group. The picture of tibial periostitis at Hull is unusual, with 81% of the observed population (male and female) being affected by the condition, which is a considerably higher prevalence than is seen elsewhere. Raunds females died in even greater numbers in the 17-24.9 year age group if affected by tibial periostitis (n = 15) compared to those without the condition

(60%:45%). Survival through subsequent age groups was also affected, but a greater proportion of affected survived into the 45+ age group than those unaffected.

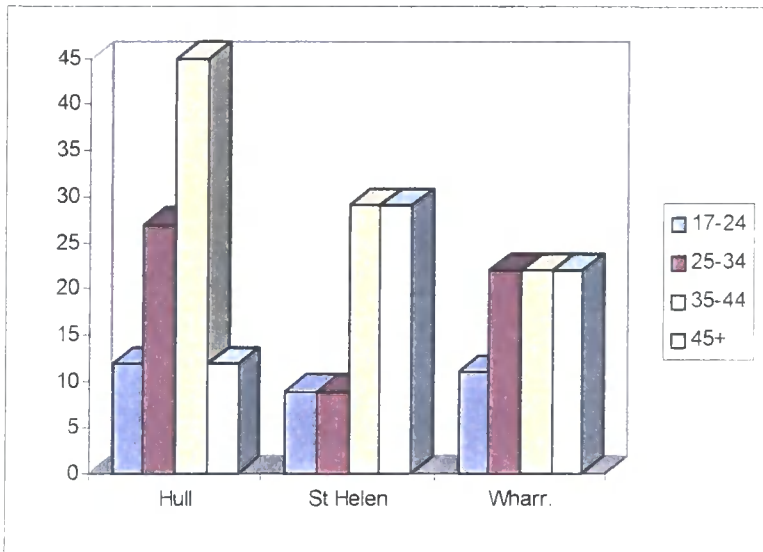


Fig. 6.41: male tibial periostitis and age-at-death (n=124)

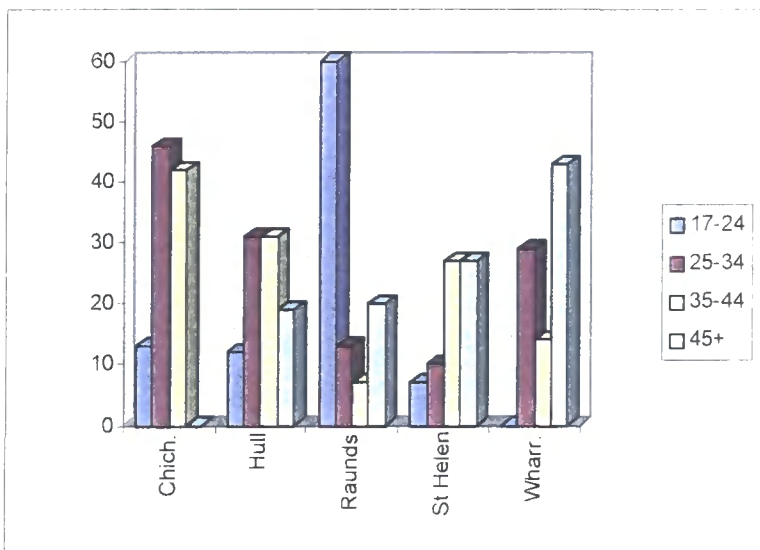


Fig. 6.42: female tibial periostitis and age-at-death (n=103)

York males with tibial periostitis (n = 56) did not seem significantly affected by the condition in terms of changes to the age-at-death pattern. Affected York females (n = 30) however, tended to survive the early years of adulthood better, with 17% of those

affected and 36% of those unaffected dead by the age of 34.9 years. Affected women died in equal proportions in the 35-44.9 year and 45+ age groups whereas unaffected women survived well into the 45+ age group (27%:50%). Wharram males with tibial periostitis (n = 9) tended to die in greater proportions throughout the 25-34.9 year and 35-44.9 year age groups rather than surviving into old age. The 17-24.9 year age group had a similar risk of death for all males, but of the affected men, only 22% survived to the 45+ age group as opposed to 47% of those unaffected. Affected Wharram females (n = 7) were not represented in the 17-24.9 year age group, but 29% of those unaffected were. By the age of 34.9 years the proportions of affected and unaffected women had changed, however, with 38% of those unaffected dead compared to only 29% of those with periostitis. Similar substantial proportions survived into the 45+ age group (43%:52%).

## **6.6 Stature And Conditions**

Stature was observed where possible, using available long bones. Data for Wharram Percy, Raunds, Hull and Chichester were taken from other workers (Grauer, 1989; Mays, unpublished; Powell, 1996; Ward, 1996). York statures were calculated according to the method outlined by Trotter (1970). Average stature was calculated for male and female populations at each site, and again for those males and females with or without defects. The results are laid out in Tables 6.XI - 6.XV. Mean population statures are laid out in Table 6.X.

Table 6.X: mean population stature in centimetres.

Site	Male	Female	N =
Chichester	169.8	159.8	215
Hull Magistrates' Court	169.3	158.5	89
Raunds Furnells	168.6	159.7	103
St Helen-on-the-Walls	166.4	157.6	158
Wharram Percy	168.8	159.5	251

Chichester had the greatest male and female mean statures, and St. Helen-on-the-Walls the lowest. In terms of stature and skeletal anomalies, males and females expressed varying responses to the presence or absence of most defects. The tables set out below show the average stature at each site related to those with/without specific conditions. The following abbreviations are used:

SBO = spina bifida occulta  
 CA = cleft neural arch  
 SV = supernumerary vertebrae  
 AV = absent vertebrae  
 CO = cribra orbitalia  
 EH = enamel hypoplasias  
 TP = tibial periostitis

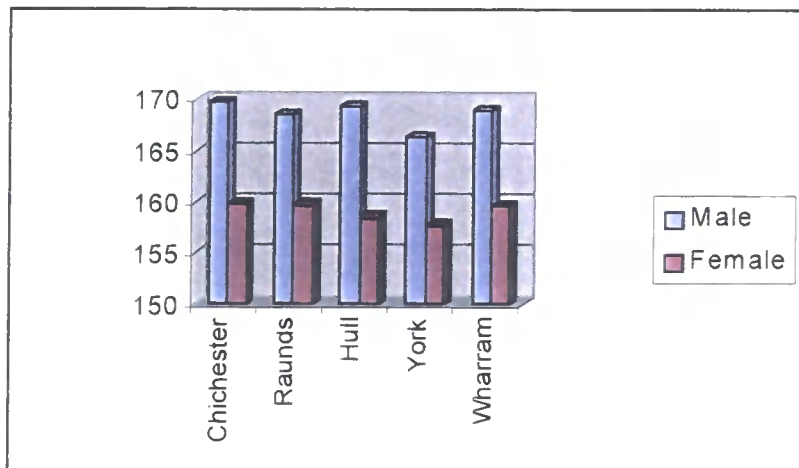


Fig. 6.43: mean stature (cms.) by population and sex (n=816)

Chichester	Males with defect	Males without defect	Difference (cms.)	Females with defect	Females without defect	Difference (cms.)
sacral.	165.9	169.6	-3.7	157.1	154.5	2.6
lumbar.	170.2	171.3	-1.1	160.3	156.4	3.9
SBO	172.5	168.2	4.3	159.2	154.4	4.8
CA	164.9	171.7	-6.8	161.1	151.4	9.7
SV	170.4	169.8	0.6	160.3	155.2	5.1
AV	170.5	169.8	0.7	162.8	159.6	3.2
spondylo.	169.1	172.9	-3.8	161.7	162.7	-1.0
olisthesis	167.6	170.0	-2.4	162.2	162.7	-0.5
CO	-	-	-	171.9	164.1	7.8
EH	-	-	-	158.7	159.2	-0.5
TP	-	-	-	160.9	159.7	1.2

Table 6.XI: mean stature, Chichester, with/without defects (n=215)

Hull Mag. Court	Males with defect	Males without defect	Difference (cms.)	Females with defect	Females without defect	Difference (cms.)
sacral.	167.3	169.8	-2.5	162.0	158.3	3.7
lumbar.	171.0	168.7	2.3	158.6	158.6	0
SBO	-	-	-	-	-	-
CA	166.2	170.1	-3.9	156.0	159.0	-3.0
SV	169.5	169.4	0.1	-	-	-
AV	170.4	169.3	1.1	155.4	158.2	-2.8
spondylo.	167.5	169.7	-2.2	162.0	157.7	-4.3
olisthesis	158.1	169.5	-11.4	158.6	158.1	0.5
CO	168.1	163.3	4.8	160.7	157.5	3.2
EH	166.3	164.2	2.1	160.5	158.6	1.9
TP	168.6	171.7	-3.1	158.1	159.5	-1.4

Table 6.XII: mean stature, Hull, with/without defects (n=89)

Raunds Furnells	Males with defect	Males without defect	Difference (cms.)	Females with defect	Females without defect	Difference (cms.)
sacral.	167.7	168.7	-1.0	162.9	159.8	3.1
lumbar.	169.3	161.0	8.3	169.8	156.6	13.2
SBO	169.9	168.3	1.6	-	-	-
CA	167.2	168.9	-1.7	165.3	157.9	7.4
SV	170.2	165.1	5.1	166.1	158.9	7.2
AV	171.5	168.3	3.2	-	-	-
spondylo.	158.9	168.9	-10.0	159.2	160.6	-1.4
olisthesis	168.2	168.4	-0.2	157.7	160.1	-2.4
CO	-	-	-	161.3	158.3	3.0
EH	-	-	-	162.6	158.8	3.8
TP	-	-	-	160.1	159.1	1.0

Table 6.XIII: mean stature, Raunds Furnells, with/without defects (n=103)

York	Males with defect	Males without defect	Difference (cms.)	Females with defect	Females without defect	Difference (cms.)
sacral.	167.9	168.3	-0.4	159.9	157.2	2.7
lumbar.	168.9	168.1	0.8	158.4	157.8	0.6
SBO	167.7	167.9	-0.2	159.1	157.1	2.0
CA	168.8	157.3	11.5	158.5	157.5	1.0
SV	168.5	157.2	11.3	162.4	157.2	5.2
AV	168.1	165.6	2.5	157.8	157.8	0
spondylo.	168.4	162.8	5.6	156.9	157.5	-0.6
olisthesis	166.1	168.5	-2.4	-	-	-
CO	167.5	168.4	-0.9	158.3	150.7	7.6
EH	152.7	168.1	-15.4	157.3	158.1	-0.8
TP	169.6	168.3	1.3	157.1	157.9	-0.8

Table 6.XIV: mean stature, York, with/without defects (n=158)

Wharram Percy	Males with defect	Males without defect	Difference (cms.)	Females with defect	Females without defect	Difference (cms.)
sacral.	170.2	168.2	2.0	158.2	160.7	-2.5
lumbar.	169.6	168.6	1.0	155.3	157.9	-2.6
SBO	-	-	-	-	-	-
CA	169.9	168.5	1.4	157.5	160.2	-2.7
SV	170.5	168.7	2.2	154.1	157.8	-3.7
AV	167.9	168.9	-1.0	153.1	158.1	-5.0
spondylo.	167.1	168.8	-1.7	160.2	156.9	3.3
olisthesis	164.6	168.6	-4.0	162.2	157.3	4.9
CO	165.8	168.2	-2.4	158.3	157.9	0.4
EH	169.5	175.1	-5.6	157.2	157.8	-0.6
TP	172.2	166.9	5.3	158.4	156.6	1.8

Table 6.XV: mean stature, Wharram Percy, with/without defects (n=251)



*Sacralisation:* The mean stature of affected males and females exhibited opposing patterns. The mean stature of affected males was smaller than that of unaffected males at all sites except Wharram Percy, where the affected male mean height was greater. The mean stature of affected females was greater than that of the unaffected females at all sites except Wharram Percy, where the mean height of affected women was lower.

*Lumbarisation:* Affected males and females had greater mean stature than that of unaffected people on the whole. The exceptions were the Chichester males and the Wharram Percy females, who had lower mean statures than their unaffected counterparts. At Hull, the mean statures of affected and unaffected people were the same.

*Spina bifida occulta:* Affected males had a greater mean stature than that of the unaffected males at Chichester and Raunds Furnells, but a lesser mean stature at York. Affected females also had a greater mean stature than unaffected women at Chichester and York.

*Cleft neural arch:* Affected males had a lower mean stature than unaffected males at Chichester, Raunds Furnells and Hull. At York and Wharram Percy, affected males

had a greater mean stature than their unaffected counterparts. Affected females exhibited the opposite pattern, with smaller mean heights at Wharram Percy and Hull, and greater mean heights at Chichester, Raunds Furnells and York.

*Supernumerary vertebrae:* Affected males and females with an extra vertebral element at all sites had a greater mean stature than those without. The exception was found at Wharram Percy where the affected female mean is less than that of the unaffected females, but this figure is based on only one individual.

*Absent vertebrae:* Affected males at all sites except Wharram Percy had a *greater* mean height than those with a normal complement of vertebral elements. Affected females varied in mean height, with those at Hull and Wharram Percy having a lower mean height than the unaffected women, but those at Chichester having a greater mean height. York females with or without an absent vertebra had the same mean stature.

*Spondylolysis:* Affected males at all sites except York had a lower mean height than that of unaffected males. Affected females had the same pattern, with a lesser mean stature at all sites except Wharram Percy. The affected York males and Wharram females had greater mean statures than their unaffected counterparts.

*Spondylolisthesis:* Affected males had a lower mean height than that of unaffected males at all sites. Affected females had a lower mean height than unaffected women at Chichester and Raunds and a greater mean height at Hull and Wharram Percy.

*Cribriform orbitalia:* Affected males had lower mean stature than unaffected males at York and Wharram Percy, but a greater mean height at Hull. Affected females had a greater mean height than unaffected women at all sites.

*Enamel hypoplasias:* Affected males had a lower mean stature at York and Wharram Percy than their unaffected counterparts, but a greater mean height at Hull. Affected females had a lower mean stature than unaffected females at Chichester, York and Wharram Percy, but a greater mean stature at Hull and Raunds Furnells.

*Tibial periostitis:* Affected males had a greater mean stature than that of unaffected males at York and Wharram Percy, but a lesser mean stature at Hull. Affected females had a greater mean stature than unaffected females at Chichester, Raunds Furnells, and Wharram Percy, but a lower mean stature at Hull and York.

## CHAPTER SEVEN

### Discussion

#### 7.0 URBAN-RURAL DEFECT FREQUENCY PATTERNS

Prevalence of axial skeleton anomalies has varied considerably along urban-rural lines as anticipated, but the pattern of some conditions has been unexpected.

The urban sites of St Helen-on-the-Walls, York, and St. James and St Mary Magdalene, Chichester, had the highest defect prevalence overall, in accordance with the hypothesis of the study. The rural sites of Raunds and Wharram Percy shared similar defect rates, although the Ruands females, had higher rates of stress indicators (with or without defects also), which may be indicative of a poorer diet, either in the whole population, or for females within it.

##### 7.0.i Hull – an urban site with a rural defect frequency pattern

The apparently rural pattern of defects observed among the population of the Hull cemetery appears, however, to contradict the hypothesis of this study. The pattern varies from that observed at the Blackfriar's Friary in Suffolk (Mays, 1991) in which higher rates of congenitally-fused vertebrae were observed. Whilst not intending to interpret too freely, the "rural" effect at Hull may be due to some socio-cultural factor(s) affecting the selection of burials there. For example, the observed pattern may be due to the effect of social status. Perhaps the majority of those buried in the cemetery were wealthy benefactors and friars rather than the urban poor or sick, although such individuals may be more likely found in the nave or aisles of the church itself (Foreman, 1998),

but such identification is currently impossible. For example, the remains of four adult males were excavated from the cloister walks and the south range of Jedburgh Friary, along with a number of disarticulated bones representing an unknown number of individuals from other disturbed areas of the friary (Dixon *et al.*, 2000). Whether the four relatively complete skeletons belonged to the monastic community or not is unclear, but two of the four had significant health problems (Dupuytren's contracture in the hand of Skeleton 1, a male of 25-30 years, and a chronic abscess in the tibia of Skeleton 2, a male of 25-35 years), both of which would have caused physical difficulties for the sufferers (Dixon *et al.*, 2000:56). Burial grounds in monastic cemeteries have traditionally been believed to contain (as well as the monastic community) considerable numbers of the sick who had died in the associated hospital, and therefore it had been expected that the Hull pattern would echo that of the Chichester sample to some degree. Friaries, were, however, popular burial places, and often attracted people from outside the monastic community for burial (Foreman, 1998:47). Keeping (2000) found evidence that suggests better health (and therefore probably higher social status) burials in a monastic context when she found that those buried in Medieval nunnery cemeteries had an apparently better quality of life than those examined in the same study who had been buried elsewhere. The food quality and quantity available to richer individuals would have been better, and they would have enjoyed a better standard of living than the poor of the same period (Dyer, 1994), thus impacting on their health-status. Obviously, assignment of social status is beyond the scope of this study, but it may be a partial explanation of the results seen at Hull. Of course, many of those buried in the

monastic cemetery, whether benefactors or other relatively wealthy individuals who had bought an association with the foundation, may have actually originated from, and lived in, rural areas, and sought burial in the urban setting for religious reasons, such as a desire to be buried close to an important religious building, or to benefit from chantry masses for their soul (Foreman, 1998). The evidence of disease at the Hull site, including the high prevalence of tibial periostitis, may therefore illustrate a disease pattern associated with affluence in the area or among those associated with the friary, but this will no doubt be subject to further discussion elsewhere. Periostitis was recorded on numerous disarticulated bones from a pit at Merton Priory (Waldron, 1983), with one possible case of treponemal disease, which is a possibility at Hull. Alternatively, the “non-urban” patterns observed at Hull may be a true picture of Hull’s urban dwellers, which has been influenced by some unknown factor(s) in the environment. A range of congenital dental anomalies, sacro-lumbar border-shifting and a cervical abnormality (C7) were observed also at Chelmsford Dominican Priory (Bayley, 1990) and a considerable number of cervical anomalies were noted at Merton Priory (Waldron, 1983). The pattern emerging from other friary sites seems to be related to cervical fusions – perhaps those suffering from the debilitating neurological effects of these were typically cared for at friary foundations? If this was the case, then Hull did not mirror this pattern.

### **7.1 URBAN-RURAL PREVALENCE ASSESSMENT**

The discussion below focuses on the observed conditions in turn, commenting on the developmental field involved in the origin of the defect, and on any

associations with soft-tissue anomalies that have become apparent during research into the medical literature.

#### **7.1.a. Border shifting**

Both lumbarisation and sacralisation showed distinct patterns between the sites observed in this study. Barnes states that lumbarisation is more common in humans than sacralisation, and that the latter is more common in females (1994:80), but the results of this study appear to contradict this finding. Sacralisation was more common than lumbarisation amongst the samples observed here. Significant differences were noted between lumbarisation prevalence at urban and rural sites, and between some individual sites, but for sacralisation, significant differences were only noted between individual sites. Why lumbarisation, and not sacralisation also, should be apparently influenced by environmental factors to such a degree is uncertain. Both defects originate in the same developmental field (see below) but the apparent differences in prevalence seem to beg some explanation related to environment. It may be that some environmental variable has an effect on the expression of the homeobox genes which are known to control the development of the lumbosacral border (Weiss, 1993). Also contrary to Barnes' statement, males were more frequently affected by sacralisation than females in the populations observed here. The higher female:male ratio of sacralisation seen at Wharram Percy is unique among the sites, making it the only population to support Barnes' statement about higher female cranial shifting tendencies. Overall, males were more frequently affected by all border shifting than females.

Sacralisation involves a reduction in mobility at the lumbo-sacral junction, and is also associated with increased risk of intervertebral disc degeneration and herniation in the adjoining elements, and with sciatica and other nerve root problems (Castellvi *et al.*, 1984; Cialliet, 1995; Jonsson *et al.*, 1989). All noted significant associations with moderate to severe back pain as a result of the sacralisation/lumbarisation in their patients. It is to be expected, therefore, that similar associations would have been common among the Medieval populations, and this is particularly supported by the high prevalence of sacralisation at Chichester, the hospital/almshouse. It would be interesting to study Medieval hospital populations in other cities and to compare their defect rates.

Lumbarisation is associated with increased mobility at the lumbo-sacral junction, and may be beneficial to those involved with heavy work as there are fewer adverse forces exerted on the surrounding elements and tissues (pers. comm. K. Brewster). The high sacralisation rate seen in Chichester males (29%) may well be related to admission to the Chichester hospital/almshouse as this defect is closely associated with low back pain syndrome today. In addition, 77% of those with sacralisation at Chichester also had other spinal anomalies (the average across all the populations was 69%), which was the highest rate of co-occurrence with sacralisation of all the sites. As co-occurrence of soft-tissue anomalies is associated with skeletal defects (Warkany, 1971), particularly multiple skeletal defects, such occurrence equates well with a hospital population. Some affected individuals may well have been unable to support themselves by work and thus been reliant on help. Raunds and York males also



showed similarly high rates, but did not represent a separate section of the population which may have been deemed poor or sick - even so, the co-occurrence rates were 75% and 76% of those with border shifting, respectively. The York population was taken from one of the poorest parishes, however, so there may be a socio-economic parallel. Co-occurrence in affected males at Hull and Wharram was only 50% and 45% respectively, representing a highly significant difference between the co-occurrence rates in affected individuals between Hull and Wharram, and Raunds, York and Chichester. It is interesting that the Hull rate equates more to the Wharram profile: the Raunds rate may simply represent an early Medieval picture of a population with a restricted gene pool, leaving the York and Chichester groups to offer an urban picture of co-occurrence. If this is the case, then once again the Hull site has shown a rural pattern of defect prevalence. Of course, one could argue that the Wharram population may also have a restricted gene pool, but the movement of people during the later Medieval period was arguably able to mix genetic material to a greater extent than was seen earlier (Razi, 1980).

Lumbarisation generally showed a higher rate of co-occurrence with other defects than did sacralisation. This suggests that there may be different aetiological mechanisms at work in the expression of sacralisation and lumbarisation, and that the two forms of border shift may not be simply two variations of the same defect. In addition, as the majority of sacralisation and lumbarisation cases in the study also had other spinal anomalies, it is highly likely that other health complications would also have been present. The high

prevalence of border shifting and associated anomalies observed at Chichester supports this.

#### 7.1.a.i *Embryological origins of border shifting defects*

Lumbo-sacral segmentation errors can be traced to the blastemal stage in the paraxial mesoderm developmental field, during which the sclerotomes divide and take on segmental characteristics (see fig. 3.3). The presence of border shifting indicates some delay in the normal division, recombination and differentiation of the embryonic vertebral bodies, which may well be caused by some unidentified environmental factor. As there are significant differences in segmentation error prevalence between specific urban and rural populations, it may be supposed that some environmental agent(s) is at work here, disturbing the normal segmentation and differentiation of the sclerotome cells. Why there should be distinctions between the prevalence of lumbarisation and of sacralisation when both anomalies originate in the same developmental field is unclear. It may well be due to some unidentified environmentally-mediated effect acting on the homeobox genes which are known to affect vertebral segmentation (see section on genetic influences below).

#### 7.1.b *Spina bifida occulta*

The trend within palaeopathology to describe any clefting of the neural arch, especially in the lumbo-sacral region, as *spina bifida occulta* is confusing. Barnes rightly pointed out in 1994 that this practice should stop, and that the term should be reserved for those individuals who appear to have neural tube

involvement. The author has conformed to this diagnosis and thus diagnosed all non-flaring clefts as cleft neural arch. As a result, the rates for neural clefting in this study are high. What is described as cleft neural arch in this study would usually be diagnosed as *spina bifida occulta* in previous studies.

*Spina bifida occulta* (cleft neural arch with probable neural involvement)

prevalence was very low. The observed frequencies fall well below modern rates which run at about 10% of the modern population, although that figure refers to all modern lumbo-sacral clefting which is considered asymptomatic. Even so, the highest rate was seen at Chichester, which could equate with a hospital/almshouse population, and York had the next highest rate. Whether the cases diagnosed here were truly asymptomatic is unknown, but the high rate at Chichester may suggest that the condition is either symptomatic in itself, or is associated with some other health issue which brings people into the hospital/almshouse. Rural rates were very low at Raunds and Wharram. The condition was not observed at Hull. Among modern populations, Laurence noted comparable frequencies of neural tube defects in both urban and rural settings in Wales (Laurence, 1966), suggesting a complex interaction of genetic and environmental factors. Jorde *et al.* (1983) found that females were more commonly affected than males with neural tube defects in modern populations, but the figures obtained in this study show that more males were affected in these Medieval populations. This may, of course, be due to cultural preferential treatment of males during infancy and childhood, enabling greater numbers of affected boys to survive infancy. If the condition observed in this study was

associated with other health anomalies, then affected females may have suffered as a result of cultural sex discrimination, receiving less care, or food, or both, than a similarly-affected boy, leading to an early death. The observed high rate of association with other spinal defects (60%) also suggests the presence of other health complications.

The apparent lack of *spina bifida occulta* in these archaeological populations may be due to either inaccurate diagnosis, or it may reflect the true picture of Medieval prevalence. If this is the case, then rates have risen considerably since the Medieval period. Another explanation may be that affected individuals, perhaps with other visible defects, were subject to direct or indirect infanticide (see below).

#### 7.1.b.i *Embryological origins of spina bifida occulta*

True spina bifida occulta is a mild expression of a neural tube defect, and originates during the blastemal embryonic period. Closure of the neural tube is associated with the development of its vascular circulation at the end of the fourth week of gestation, and any delay or fault in this development may result in the tube failing to close. The condition in the *cystica* form is known to be related to folate deficiency. Whether this also relates to the *occulta* form with neural involvement is uncertain. Although some likely cases of *spina bifida cystica* have been observed by Barnes (1994) among native American populations, none were observed here.

### 7.1.c Cleft neural arch

This was the most commonly observed defect in the study, consisting of both bifid (almost-meeting arch halves) and open neural arches, with the urban sites having the highest rates. York had the highest prevalence among males and females, affecting 51% of males and 32% of females. Barnes previously considered a 25% prevalence amongst Native Americans to be a high level (1994:49). There was a highly significant difference between urban and rural populations. Interestingly, there was also a highly significant difference specifically between the York and Wharram populations, which was unexpected. It has been suggested that rural immigrants in York originated from such villages as Wharram Percy, and that many of these migrants would be likely to live in the poorer parishes of York, such as St Helen's (Goldberg, 1992; Mays, 1997). The highly significant difference between the two populations suggests that they are two distinct populations in terms of cleft neural arch prevalence. If migrants were such a major proportion of city populations, one would expect the difference, particularly in the poorer quarters of the city, to be less. It is perhaps unlikely that such differences would disappear within a generation, unless the relevant environmental factor is particularly influential. It may be therefore that migrants did not make up such a significant proportion of the population in St Helen's as has been thought.

Interpretation of the cleft neural arch results is not as simple as it may seem.

Studies in Japan have shown that aborted foetuses have a high rate of neural arch anomalies (Laurence, 1967). The apparent high prevalence in the urban

sites may therefore reflect a higher degree of reproductive success than in the rural sites. It may be that some environmental factor is enabling greater numbers of affected foetuses to survive to birth and beyond in urban settings, and that the “adverse” factors are in fact acting on the rural populations, where affected foetuses are more often spontaneously aborted. A similar effect may also be behind the higher rates of *spina bifida occulta* observed in York and Chichester (see below on teratogenic effects).

Co-occurrence with other spinal anomalies varied between the sites. The greatest rates of co-occurrence were observed at Chichester and York, and the lowest at Hull and Wharram (as with border shifting patterns). As with some other defects, Hull mirrored the rural pattern of co-occurrence. Again, this may be a reflection of some unknown factor enhancing the survival to birth of affected foetuses in urban settings.

#### 7.1.c.i *Embryological origins of cleft neural arch*

The condition originates as a defect of the paraxial mesoderm, and involves either a major or minor delay in normal development. This delay can be traced to the onset of chondrification; the development of the cartilage form of the element cannot begin until the element reaches a critical size during the blastemal phase. Chondrification centres usually appear at around six weeks' gestation, so any delay will result in a smaller, or absent element. Any cleft is covered with a fibrous band of tissue to “bridge” the gap. Thus, cleft neural arches originate in the first five weeks of gestation, and reflect the action of some agent on the paraxial mesoderm cells, as with segmentation errors outline

above. The condition is usually considered clinically insignificant today. It did appear have some effect on female age-at-death rates, and slightly less on male age-at-death rates. There may therefore be some associated soft-tissue disease present alongside the defect, which is influencing longevity. The highly significant differences noted between rural and urban populations indicates some environmental mechanism at work, of which the defect is a sign.

#### **7.1.d Numerical anomalies**

The rates of supernumerary vertebrae at Raunds and Chichester far exceed rates elsewhere. The presence of so many affected individuals at Chichester suggests that there are other health issues associated with the condition which rendered the sufferers more likely to be admitted to the hospital/almshouse. Extra vertebrae associated with sacralisation were especially common at Chichester, with 73% of those with an extra lumbar vertebra also having some degree of sacralisation. The high levels seen at Raunds may well indicate some local factor acting on the population; alternatively, it may be a reflection of the gene pool among the Anglo-Saxon population. However, the high frequency at Chichester could not be ascribed to the same source. The prevalence among the other late Medieval sites (Hull, Wharram and York) were all much lower, possibly suggesting a change over time, with the affected proportion of the city of Chichester being concentrated in the hospital. The low rate at Hull may be associated with socio-economic status, if the Hull cemetery genuinely holds the better-off population of the area. The lack of overall urban-rural difference in

prevalence could suggest a genetic or a socio-economic cause. Absent vertebrae were less commonly observed than extra vertebrae. Supernumerary vertebrae were more heavily associated with other spinal anomalies than absent vertebrae.

De Beer Kaufman (1977) found that numerical variation in vertebral elements was heavily associated with ethnicity (genetic factors), and that males were more likely to have extra vertebrae, and females more likely to have absent vertebrae. Extra vertebrae were found more commonly among males in this study, although at York the females had the greater prevalence; for absent vertebrae, females had the greatest prevalence at Hull, Raunds and Wharram, but it was males who had the greatest prevalence at Chichester and York.

#### 7.1.d.i *Embryological origins of the defects*

Numerical variation in the pre-coccygeal vertebrae is associated with an unusual number of somites and/or sclerotome division in the blastemal phase in the paraxial mesoderm (see fig. 3.3). Whereas lumbarisation and sacralisation may be also related to somite number variation, with transitional elements fusing partially or completely, in cases of numerical variation an extra vertebra is formed or a vertebra fails to form for lack of a sclerotome pair at the re-alignment phase. De Beer Kaufman's findings supporting a heavy genetic influence may also be applicable here, although the differences observed between the early and late Medieval sites may suggest the influence of a time-dependent factor related to environmental change. De Beer Kaufman studied



African groups and compared them with American blacks, Mongoloid groups and Caucasians. The findings showed a lower rate of extra vertebrae among Caucasians and Mongoloids than among black populations, but that American blacks also had a lower frequency than African blacks. De Beer Kaufman put this reduction down to the intermixing of American blacks with whites, but did not consider the possibility that changes in the environment of American blacks could also be a factor. Obviously many generations had passed since the ancestors of the observed American black populations had arrived from Africa, and the changes in diet and living conditions may also have had an effect equal to, or greater than, the genetic alterations due to inter-racial reproduction. A similar temporal change may have been acting on the English Medieval populations observed here, with rates reducing between the early and later Medieval periods.

#### **7.1.e Block vertebrae**

The prevalence of fused or block vertebrae was very low. Only 10 individuals were affected in all, and none were from the York site. Klippel-Feil syndrome is most commonly seen in modern populations affecting C2 – C3, but a defect at this level was not found in any of the cases observed. Type II Klippel-Feil syndrome was noted in 2 individuals, and type III in a third, involving sacralisation of L5. At Raunds, two of the individuals had type II Klippel-Feil syndrome, which has a strong genetic component, suggesting that those individuals may have been related to each other. The single case of a block vertebra at Hull was in contrast with the rates of block vertebrae observed at

Blackfriar's Friary in Ipswich (Mays, 1991). There, ten cases of fusion were observed, with several suffering from cervical blocks affecting the C2-C3 joint, with the remainder affected at C3-C4, C5-C6 or in the mid-thoracic area.

Individuals with cervical block vertebrae are expected to have some symptoms of disability or neurological weakness (Pizzutillo, 1983; Smith & Micheli, 1995), so the high rate observed at Blackfriar's may indicate a focus of physically handicapped individuals.

Thoracic developmental unions are the least common type of block vertebrae (Barnes 1994:67), but in this study, 50% of cases involved fusion of thoracic vertebrae, which are believed to be asymptomatic according to Smith and Micheli (1995). Barnes (1994: 69-71) reviewed cases of Klippel-Feil syndrome and block vertebrae among Native American skeletal series and seemed to find the condition more commonly among males. With such low rates, it is difficult to make any serious comment about block vertebrae, other than to say that the Raunds population had the highest prevalence, and there were no cases in the York cemetery. Chichester had three cases, and two of these were females with mid-thoracic blocks. This could be a contradiction of Smith and Micheli's comments that such blocks are usually asymptomatic, as the women were buried in a hospital cemetery, but obviously, such a suggestion could not be easily supported.

#### **7.1.e.i** *Embryological origins of block vertebrae*

Block vertebrae originate during the blastemal stage in the paraxial mesoderm field and typically represent a developmental union, in which failure of the sclerotome pairs to divide into separate vertebral elements has occurred. This is the type of union observed in the archaeological cases noted in this study. There is believed to be a strong genetic component to the Type II condition (which was the most common type observed here) but as the prevalence was so low in this study, it is difficult to suggest any cause which may contradict this as the principal mediating factor. Pizutillo *et al.* (1994) noted that several associated soft-tissue conditions were noted in a series of Klippel-Feil patients, which may be involved in some way as a result of the vertebral defect (see below). This indicates some sort of relationship between the development of the vertebral column and that of other organs, which could be further investigated among archaeological populations.

#### **7.1.f Spondylolysis and spondylolisthesis**

Spondylolysis is associated with strenuous activity that puts a strain on the lower back and is not uncommon among sports players. It would be expected to appear in susceptible individuals more frequently among agricultural communities, on the assumption that a greater proportion of these populations would be involved in farm labouring. That is not to say that urban work could not be hard, or prolonged, simply that a lesser proportion of an urban population is likely to have been involved in similar heavy work. In practice, any job requiring persistent bending and lifting is likely to exacerbate spondylolysis and spondylolisthesis ( Jayson, 1992). Males were affected more commonly

than females on the whole. Wharram males had the highest rate, which suggests that they were bearing the brunt of the heavy farm work. Chichester males were also highly represented, which may be associated with their hospital/almshouse admission. As a living could be managed without physical hard work in the hospital, it may have been a factor in their admission, whereas a farmer would have probably had to continue working with the condition. Of course, active individuals who maintain good musculature would probably have fewer symptoms than those who were less active, and may have been virtually asymptomatic until they stopped vigorous work in older age. Modern rates of spondylolysis run at 4-7% of the general population, although 2-10% among active young individuals has been suggested (Burkus, 1990; Ferriter *et al.*, 1984; Lowe *et al.*, 1987:582). These rates are far exceeded by the Wharram and Chichester rates among males (15% and 11% respectively). Rates at other sites are also on the high side of the normal modern range. Hull showed a similar picture of spondylolysis to that at Blackfriar's (Mays, 1991).

The majority of the observed cases involved a bilateral spondylolysis. This is significantly associated with nerve root compression (Burkus, 1990:558), and may therefore represent a significant health issue in terms of low back pain, often severe. Burkus (1990) and Porter and Park (1982) found that *spina bifida occulta* was heavily associated with spondylolysis in modern populations. This was borne out in the archaeological populations as well, with between 10% and 14% at Hull, Raunds and Wharram having *spina bifida occulta* or cleft neural arch associated with a *pars* defect (clinicians do not distinguish *spina bifida*

*occulta* from cleft neural arch as palaeopathologists do now). However, the association between *pars* defects and neural arch defects was 29% at Chichester and 54% at York. The Chichester figure could be related to hospital/almshouse admission, but the York figure stands out as unexpected. Whether some environmental force is at work in York is uncertain, but the fact that both sites with a high prevalence are the cities, is probably significant. Burkus suggested that the presence of a midline weakness due to the neural arch defect led to the spondylolysis, but many of the observed *pars* defects in this study did not involve a complete break. This could simply be a reflection of the higher rates of physical activity maintained by workers in the past, however, with a well maintained musculature supporting the weak *pars interarticularis* and preventing a break from occurring as often as in modern cases.

Only a minority of individuals had an associated spondylolisthesis, and none of these was severe enough to cause any significant slippage. This also supports the suggestion of the past populations maintaining a higher rate of physical activity, as strong musculature also prevents or lessens slippage. The highest rate of spondylolisthesis associated with spondylolysis was observed at Chichester, which would again appear to be appropriate for a hospital/almshouse.

#### 7.1.f.i *Embryological origins of spondylolysis*

The dysplastic and isthmic forms of spondylolysis are the only types which have their origins in foetal life, with the dysplastic disturbance of growth producing an

abnormally shaped/formed neural arch, and the isthmic form producing a *pars interarticularis* with an inherent physical weakness. To the author's knowledge, no study has been conducted specifically on the origins of these two forms. This is probably due to lack of cases to study, as Grieve (1981) believes that most modern cases are of other forms, such as traumatic, pathological or degenerative origin. This study did not aim to distinguish between the types of spondylolysis, merely to diagnose the condition as either present or absent, although further research is advised.

#### **7.1.g Cervical ribs**

This condition was found in all sites except Wharram Percy, affecting a total of 11 individuals. The highest prevalence was found in York (four), but numbers were low at all sites, making interpretation difficult. Black and Scheuer (1997) believed that females were more commonly affected than males, but in this study, more males were affected (64%), although there are only 11 cases, so this may not be an appropriate interpretation. Neurological impairment, often with associated circulatory disturbances, may result in weakening and sometimes loss of function of the arm (Barnes, 1994; Pizzutillo, 1983; Smith & Micheli, 1995). This is often an unavoidable result of the presence of the extra rib or bony nodule, with the single congenital defect potentially resulting in serious disability to the individual. Barnes appears to contradict other authorities on the frequency of such side effects, stating that the majority of individuals remain asymptomatic, but it is difficult to see how this could be the

case, as the passage of the brachial plexus is so involved with the morphology of the lowest cervical vertebra.

#### **7.1.g.i** *Embryological origins of cervical ribs*

Cervical ribs represent a confusion of vertebral characteristics at the cervico-thoracic border. Again, the condition originates in the blastemal stage in the paraxial mesoderm field, with the costal processes of the last cervical vertebra becoming extended into a bony tubercle, spur or rib-like extension (Barnes, 1994:100). The condition is, in effect, a border shift affecting the cervico-thoracic border, and thus originates in much the same way as outlined above for lumbarisation/sacralisation.

#### **7.1.h** **Oro-facial cleft defects**

The conditions associated with the occurrence of clefts nowadays were also present in the Medieval period (for example, working with pelts, and tanning and curing of leather). However, the modern use of solvents may now be a significant contributory factor, which would not have been the case during the Medieval period. Alternatively, it may be feasible to assume higher defect rates associated with leather-working in the past, as more leather per person was produced, and the work was more labour-intensive (pers.comm. A. Millard). This, of course, would be dependent on the assumption that pathogens within the curing and tanning processes were contributing to defect rates.

Midline cleft palate affecting the back of the mouth is the commonest cleft defect (1:400, Levene & Tudehope, 1993), and it had been expected that some examples may be found (a total of 730 maxillae were observed). No clefts affecting the palatine process were observed, however. The only clefts observed affected the front of the mouth, in the form of probable cleft lip and, in three cases, a probable associated cleft palate. This was, to some degree, unexpected, as cases of visible clefts being reported among English populations in the literature are rare (see Chapter 2). On statistical analysis, this figure of 6/730 is statistically significant (at 95% chi-square) when compared with the modern rate of 1 in 500 births. On commencing this study, it was thought that such individuals may well have been subject to infanticide, but this evidence suggests otherwise, at least for some affected individuals. The presence of these individuals indicates that those with an abnormal facial appearance were at least tolerated by society. The absence of any visible clefts at Wharram may simply indicate that either there were no affected individuals buried there.

Females were more commonly affected than males in this study. Bilateral and unilateral defects were noted, although unilateral defects were more common. The York population had the highest rate, with four cases of cleft lip/facial abnormality, and Raunds had three cases. Only two were observed at Chichester and one at Hull. No cases of oro-facial clefts were observed at Wharram Percy. There does not seem to be a discernible pattern, but the numbers are very low. It could, however, be said that the condition seems to be linked to the poorer socio-economic groups. The fact that only two cases were



noted at Chichester may suggest that it was possible for affected individuals to live in society without having to resort to the hospital/almshouse, assuming that other cases were to be found elsewhere in Chichester. Alternatively, as has already been discussed elsewhere, selective admission to the hospital may have effectively excluded greater numbers of individuals with cleft defects. The two observed are highly unlikely to have been the only affected individuals in Chichester. Three adolescents (Raunds and York) had cleft defects, suggesting the co-occurrence of other health issues that may have contributed to their early deaths. The Raunds, Hull and Chichester individuals died in early to mid adulthood, but the one affected York female who could be aged lived past 35 years, and the York male was in the same age group. What this indicates is uncertain.

Co-occurrence of cleft defects with other axial anomalies was not apparent. Only a small number of those affected had associated axial anomalies which consisted of cleft neural arch or numerical variation. Soft-tissue defects such as genito-urinary anomalies and inguinal hernia are associated with cleft defects (Emanuel *et al.*, 1973; Ikeda, 1973), and these may, of course, have been present in some of the individuals observed in the study. Nutritional and metabolic issues have been related to the frequency of cleft defects (Avery & Taeusch, 1984; Barnes, 1994:187; Levene, 1991; Levene & Tudehope, 1993; Miller, 1973), and these may be suggested as possible causal factors in the past. The Raunds and York populations may well have had greater nutritional problems than those of the other sites. There was a relatively high rate of stress

indicators present at Raunds, all of which are associated with nutritional stresses (Goodman *et al.*, 1988). In York, represented by one of its poorest parishes, low socio-economic status, population density and the health issues associated with these may have led to nutritional and metabolic impairment. It is difficult, however, with such low numbers of observed cleft defects, to reach significant conclusions about rates in the past.

With regard to the infanticide issue, the evidence seems to suggest that this practice may not have had the role originally assumed by the author. The presence of several individuals with apparent facial abnormalities suggests that visible defects did not necessarily result in the death in childhood of those affected. On the other hand, only a small number of affected individuals were observed out of the large number of skulls examined, so a fairly accurate prevalence may have been ascertained here. Anderson's reference (1994a) to the repair of a cleft defect also suggests that affected people were treated with some degree of sympathy. The apparent absence of palatine process clefts (at the back of the mouth) may suggest, however, that affected infants had a high risk of death due to their failure to thrive. Although such babies do not all have serious feeding difficulties today, it may be that those in the past succumbed in greater numbers to the commonly-associated upper respiratory tract and ear infections, which may have contributed to their deaths. Alternatively, the paucity of cases may well be an accurate reflection of the rates in the past. Modern-day frequencies may be higher than among past populations, due to

better medical treatment and changing attitudes in the developed world to those with facial abnormalities.

## 7.2 STRESS INDICATOR PREVALENCE AND HEALTH

### 7.2.a Male and female responses to stress

What the relatively high rate of birth defects among males indicates is uncertain. It could be that males are indeed less buffered against environmental insults than are females, when *in utero*. Alternatively, the high prevalence in males may be a sign of their success against the odds - perhaps more affected males were surviving to birth, and the majority of affected females were being lost to spontaneous abortion. Without investigation of modern sex and defect analysis of aborted foetuses, this will be impossible to ascertain in modern populations. On the other hand, the high rates seen in urban environments may reflect a greater survival-to-birth of affected individuals among urban populations, and the lower rural rates may be because of an increase in spontaneous abortion of affected individuals. However, the author cannot accept this suggestion. The notion that, in effect, population density leads to an increase in the reproductive success of females (seemingly against the evolutionary improvement of the population), when they are exposed to greater degrees of pollutants, pathogens and poorer quality food and water, does not seem a tenable one.

Overall stress indicator frequency was highest in the Chichester sample, which equates well with a hospital population. It does not, however, match well with the mean stature of the Chichester sample, unless one considers the presence of

stress indicators a sign of successful survival against physical odds. The general health of those buried at the Chichester hospital/almshouse was expected to be less robust than elsewhere, as evidenced by Boocock *et al.* (1995) who found high rates of maxillary sinusitis among the population (5.4%). This finding was echoed at York, where similar high urban rates of sinusitis were observed (see below). The overall rates of stress indicators at Hull appear to be high, but this is largely due to the unusually high rate of tibial periostitis. The Wharram population had the lowest overall rate of stress indicators, suggesting, the author believes, a population in a state of equilibrium with its environment. The overall results of stress indicator prevalence seem to suggest that males are less buffered against environmental stresses than are females, once female reproductive health has been considered.

The picture of stature related to stress indicator patterns at Chichester (and at the other sites) has raised an interesting point. Among females, those with *cribra orbitalia* had a taller than average mean stature, and all of the rural women, and those in the hospital, with tibial periostitis, also had a taller than average mean stature. The pattern of male stress indicators and stature is, however, more uneven. It appears that a raised stress indicator prevalence is associated with those whose stature is on the taller side of average for any given population. This probably warrants further investigation, during which appropriate statistical analysis could be undertaken. The results from this study show that individuals who reached the taller levels of growth within their population were more susceptible to stress indicators, particularly *cribra*

*orbitalia* and tibial periostitis. As these indicators are largely associated with pathogen load and compromised dietary intake/uptake, it may be a sign that attaining a taller than average stature incurs a price – that of susceptibility to pathogenic action, resulting in reduced nutritional and possibly immune status. Furthermore, the picture at Chichester, which has the greatest mean stature of all the sites observed, suggests that taller individuals may be more likely to be subject to adverse health responses that express as stress indicators than their shorter counterparts. The mean stature which was observed among the Chichester population may perhaps be best explained by suggesting the action of some mechanism by which taller people who also have skeletal defects and, presumably, other soft tissue anomalies or disease, are more likely to succumb to those conditions which produce stress indicators on the bones. Perhaps the process of growth retardation which results in adults achieving less than their genetic height potential is actually a form of adaptation which allows the more healthy functioning of the body. Those who fail to “tailor” their growth to the prevailing circumstances may then have to pay the price – the nutritional status required to support the larger /taller body may be too thinly stretched, thus rendering them more at risk to metabolic stress. Presumably this could in turn have an effect on their reproductive health (see Conclusions).

Overall, females had a greater burden of stress indicators. This may be a reflection of females' improved response to stress, or of greater female susceptibility to infection or other environmental insults. Discounting the Chichester population for a moment, the rural females had the highest rates of

*cribra orbitalia*, suggesting either a greater female pathogen burden, or a nutritionally-related iron deficiency anaemia in addition to the greater female requirement for iron. Given the issues of population density and its associated hazards, the pathogen load argument is less convincing than the nutritional one. It is possible that rural males had the pick of the best food, and the source of the rural female metabolic stress was a nutritional deficit rather than a pathogen overload leading to a nutritional deficit. Alternatively, rural women may have begun reproduction at an earlier age than their urban counterparts, thus incurring greater physical demands on their bodies at a younger age.

It appears that susceptibility to stress, or at least a physiological response to stress, is related to site-dependant conditions. The high proportion of defect sufferers with associated stress indicators at Chichester (81%) has already been suggested to be associated with hospital/almshouse admission (even on a selective basis), and the high Hull rates (81% of male defect sufferers, 75% female defect sufferers) are skewed due to the tibial periostitis profile there. The relatively low rate of stress among York females (40% of defect sufferers) may be due to some delay in child-bearing until a slightly later age among urban women. Alternatively, York women may have simply been better adapted at dealing with environmental stresses. Defect sufferers at Wharram Percy had the lowest proportions of associated stress indicators (36% and 39% respectively in males and females), which possibly suggests a constitution in a relatively stable relationship with the local environment.

### 7.2.b *Cribra orbitalia*

The condition is frequently related to nutritional problems leading to reduced uptake of dietary iron. Gastro-intestinal conditions leading to malabsorption through parasitosis or diarrhoea, for example, often produce iron deficiency. A raised pathogen load is also a likely cause of malabsorption due to impaired activity of the normal flora of the gut. In females, blood loss conditions also exacerbate the potential anaemia. It was therefore unsurprising to find that females were most commonly affected by *cribra orbitalia*. Significant blood loss through menstruation, post-partum complications and gynaecological problems were probably implicated in this prevalence. The high rate at Raunds (71%) suggests a particularly harsh health picture for young women. Chichester females also had high rates of *cribra orbitalia* (52%), a higher rate than that of the Wharram females (42%). Higher female *cribra orbitalia* rates may also indicate cultural preferential dietary provision for males, particularly in times of economic stress, which is a recognised health issue today among some cultures (Lindenbaum, 1977; Harris & Ross, 1987). If such nutritional discrimination is extended to girls as well as to adult females, a pattern of female undernutrition is set up (Hagen, 1999), which would have jeopardized the reproductive health of the population in the past just as it does now (Allen, 1986). A significant difference was observed between the two urban sites, with Chichester having the higher rate among females. This may well be due to a “successful survivor” phenomenon, in which affected females were actually physically successful “against the odds”, and their *cribra orbitalia* a sign of that success. Females

with anaemia in York (29% *cribra orbitalia* prevalence) who did not have the group care and diet of the hospital environment, may well have died earlier before the signs of *cribra orbitalia* appeared. Alternatively, as many individuals suffer low-grade iron deficiency for years without having clinical anaemia, the York women may have been more successful at adapting to reduced iron uptake and therefore did not develop *cribra orbitalia* in such high numbers.

### 7.2.c Enamel hypoplasias

Enamel hypoplasias were the least commonly-observed stress indicator. However, 40% of females at Chichester had this indicator present, supporting the suggestion that a period of stress during the early years has on-going repercussions throughout life. The stressful episodes that resulted in the development of enamel defects may well have permanently reduced their state of health, leading to their admission to the hospital/almshouse. The Raunds females had the lowest rate of enamel defects (17%), which is possibly a reflection of the death of affected females before reaching adulthood. At York (24% female, 26% male) and Wharram (31%female, 41% male), more males than females had enamel defects, again suggesting either a higher male survival rate, possibly due in turn to preferential male diet and care, or a truly higher male prevalence. At Hull, females had the greater rate of enamel defects (28% male, 32% female), which may reflect the better quality of post-childhood life for females among the better-off (enabling more of them to survive to adulthood) - if this is truly the social make-up of the Hull cemetery. Significant differences between the rates among females at Raunds and Chichester and



Raunds and Hull support the suggestion that cultural factors may well be influential in the survival of females with enamel defects.

#### 7.2.d Tibial periostitis

Tibial periostitis is recognised as a response to pathogen load in the body (Goodman *et al.*, 1988), and is therefore an indicator of infection. The urban populations had higher rates of tibial periostitis than the rural groups, suggesting a greater urban exposure and/or greater susceptibility to pathogens. Raunds females had a relatively low prevalence of periostitis (28%), but the age-at-death pattern of those affected shows that the condition was strongly related to the early child-bearing phase and indicates a significantly reduced health status amongst young women at Raunds. Tibial periostitis was unusually highly-represented at Hull. This must be the subject of further debate, and may be related to the presence of treponemal disease at the site. Whether the high rate of periostitis is directly related to trepanematosiis is uncertain, but if periostitis is present at the site only as a stress indicator, it does not equate with a higher rate of other stress indicators, which one would expect. At York, 48% of women had tibial periostitis, an even higher figure than that of Chichester females (42%). Hall *et al.*, (1988) suggested that the predominance of females in the St Helen's cemetery may be due to the number of prostitutes living nearby in the tenements of Aldwark. Women engaged in casual or long-term prostitution would be at greater risk of frequent infection, and may therefore be expected to show higher rates of tibial periostitis, and possible cribra orbitalia as a result of anaemia related to compromised nutritional status. Casual

prostitution was apparently a way of earning a living for poorer women between jobs in the textile industries of York (Hall *et al.*, 1987 ;Goldberg, 1992), and such women would have been unlikely to eat a good diet due to their financial problems. In fact, the Aldwark cemetery, being one of the poorest in York, is a likely focus for the poorer inhabitants, whether migrants or long-term city-dwellers. Poorer nutritional and immune status would therefore be expected among such a population. The general health of those buried at St Helen's is also illustrated by the findings of Lewis *et al.* (1995) who found a prevalence of 55% with maxillary sinusitis. This rate was much higher than that seen at Wharram Percy (39%), suggesting an urban environment more conducive to upper respiratory tract infections and, due to the longstanding nature of the condition in order to develop bone changes, the reduced immune status of the affected individuals. Wharram inhabitants had the lowest rate of periostitis (26% female. 23% male), suggesting that they were either more able to resist infection, or that that they fought it more efficiently and tibial periostitis did not have time to develop. Alternatively, they may have succumbed to illness and died so quickly that a bone response did not occur, as Mays suggested (1997). However, the peasants of Wharram may not have been as "unhealthy" as has been thought previously, despite their existence at the bottom of a "highly stratified society" (Mays, 1997:125). It has been said that only long-lasting infectious conditions leave periostitis on the bones (Mays, 1997:123), and that the lower rates of periostitis seen among some populations indicates that they died in greater numbers before their bone had time to respond to the infection. However, if an individual is continually affected by infectious agents for a long

period *after* having already been subject to a serious initial infection, then that individual's ability to fight off the subsequent infection efficiently is likely to be impaired. The periostitis, once established, is therefore likely to persist for a longer period of time, as the individual's immune response is impaired and the lesion will not be healed as quickly as it would have been had there been only one episode of infection. Hence, when we see higher rates of periostitis in the St Helen's population, and lower rates among the Wharram population, it is not entirely accurate to say that this means that Wharram people may have died quickly of the infection before periostitis could develop. Wharram people may simply have been exposed to fewer episodes of infection, and have had the time and the efficient immune response to heal any periostitis lesions completely, without being re-infected and prolonging the life of the lesion. The fact that the majority of lesions observed in the St Helen's population were healing simply indicates that the individuals were fighting the infection successfully. The Wharram lesions were also predominantly healing, but there were fewer individuals affected, suggesting a lesser pathogen load rather than the early death of infected individuals. The age-at-death patterns at Wharram do not necessarily support the notion of quick death as a response to infection.

Males had higher rates of periostitis at all sites where they were examined for the indicator, suggesting that either males were engaged in more traumatic activities, were more susceptible to infection than females, or were more successful at fighting infection. There was a significant difference between affected males at York and at Wharram (and between females), suggesting that York males were more susceptible to, or poor at resisting, infection. Higher

rates as a result of trauma to the shins may be expected among an agricultural group, but this did not appear. The high rates of periostitis seen among York males may well be related to nutritional factors and the reduced mean stature of males at York (see below "sexual dimorphism") compared to other populations. On the other hand, there may have been a greater risk of shin injuries in towns with wheeled traffic and horses in the crowded streets. It seems likely that the high infection loads implied by the periostitis rates have also had a bearing on the overall growth of the populations affected, particularly amongst the males.

### 7.3 ADULT AGE-AT-DEATH PATTERNS AND DEFECTS

Sacralisation Female longevity appears to reflect the presence of sacralisation. Women at Chichester and Raunds had a greater risk of dying in young adulthood if they had the condition. In Raunds, where women would be expected to be sexually active and reproducing, the high death rate in the 17-24.9 year age group may well indicate a problem associated with childbirth. All the affected Raunds women died by the age of 44.9 years, which effectively covers the reproductive period. Reduced mobility in the lower spine may have been a complicating factor in labour, either increasing the risk of death at the time of parturition by prolonging or indirectly obstructing labour. Anecdotal evidence noted by the author, on observing several photographs of female graves with infants, has associated sacralisation and/or an extra lumbar element with those women. Chichester women, even though they were residents in the hospital /almshouse, and would not be expected to be reproducing, also showed a similar pattern of early adult death. They could have been admitted however,

following childbirth, as a result of associated illness. Wharram Percy females, however, were also agricultural peasants, but did not share the age-at-death pattern of the Raunds women, generally surviving in greater numbers into the older age categories. Wharram women with sacralisation did die in higher numbers in the 17-24.9 year group than those unaffected, but the better survival rate (compared to Raunds) may indicate better general female health, improved midwifery practice, more help within the family/community for pregnant and labouring women, or better work conditions which did not require women to work at such physically hard tasks during or after pregnancy and childbirth. York females did not have such a high risk of death in the young adult age group, with the majority surviving into the older age groups. This may well be due to different reproductive patterns in the city, with delayed marriage and motherhood being practised by women who were working to support themselves. In addition, those women in York who did have children and also worked, would have not been subject to the same heavy agriculturally based work (which could cause further back problems) as women in the rural setting.

Overall, male longevity does not appear to significantly reflect the presence of sacralisation. Males with and without sacralisation at Chichester and York did not show any difference in age-at-death patterns. At Raunds, however, affected males had a greater risk of dying in the 17-24.9 year age group. This may also be a reflection on the reduced mobility of the lower spine, perhaps rendering such young men more likely to injury during agricultural work. In an agricultural subsistence setting, low back pain syndrome would have been an

economic as well as a health issue, impacting on the sufferer and his family's well being. Wharram males with sacralisation did not have an increased risk of death in early adulthood. This risk fell to the unaffected males, suggesting that perhaps the stronger unaffected males were bearing the brunt of the heavy work on the land and with animals, and perhaps therefore also suffering the resulting trauma and general "wear" of the work as a result. It seems unlikely that sacralisation alone would remove or significantly reduce a man's working capabilities to such an extent, but if the condition is associated with some unseen soft tissue disease, then this could also be a factor in the observed death rate patterns. Wharram had a 45% overall rate of co-occurrence of other spinal anomalies with sacralisation, and a rate of 88% among males with sacralisation, so affected males may well have been effectively removed from the heavy work force due to other health issues, or at least taken a reduced role in it.

Lumbarisation Bearing in mind the possible beneficial effects of increased mobility at the lumbo-sacral junction, it was hoped that sufficient numbers of Raunds females would have the condition to enable a comparison to be made with the sacralisation cases. Unfortunately, only two women at Raunds were affected by lumbarisation, so the possible benefits of the condition in terms of childbirth cannot be assessed. Both women died by 44.9 years, however. Affected females at Chichester and York had a reduced risk of death in early and mid-adulthood, which may reflect the positive effects of lumbarisation on childbearing. Overall, a greater proportion of women with lumbarisation survived into old age than those without the condition, suggesting the action of

some sort of protective mechanism. As with sacralisation, however, affected Wharram females show the opposite pattern, having a higher risk of early death than those without the condition. Why this should be is uncertain. Among affected York and Wharram males, lumbarisation is associated with a lower risk of early death also. This may reflect the relative ease with which heavy work could be done with fewer strains on the back. These patterns are confused by the fact that overall, lumbarisation was associated with a very high co-occurrence rate with other defects (87%). The Wharram population showed the lowest co-occurrence rate, at 55%, but this does not explain the higher risk of early death among females with the condition there.

Supernumerary vertebrae In all sites except Hull, affected males were at a higher risk of early death than their unaffected counterparts. The same pattern appears in females also. At Wharram, all affected women died by 34.9 years. A generalised raised risk of early death among the observed populations seems to be associated with the presence of extra vertebrae.

Absent vertebrae Females at Chichester and Wharram do not appear to have been adversely affected, in terms of early death, by the absence of a vertebral element, but at all other sites both males and females seem to have been at a raised risk of early death than those with a normal number of vertebrae.

Spondylolysis and spondylolisthesis The former condition is associated with an increase in risk of early or mid-adulthood death among males and females.

York is the exception, as there was no obvious difference in age-at-death profiles between those with and without the condition. This may well be a reflection of cultural practices, with city dwellers not being subject to a heavy agricultural lifestyle. Few individuals were found to have spondylolisthesis, but of those that were, at Chichester, Raunds and Hull, affected individuals died before the age of 45 years. At Wharram and York there was some survival post-45 years, indicating that the condition was either of little effect, or alternatively, that the individuals concerned may have remained very active until old age, thus maintaining muscle tone to support the defect.

*Cribra orbitalia* This stress indicator was widely associated with an increased risk of death in early adulthood. At Hull, Raunds York and Wharram, the majority of those affected died before 45 years, but at Chichester, the condition is associated with a greater survival rate into the old age group. This may be because the females of the Chichester site were spared the risks of child bearing, or because they were simply more susceptible to stress indicator-inducing conditions due to their taller than average mean stature (see above). Raunds and Hull young females had particularly high death rates in age groups 17-24.9 and 25-34.9 years. Menstrual blood loss that was not compensated for by an adequate uptake of iron may have been a contributing factor. This may also be related to childbirth and pregnancy issues, with young women at Raunds likely to be producing their first infants in the early adult age group, and thus suffering the difficulties of the first pregnancy and delivery. In addition, if females at Raunds were suffering nutritional deficits because of cultural preferential



treatment of males, then their lack of iron would only be exacerbated (Hagen, 1999:354). The marriage age tended to go up as the Medieval period progressed, which may explain the slightly older women at Hull appearing to suffer the same fate. Long-standing anaemia would, of course, reduce the overall strength of women, and may well contribute to reproductive problems, due to both physical and metabolic factors.

Males had lower rates of *cribra orbitalia* at all three sites where males were observed for the condition. This alone suggests that some reproductive/menstrual element may be an issue among females, but may also suggest a preferential diet allowed to males. Alternatively, males may be buffered against the effects of reduced iron uptake compared to females. Males at Hull and Wharram had very similar rates, and there was no significant difference between the female rates at either site, suggesting some common factor at work.

Enamel hypoplasias This stress indicator appears to have had mixed effects on age-at-death patterns. No differences were noted in age-at-death profiles of affected and unaffected females at Chichester and York, or of males at Hull. However, affected females at Hull, Raunds and Wharram died in greater proportions in the 17-24.9 age group, again suggesting a possible link with reproduction within those sample populations. Barker's work on maternal childhood health indicates that stresses on the mother during her own childhood have a significant impact on her reproductive health, as well as that of her

children (Barker, 1994). The observations made here may well reflect this phenomenon, showing that women with enamel defects have suffered significant stress during their own childhoods, and then pay the price with an early death. Affected males at Wharram and York were also at greater risk of early death than unaffected males, suggesting the presence of an on-going reduction in health status resulting from childhood stresses.

Tibial periostitis This condition was generally associated with those dying before the age of 45 years in both males and females. Raunds females in particular had massive rates of periostitis (60%) in the 17-24.9 year age group. The large proportion of Raunds women who died by the age of 25 years, and who also had tibial periostitis, may therefore be an indication of both their general health (very run-down) and also of the hardship of their life.

#### 7.4 STATURE AND BIRTH DEFECTS

The groups in this study have had stature recorded consistently by the same method, therefore comparison is feasible (Waldron, 1998). Goodman *et al.* (1988) found that sexual dimorphism decreases under conditions of increasing stress and therefore, if the hypothesis of this study is correct, reduced sexual dimorphism should be observed among the urban groups, assuming that there is no significant disease present among the comparable rural populations. The results supported this, with the York population having the least sexual dimorphism.

Gray and Wolfe (1980) found that sexual dimorphism of stature was strongly influenced by diet as well as genetics. They found that female height was negatively correlated with dimorphism, but male height positively (1980:445ff). Protein availability was related to male and female tallness in their study. Female height was unaffected by this, although male height was. They concluded that sexual dimorphism is related to protein availability, and that people living in higher protein availability conditions are more likely to reach their genetic potential for tallness than those with less protein availability. They also found that sexual dimorphism is also associated with food plentifulness and security of food supply. Those societies with secure and/or plentiful supplies show the greatest dimorphism. Settlement size was significantly related to male height in their study. Males tended to be taller in societies with small settlement size (polygyny and high protein). This does not necessarily imply a cause and effect relationship, but it does suggest that there may be other factors at work. Frisancho *et al.* (1980: 367) found that environmental factors such as nutrition exert more influence over body size during childhood rather than adolescence, and vice versa in terms of genetic control over body size. However, under conditions of malnutrition, the genetic influence was diminished. This implies that large proportions of populations with a reduced mean stature may well have been subject to nutritional deficiencies in childhood.

Sexual dimorphism of stature was greatest at Hull (10.8 centimetres difference between the male and female mean statures), suggesting a higher status population who had access to a good diet, certainly during the sub-adult years

(Gray & Wolfe, 1980; Frisancho *et al.*, 1980), and least at York (8.8 centimetres difference). A reduction in sexual dimorphism is well associated with stress (see above regarding nutrition and infection stresses), as environmental stressors have been found to cause growth retardation in males (Stinson, 1985). Chichester had the second highest rate of sexual dimorphism (10 centimetres difference), supporting the idea that residents had enjoyed a reasonable diet in earlier life and suffered fewer environmental stresses despite being in a hospital/almshouse. This seems contradictory, as it is assumed that the hospital/almshouse residents were the poorest and the sick of the area. One reason which may explain the mean stature and the relatively high degree of sexual dimorphism of stature in the Chichester cemetery may be that the majority of the residents were not in fact the poorest, but rather, those who were sick, but who were able to pay for their admission and "keep" at the institution, as outlined by Clay (1966). As Gray and Wolfe (1980) and Frisancho *et al.* (1980) found, the greatest sexual dimorphism of stature seems to be associated with a high quality, plentiful supply of food, particularly during the growing years of childhood and adolescence (see below). If this is the case, then our view of social and charitable provision at the Chichester hospital/almshouse may have to be reassessed slightly. Raunds (8.9 centimetres difference between the male and female mean stature) had a lower dimorphism than Wharram (9.3 centimetres), which may suggest a harsher life for the Anglo-Saxons, reflected in the reduced mean male stature.

The results of this study show that the York population had the smallest mean stature, in both males and females. This is usually associated with nutritional impairment and/or raised infection rates (Chandra, 1975; Goodman *et al.*, 1988) with the genetic influence on stature being curtailed by nutritional problems (Gray & Wolfe, 1980). These nutritional-infection factors may be significantly related to population density, and offer an explanation for the low mean stature of the St Helen's population. This was expected in a city environment with a relatively greater population density than the rural sites, and pollution from metal-working, tanning, butchery, poor sanitation and so on being prevalent on a permanent, industrial scale.

Chichester had the greatest mean stature of both males and females, even though it is the cemetery of a hospital/almshouse. These results were unexpected, and as outlined above, it seems that as stress indicators were frequently associated with taller individuals (both male and female), it may be that there is some unidentified interaction between stature, nutrition, pathogen load, and immune response, with taller people stressed as a function of their stature.

The Hull population had the second largest mean stature for males and females, which would normally be taken as suggestive evidence of a well-off or well-provided for group of people. As this cemetery would contain Augustinian brothers and their benefactors, this is not particularly surprising, although it does beg the question as to the whereabouts of the many sick who would have

been expected to be buried in the monastic cemetery if they died in the hospital. It may be that there was a separate cemetery for the sick, or that they were not specifically catered for on-site in great numbers. However, if the stature and ill-health issue as outlined for the Chichester sample is accurate, the stature of the Hull sample may also be more likely to be indicative of a sick and possibly poor population.

Wharram Percy and Raunds had very similar mean stature figures for males and females, which would be expected for two agriculturally-based peasant subsistence communities. It had been expected that the Raunds population would have been perhaps even shorter than the Wharram group, but this was not the case. Wharram males were taller than Raunds males, but Raunds females were slightly taller than Wharram females. The relative shortness of Raunds males compared to Wharram males may be a result of the disparity in protein availability as outlined below.

In terms of conditions affecting stature, the results show that certain defects were associated, amongst the observed populations, with reduced stature, and other with increased stature. This also varied between males and females. For example, most males with sacralisation were shorter than those males without the condition (except for Wharram males), but this was reversed in females, with affected women tending to be slightly taller than unaffected women. Lumbarisation, on the other hand, tended to be associated with an increase in height in both males and females (except for Wharram females).

The most interesting results for stature involved the stress indicators. *Cribra orbitalia* was associated with taller individuals as discussed above, but tibial periostitis was also associated with taller females at several sites, and may have been due to the same effect, i.e. stress as a "pay-off" for their physical success. Enamel hypoplasias however, were largely associated with reduced average stature, although there were exceptions. As enamel defects relate to periods of stress during growth, this is not surprising.

#### 7.5 JUVENILE DEFECT PREVALENCE

There was a discernible pattern of defect prevalence along urban/rural lines, with Raunds and Wharram having lower rates than Chichester and York. The rate of defects among juveniles at Hull was much greater than at either of the other urban sites. This is discussed below. As with adults, cleft neural arch was the most commonly observed condition among juveniles. There was also a significant proportion of supernumerary and absent vertebra cases among the juveniles, suggesting a link with early death. Among adults, these conditions were among the least well represented, suggesting that those individuals who survived into adulthood with numerical variation in the spinal column were the exception rather than the rule.

Hull had a much greater defect prevalence among juveniles than any of the other sites (42% as against 12% - 26% elsewhere). If this population is truly derived from the higher social ranks, then this prevalence may be due to some socio-

economic cultural effect that is not in action at any of the other sites. What this may be is uncertain. One suggestion is that the children of the richer members of society may have been actually more susceptible to infections, or some other health issue, which may or may not have been particularly prevalent at Hull. Perhaps if the if the treponemal disease hypothesis is correct, and a large proportion of the adults present were afflicted, then their children (if these juveniles are the children of the adults here) were also affected and were subject to the disease killing them, as they were already constitutionally weakened by health issues indicated by the presence of spinal anomalies. Even if treponemal disease itself did not directly cause the deaths of many of the children, it may have sufficiently weakened them further so that they succumbed to other infections that led to their deaths.

Most of the juveniles observed in the study had only a single defect, which is the opposite of the adult pattern at Raunds, York and Chichester. Only at Hull and Wharram did the high rate of single defects affect juveniles and adults predominantly. At all sites, juvenile prevalence of defects was lower than adult prevalence. The only exceptions to this rule were observed in the prevalence of cleft neural arch rates at Hull and Wharram, where juveniles had a higher rate than adults; juveniles at York, Raunds and Wharram also had a higher prevalence than adults of absent vertebrae, and supernumerary vertebra prevalence was also higher among juveniles than adults at Hull.

In terms of age-at-death patterns, the greatest numbers of children appear to have died in the 6 - 12.9 year category. Wharram had a particularly high death



rate in this group, with over 45% of the juveniles represented dying between 6 and 12.9 years, and Raunds lost 30% of its observed juveniles in the same age range. This suggests the concentration of one or more health factors acting on this age category. In the older category, 13 - 16.9 years, figures are similar for all sites. In all populations, the relative lack of neonates and under-1-year-olds is noticeable. This is partly due to the fact that the remains of many infants and toddlers did not contain sufficient material to be of use in this study. For example, the non-fusion of neural arch halves makes diagnosis of anomalies difficult or impossible, depending on age and preservation. Large numbers of maxillae survived, however, and cleft defects were not observed in any, except a possible case at Raunds which was difficult to identify definitively.

It is difficult to suggest reasons for the variation in prevalence of defects in juveniles between sites. Raunds has the lowest rate of defects among children and adolescents, but if the prevalence of cleft neural arch and absent vertebrae were to be discounted, the Raunds rates would be very similar to the Chichester, York and Wharram rates. Cleft neural arch rates and numerical variation comprised the highest prevalences, with supernumerary vertebrae more common at Hull and absent vertebrae more common at York. Why this pattern of numerical variation should be is uncertain. Sacralisation and lumbarisation were only observed among juveniles at York, not Hull. The only other site where lumbarisation was observed in juveniles was Raunds. Whether these sub-adults died before reaching adulthood because of health effects related to their defects is unknown. The only cases in which this could possibly be

suggested with any degree of confidence are those children who had a cleft sacrum or a sacral *spina bifida occulta*. As this condition can be related to a tethered *cauda equina*, which is associated with increasing paralysis of the lower limbs (and bladder) as the child grows, it may be possible by metric analysis to show evidence of disuse atrophy in one or more of their legs, which could suggest a death associated with the condition. Other conditions, being frequently single anomalies, cannot be attributed as cause of death with ease.

#### 7.6 PSYCHOIMMUNOLOGY AND ARCHAEOLOGICAL POPULATIONS

Stress and/or distress are now recognised as stimuli of physiological responses in both humans and animals, affecting the nervous and immune systems. The alarm response to stress is generally seen as an *acute* event (fight or flight), but the evidence derived from this study suggests that there may also be *chronic* stress mechanisms acting on the human body, resulting in an altered frequency of certain skeletal anomalies and stress indicators. These variable frequencies appear to be linked to some environmental factors acting on the populations observed in this study. Whether these mechanisms are also related to the human mind is a possibility that should also be addressed. Concepts such as the ability to cope, and feelings of helplessness and hopelessness within a stressful situation, are among those which have been shown to have a relationship with illness (Ursin, 1994). They are all implicated in the response of the brain, and therefore the body, to stress.

This study has observed populations taken from urban and rural environments. Whilst the definitions of "urban" and "rural" must remain somewhat flexible, (effects of population density may be said to be most influential on this) the number of individuals observed in the study gives statistical significance. The data suggest that there are quantifiable, significant differences in the response to environmental stimuli between urban and rural populations. The author suggests that these stimuli are largely related to population density and its correlates, nutritional quality and quantity, and to the altered immune response which results from the exposure to these factors. This hypothesis is based on the observed association between defect and stress frequency and urban or rural environment found in this study. There is no proof of a causal effect within these results. Further research is required to elucidate this matter, for example, focusing on a single, transitional urbanizing population.

By using skeletal markers of stress, it is also possible to make comparisons between the health status of specific populations. Stress indicators on bone and teeth are permanent or semi-permanent. Unlike fluctuations in the levels of circulatory immunoglobulins, for example, which may vary over relatively short periods in response to stress, bony and dental stress indicators offer a more stable perspective on the well-being of the individual. The variations observed in this study between and within populations reveal environmentally-mediated biological responses to stressors which can be measured. The large number of individuals observed, and the population-scale differences in the prevalence of certain skeletal anomalies suggests that environmental stressors are having a

substantial and widespread effect on those populations. Genetically, the populations observed in this study are homogenous. The variations in defect prevalence, taken over a time span of several centuries, therefore would seem to be related to some environmental factor(s) acting on the genetic substrate.

Stress differences between the two environmental categories (urban and rural) is an obvious, although not simple, possible causative factor. Such stresses would include physical, nutritional, and pathogenic variables as well as psychological ones.

The hypothesis of this study suggested that urban populations would express a higher rate of skeletal defects than rural populations. This assumed that higher defect rates would reflect increased rates of stress within urban populations. Of course, the opposite could be the case. For example, as Ursin pointed out, a reduced rate of circulatory immunoglobulins in the blood of people removed from wide human contact for prolonged periods may be either a reflection of a low immune status, or of a lack of challenges to the immune system, leading to a reduced response. This latter condition would not be a health problem, merely a reflection of a "quiet" period of immunological activity (Ursin, 1994). A similar phenomenon may be happening with rural and urban populations. Rural populations may have fewer skeletal anomalies, but may be in poorer general health overall. It may be that a certain amount of eustress (beneficial stress) may be required for normal immunological activity. In this case, urban populations may be the more healthy of the two groups. However, as urban population density is associated today with major health problems (WHO 1997),

it seems reasonable to assume that the data indicate a reduction in health status in the archaeological urban populations.

#### 7.7 RELATED DISABILITY AND SOCIOECONOMIC EFFECTS

This study has observed many defects of vertebral segmentation (sacralisation, lumbarisation, cervical ribs, numerical variations) amongst Medieval population samples. Whilst segmentation defects of the vertebral column may not seem high on the list of physical handicaps, they must surely be seen as “more subtle” manifestations (Goldsmith, 1986) of a population reacting to an environmental health challenge over a long period, at a lesser severity than that resulting in excess death rates, such as the plague. Relatively high rates of segmentation anomalies were observed by the author, and when these conditions are considered from a clinical perspective, many are found to be associated with soft tissue dysfunction. For example, the association of lumbo-sacral border shifting with prolapsed intervertebral discs and sciatic disease (Castellvi *et al.*, 1984) constitutes a significant potential loss of working days, and therefore income, to those affected. Rural populations may arguably have been the most influenced by this health impairment, as the agricultural lifestyle was heavy, involving considerable bending, twisting, lifting and carrying throughout the year, and a larger proportion of the population would have been involved in such work. Resulting from this, communities experiencing high rates of these anomalies would have had to share out the load between those who were capable of doing the work. This could lead to undue strain being put on the healthy individuals and result in them suffering greater injury or stress, which

may explain the pattern of earlier death of Wharram males who *did not* have sacralisation.

In towns, notwithstanding the heavy nature of certain types of urban work, a smaller proportion of the population would arguably be affected by spondylolysis, or lumbo-sacral anomalies, for example. Workers such as carpenters, stone masons and some metal workers would have experienced heavy physical work on a daily basis, just as agricultural labourers and farmers did in the countryside. Even so, affected individuals may well have lost work due to persistent back and neurological pain, and may have been restricted in the kind of work they performed. Persistent or regular incapacity must have had a significant impact on the socio-economic wellbeing of affected individuals and their families, particularly for journeymen, who were paid based on their daily work.

The association with spondylolysis of hypoplasia of adjacent vertebral structures (Porter & Park, 1982) is particularly relevant to populations in an agricultural setting. The high rates observed at Chichester and Wharram suggest that the condition was a contributory factor to admission to the hospital/almshouse, and that the farming practices of the Medieval period resulted in a greater degree of wear and tear on the backs of those involved (principally the men). A rigorous study of the cases of spondylolysis observed here may be able to further define the likely origins of the defect (e.g. dysplastic, traumatic and so on), but it appears from the results of this study that if the examples observed here were of

the dysplastic form, then the Wharram people were more likely to actually suffer a *pars* break, probably due to the nature of their physically heavy work.

Whether the high rate at Chichester is due to rural poor seeking admission to the hospital/almshouse, or whether the urban sufferers gravitated towards the hospital producing an abnormally high focus for an urban site is unclear. *Spina bifida* may be one of the conditions particularly influenced by the intergenerational effects proposed by Emanuel and Sever (1973), and by Barker (1994) and others. The findings of Jorde *et al.* (1983) based on the Utah Genealogical Database showed a 70 % rate of heritability of the condition, which supports other workers' findings as to the genetic role in the incidence. Perhaps changing environmental factors may also have a measurable effect. For example, the low rates of 0.2/1000 live births observed in Japan (Laurence & Tew, 1971) must be viewed in the context of the correspondingly high percentage of spontaneously aborted foetuses in Japan with neural arch defects (Laurence, 1966). For some reason, a higher rate of foetuses was aborted than survived to birth at that time. In Ireland, on the other hand, the *spina bifida* incidence in 1971 was 4.2/1000 live births (Laurence & Tew, 1971). No data on contemporary Irish spontaneously aborted foetuses are available to the author's knowledge, but it would be interesting to compare more recent live/aborted rates. The question is, how far are environmental factors influencing the rates of abortion/live births of affected babies? It may be that some environmental factor, probably diet, is producing or perpetuating an intergenerational effect. Perhaps the Irish population is suffering the ongoing ill-effects of a nationally poor diet during periods of extreme poverty in the not-

too-distant past. If this is the case, then perhaps a more recent assessment of Irish incidence would show an improvement in rates of *spina bifida* in subsequent generations as the benefits of a better diet take effect.

Cervical ribs were found in a few individuals. It is likely that at least a third of these people (Black & Scheuer, 1997) suffered some neurological impairment, probably in their arms and hands, as a result of the required adaptation by the spinal nerves exiting the spinal canal. This may have resulted in either motor or sensory impairment, or some degree of both. A metrical analysis of both arms in affected individuals would possibly help with a diagnosis of such impairment. Obviously, affected individuals would have had reduced work capabilities, and in the absence of work which they could manage, may have had to rely on others for their welfare. The clinical literature often focuses on individuals who have experienced a late onset of symptoms following a particular activity which has aggravated the nerve supply to the arms, and it may be that this was the pattern in the past.

Block vertebrae (including Klippel-Feil syndrome) are associated often with hypermobility at the vertebral joints adjacent to the block, which may lead to degeneration in those areas. Neural stenosis (narrowing of the neural canal) and stenosis of the transverse foraminae may result, producing neurological deficits in the arms and neck. Of course, such deficits could have had a serious effect on the economic status of the individual. Soft tissue disease was clearly associated with congenital cervical fusion, as well as co-occurrence of



intervertebral disc degeneration in adjacent areas. Cardiovascular disease, especially ventricular septal defect, hearing impairment, and dysplasias of the arms and hands were found by Pizzutillo *et al.* (1994). It is to be inferred that these conditions also occurred in conjunction with Klippel-Feil syndrome in the past. A survey of those diagnosed in skeletal collections could serve as a base from which to estimate the prevalence of such soft tissue anomalies. Those children with ventricular septal defect, for example, would be unlikely to have survived to adulthood. Other friaries/priories appear to have had several individuals with cervical fusion who may well have suffered some of these soft-tissue anomalies and sought some form of admittance to the institutions, but the Hull friary does not reflect this pattern (see earlier sections of this chapter).

Oro-facial clefts or other facial morphological anomalies were observed in all populations except that of Wharram Percy. The majority of those affected were females, although affected males were observed. This is the opposite of the modern pattern, in which males are more commonly affected. However, the archaeological picture does not tell us the true prevalence, and there may be cultural factors masking the true numbers of affected males. Three of those affected were adolescents, which may suggest that either other soft tissue anomalies or infectious disease were implicated in their early deaths, or that they failed to thrive due to some cultural bias against them. Without further investigation it is difficult to make useful comments about the frequency and disabling effects of oro-facial clefts in these populations, but the initial expectation, that very few, or no, visible defects would be found due to

infanticide eliminating them from the record, has proved to be unfounded. Of course, large numbers may well have been subject to infanticide, and we may be seeing only a small number of survivors, but larger numbers would be required to investigate this further. Genito-urinary anomalies have been associated with oro-facial clefts in the medical literature, so there *may* be some association in the younger individuals observed here, which led to their early deaths. Social bits.

### **7.8 INTERGENERATIONAL ASPECTS OF REPRODUCTION AND EMBRYONIC REQUIREMENTS**

Intergenerational aspects of health are now recognised as playing an important part in our lifelong wellbeing. Environmental factors have been related to increased risk of birth defects in offspring (at a population level) when mothers were exposed to nutritional deficiencies, severe illness, poor sanitation, and socio-cultural and/or economic deprivation during their own childhood or adolescence, even during their own pre-birth development (Emanuel & Sever, 1973; Barker, 1994). These environmental factors may all be associated with increased population density, particularly, but not exclusively, affecting the poorer members of society. The requirements for the normal development of the human foetus include sustained correct levels of temperature, oxygen tension, water and food, but also, crucially, the quality of food (Hahn, 1979). Of course, these all come to the foetus via the mother. Whilst the health of women during pregnancy has long been recognised, it is not until relatively recently that the importance of the pre-pregnant health status of women has been appreciated, for example in the folic acid - neural tube defect issue

(Smithells *et al.*, 1981). Studies have already shown that variation in maternal heat exposure at specific times during pregnancy have been associated with vertebral anomalies in mice and rats (Bennett, 1972; Kimmel *et al.*, 1993). It seems reasonable to suggest that such variables as maternal temperature may be implicated in the activities of genes as they act upon the developing embryo, both in terms of enhancing or impairing the action of the gene(s). This interaction between heat and embryonic development could be due to two different mechanisms: external heat exposure could lead to increased maternal cooling, which would lower the metabolic rate (and perhaps interfere with nutrient uptake by the foetus), whereas a raised temperature due to maternal infection would lead to an increased metabolic rate (which may interfere in other ways with foetal nutrient uptake). Nifuji *et al.* (1997) have shown that manipulation of the application of bone morphogenetic proteins (genetic materials) can have a modulating effect on somite development, resulting in vertebral anomalies. Maternal temperature would, of course, vary depending on the nature of the relevant pathogen(s) and on her nutritional, and therefore, immunological status. In conditions of high population density, ongoing infectious processes must have been a relatively normal state of affairs. Even a raised pathogen load resulting in a sub-clinical illness, when combined with poor nutrition, damp houses, polluted water and air, and parasite infestation, could produce either a reduced immune status in which the maternal body could not adequately fight infection effectively, or an immune response producing a raised temperature at a critical period of embryonic development.

## 7.9 POPULATION DENSITY, TERATOGENIC EXPOSURE AND NUTRIENT DEFICIENCIES

*"Our knowledge is still largely incomplete regarding the relationship between dietary factors and the major illnesses of our culture. These illnesses include not only cancer and heart disease, which have received most attention, but also congenital malformations, degenerative conditions of the eye, fractures, and many infectious diseases that are hypothesized to be influenced by the nutritional status of the host...our knowledge of many of these relationships, however, depends largely on observational epidemiologic data for the near future, and, for some relationships, indefinitely. For this reason, it is crucial to refine, maximally, our methods of data collection, analytic procedures, and interpretation of findings." (Willett, 1990:17), [italics mine].*

Various nutritional issues have been associated with birth defect frequencies. For example, Emanuel and Sever noted that there is evidence for an inverse relationship between central nervous system defects and the hardness of drinking water (1973b:327). They suggested that calcium may be involved in the equation, particularly in areas where most dietary calcium was taken from water. They also proposed an inverse relationship between serum zinc levels and central nervous system defects (1973b:329). However, some epidemiologists believe that it is "unlikely that useful measurements of the diets of individual subjects within free-living populations can be collected at all." (Willett, 1990:5).

Perhaps the principal reason behind this assertion is the almost universal exposure of everyone to almost all the ranges of nutrients. For example, everyone eats fibre, fats and some vitamins. The exposures cannot therefore be measured as either "present" or "absent", but must be considered as continuous variables. Perhaps the best subjects for an environmental/nutritional comparison are those members of a population who eat a specific diet for cultural or religious reasons (Willett, 1990:9). In archaeological populations it is even harder to assess dietary intake - we rely on documentary evidence, latrine pits, butchery remains, and seeds etc. for analysis of food habits.

Brothwell (1994) suggested that some developing world populations may be useful analogues for our past populations in the West. He suggested that several categories of variation may be studied by observing such groups, for example, urban-rural contrasts in stature differences, growth and nutrition patterns, changing cultural behaviour, and importantly, psychological stresses related to migration and cultural transition (Brothwell, 1994:129). Developing world populations, increasing numbers of whom are now living in rapidly urbanizing environments, may represent a type of "semi-urban" population, neither truly rural nor truly urban. Conditions in shanty towns, or in the poorest of urbanizing areas on the metaphorical edges of large cities, may be useful subjects for the study of epidemiological transition, as they are living in an urban environment with its associated overcrowding, poor sanitation, and raised potential for disease transmission, but practising a modifying rural existence in terms of diet and cultural behaviour. Such groups are arguably "on the epidemiological fence", in a transitional phase prior to their acculturation into the true city life (which may not occur for two or more generations).

In the light of the observed associations revealed by this study, it seems that people who, through their socio-economic or cultural status, find themselves living in situations of increased, or increasing, population density, probably associated with a reduced or inadequate standard of living, would appear to be at higher risk of producing children with skeletal birth defects. It is the suggestion of the author that maternal fever, associated with infectious episodes, may be acting as a direct or an indirect teratogen on the unborn

foetus. As infectious disease is more frequent among urban populations (Kiple, 1993, 1997), then those populations will express the highest rate of resulting birth defects. Layde *et al.* (1980) considered maternal fever to be a "fairly typical teratogen", but research into this aspect of aetiology does not seem to have been developed as much as other, apparently more "media-friendly" possible causes such as overhead powerlines and food scares. Perhaps too much effort is being put into investigation of the hazards of "modern life" at the expense of more prosaic factors, of which raised maternal temperature is but one. Recent studies have implicated raised temperature under laboratory conditions as a possible teratogen (Angles *et al.*, 1990; Hiramoto *et al.*, 1999; Kimmel *et al.*, 1993); such effects could be replicating the effects of maternal fever associated with infectious episodes. Perhaps the higher rates of birth defects observed in the urban populations of Chichester and York may be at least partially explained by the raised infection rates with which population density is associated (Kiple, 1993, 1997; Lewis *et al.*, 1995; Roberts & Manchester, 1995). It is the suggestion of the author that as non-specific infections are probably the most commonly-encountered pathogenic agents in daily life, that the non-specific infections are probably more to blame for this effect than specific infections such as tuberculosis and leprosy, although these would also have played a role in maternal fever. Medical and research literature already point, tentatively, to the role of infection in the aetiology of birth defects, although conclusions have been mixed (for example, Saxen, 1973 versus Knox and Lancashire, 1991, regarding influenza as a possible cause of defects), but studies have, understandably, focused on specific infections which

are more easily recognised and quantified. For example, organisms such as the Coxsackie viruses have been associated with birth defects and others such as varicella, treponemal disease, mumps and hepatitis have also been suspected as causative factors (Leven & Tudehope, 1993; Knox & Lancashire, 1991).

However, it is unclear whether the actual agents or the fever associated with them are the possible causes of the observed birth defects. From the nutritional perspective, vitamin A toxicity, insufficient folate, zinc and calcium uptake, and alcohol consumption are some of the variables which have been implicated in defect aetiology (Allen, 1986; Lowry, 1977; Yasuda & Poland, 1974). All of these nutritional variables may be economically-mediated, with poorer sections of society and those living in areas of high population density all being more likely to suffer such nutritional deficiencies. As immune status and nutritional status are inextricably linked (Chandra, 1980), such people are arguably at greater risk of defects in their offspring.

#### **7.9.i Environmental interference with physiological mechanisms**

In "normal" health, nutritional status and an effective immune response are involved in a reciprocal relationship, each directly or indirectly enabling the other to function competently. This is achieved by maintaining a balance within the body between the *absorption and use* of required nutrients and the *effective elimination of pathogenic threats* to the body. It is already recognised that this complex interaction may be disrupted by environmental factors acting on the human body in such a way as to compromise their typical healthy

association (Chandra, 1978, 1979, 1980; Scrimshaw, 2000), with quantifiable physiological effects being observed even in the offspring of affected mothers.

For instance, many pathogens or foreign materials enter the body through the alimentary canal and, among other mechanisms, these may be dealt with by secondary lymphoid tissue such as Peyer's patches found in the small intestine. The maintenance of Peyer's patches depends on the capability of the body to maintain a healthy ilial endothelium. If this lining of the small bowel is destroyed or seriously damaged, for instance, by helminth or bacterial infection, which archaeological evidence shows was common in the past (Kiple, 1997), the lymphoid tissue comprising Peyer's patches is lost or damaged, thus reducing the effectiveness of the immune response that they normally trigger. Even if Peyer's patches are preserved in reasonable condition, they rely on an *in situ* population of potentially active lymphocytes within them to react to the stimulation by antigens in the gut. If the production of these lymphocytes is in any way impaired, for instance, by defects of haemopoiesis (which, like all physiological processes, is reliant on adequate nutrition for its healthy perpetuation) or by a simple "overloading" of the system with pathogens, then Peyer's patches would not contain the necessary components with which to mount an effective immune response.

It is suggested by the author that the body's balancing capacity between *nutrition* and *infection control* is a key mechanism in the reproductive health of populations, and that this mechanism can be influenced significantly by environmental factors. Malnutrition and infection are synergistic (Golden,



1993:451), and the risks of infectious disease taking hold in the body rise in inverse proportion to nutritional status. Skin and internal mucosa become damaged and allow entry to micro-organisms, with reduced output of IgA, gastric acids and sweat, which usually offer some protection (Golden, 1993:450). The maintenance of an adequate and efficient lymphocyte population is required for the deployment of an effective immune response (Reeves & Todd, 1996). Secondary immunodeficiencies result from malnutrition, the impact of disease upon the body, impaired protein uptake, chronic inflammatory diseases, persistent infections, trauma such as burns, and skin diseases, amongst other processes (Reeves & Todd, 1996:193-5). All of these factors involve, or are closely related to, nutritional status.

Kagnoff, in considering modern American health, suggested that even mild to moderate defects in nutrition may have a significant effect on the immune response, particularly in children (1984:252). Immune function anomalies have been associated with deficiencies of certain substances. For example, reduced levels of zinc, vitamins A, C, and E, and of folic acid and pyridoxine, have been associated with reduced cell-mediated immunity (Kagnoff, 1984:253). Iron deficiency is also believed to have an adverse effect on host immunocompetence, as are thought to be deficiencies of copper, selenium and magnesium (Kagnoff, 1984:253). Kagnoff also pointed out that individuals with such deficiencies are often concurrently infected with bacterial or viral organisms which further reduce the immune response (1984:253).

One of the features of malnutrition or undernutrition is growth stunting (Golden, 1993), and evidence shows that this is associated with an increased risk of infection and general morbidity as well as increased mortality (Golden, 1993:444). For example, studies in developing countries have found associations between height and the number of diarrhoeal episodes in children under three years, and between malnutrition, growth reduction and pneumonia in Central and South America (Kuvibidila *et al.*, 1993:123; Martotell *et al.*, 1975). Diarrhoea may, of course be the precipitating factor in protein-energy malnutrition, but in past communities with few sanitation and hygiene regulations this must have been the normal state of affairs, particularly when parasitic infection was involved. The subsequent loss of vitamins, zinc and iron not only reduce host response but also growth rates (Kuvibidila *et al.*, 1993:124).

Generalized ill-health results from protein-energy malnutrition and produces increased rates of skin and mucosal lesions, which in turn allow entry to micro-organisms which cause more infection. In addition, chronic protein-energy malnutrition results in atrophy of lymphoid organs, probably from a reduced production of lymphocytes, thus reducing host response even more (Kuvibidila *et al.*, 1993:125). Malnourishment *per se* is not associated with reduced humoral immunity (immunoglobulins), but in certain specific conditions (see above) this can occur. In terms of proteins, the amino acids arginine and glutamine have an immunomodulatory role. Arginine supplementation in particular has been related to increased lymphocyte production including

proliferation of suppressor T-cells. It is also implicated in the limitation of bacterial infections (Kuvibidila *et al.*, 1993:147).

Protein-energy malnutrition is associated with underprivileged lower socioeconomic groups, and is common in the west in mild to moderate forms today. It is probable that the type and severity of growth and nutritional deficits experienced influence the degree of immunodeficiency that results. Statistics have shown that anthropometric data such as weight-for-age, weight-for-height, and arm circumference have a direct relationship with risk of death and/or severity of an infection (Kuvibidila *et al.*, 1993:123).

Modern prevalence of anaemia is highest in southeast Asia, with some countries having rates of 80% (WHO, 1997). Maternal anaemia is associated with increases in the incidence of low birth weight, anaemia and protein-energy malnutrition in infants (WHO 1997: 50-51). Hengen (1971) made a significant observation when he noted the relationship between living conditions and anaemia - he highlighted sanitary and living conditions that would enhance parasitic infection and disease. Diet alone is probably limited in its importance with regard to the absorption of iron. As iron is present in appreciable amounts in water, and in most foods, it is relatively easily available (Kiple, 1997). Gastro-intestinal disturbances are common, including nausea, vomiting, diarrhoea or constipation, and general loss of appetite (Stuart-Macadam, 1989). Such symptoms may themselves further exacerbate dietary insufficiencies.

Whilst iron is required by the body for general health, any excess amount carried in the body at a given point in time may be deleterious. Palmer and Coggan (1997) found that levels of iron present in the body affected bacterial virulence and host immune response. Free iron carried in body fluids promoted bacterial growth, and lead to greater risk of infection in exposed individuals, although this was possibly restricted to the periods in which the raised blood iron levels persisted. Signs of possible adaptation involving iron-uptake and personal health have already been noted in infants. Stuart-Macadam noted that infants between six and eighteen months old exhibit reduced blood iron levels. She suggested that this may be a response to the loss of maternal immunity experienced at this age, and an attempt to reduce risk of infection while their own immune systems develop and mature (Stuart-Macadam, 1989:214).

The majority of studies of maternal diet and its effects on the foetus have been carried out in the developed world, and others have compared immigrant populations with more well-off indigenous populations (Allen, 1986). Although results appear to show that there are significant differences between such groups, due to cultural and/or socio-economic reasons, only very little is known about the effects of diet on the offspring of mothers in the developing world (Allen, 1986:270). It is precisely these unassessed populations who are probably in greatest need of help, often migrating to shanty towns, or living in changing environments without the required health care to attend to their changing medical needs.

*Zinc deficiency*, particularly in early pregnancy, has been associated with increased risk of "pregnancy complications, congenital malformations and prolonged

gestation" (Allen, 1986:269). Women who are "marginally zinc-deficient prior to pregnancy onset" may be especially at risk (Allen, 1986:269). Sato *et al.* found that the degree of zinc deficiency was a factor in the co-teratogenic effect of other chemicals on the foetus (Sato *et al.*, 1985). Marginally zinc-deficient mice had a similar rate of defects to a normal control group, but cadmium added to these marginally deficient mice resulted in a significant rise in frequency of malformations. In severely zinc-deficient mice the addition of cadmium made no difference to the frequency of defects. Likewise, teratogenicity of excess Vitamin A was not enhanced in zinc deficiency (Sato *et al.*, 1985:13-18). Iron interferes with zinc uptake, and women eating a high-iron diet may therefore have low serum zinc levels (Allen, 1986:269). Allen also cited research that showed higher maternal plasma zinc was associated with low birth weight. Emanuel and Sever proposed zinc deficiency as a cause of ASB (anencephaly and *spina bifida*) (1973a; 1973b:329). Hurley also supported zinc deficiency as a cause of birth defects, albeit in conjunction with other predisposing factors (1974:205). Even in areas where zinc deficiency is common, it is still widely available in the diet, indicating that it is lost or not taken up sufficiently. Zinc may be lost in sweat, as well as in blood loss due to parasitic infection (Emanuel and Sever, 1973a:117). Sato *et al.* concluded that "adequate maternal zinc level is essential for a defense mechanism against cadmium as well as other teratogens" (Stao *et al.*, 1985:17).

*Zinc, copper and magnesium* deficiency have been associated in rats with chromosomal abnormalities (Bell *et al.*, 1975). Deficiencies in maternal tissues were associated with a variety of congenital malformations in organ systems as well as chromosome abnormalities. The authors concluded that "nutritional deficiencies may

alter the mitotic activity needed for development, or cause continuous cell death and thus affect organogenesis” (Bell *et al.*, 1975: 225).

*Iron deficiency* is a common effect of pregnancy. Rates of 22% and 88% in Latin American and Indian pregnant women respectively have been recorded (Allen, 1986:268). Low haemoglobins have been associated with a variety of anomalies including low birth weight, medical complications and prematurity (Allen, 1986:268).

*Calcium deficiency* is related to toxemia (high blood pressure associated with pregnancy). Emanuel and Sever noted that calcium intake from a poor diet was largely dependent on the hardness of local drinking water (1973b:327). They suggested that calcium may be a “growth factor” in water, and that low intake /absorption resulting in maternal smallness could influence reproductive health and possibly be related to low-birth-weight, raised foetal mortality and increased rates of central nervous system defects (1973:327).

*Vitamin B6 deficiency* affects women in developed and developing countries. Allen states that half of American women eat less than 50% of the RDA of Vitamin B6 (1986:270). Women with low plasma B6 had babies with low Apgar scores (a scoring system used to assess the general condition of babies at one minute and five minutes after birth), and also had low B6 levels in their milk (Allen, 1986:270). Apgar scores rate type of breathing, colour, heart rate, muscle tone and response to stimuli, thus low scores indicate less-than-desired standards in one or more of these aspects.

*Folic acid deficiency* results in megaloblastic anaemia in “a significant number” of pregnant women in developed countries (Allen, 1986:270). Folate deficiency is associated with an increase in the frequency of neural tube defects, and also with shortened gestation (Allen, 1986:270).

Since the early 1970's research has shown that maternal nutrition affects the outcome of pregnancy (Allen, 1986:265). Investigation into the relationship between maternal health and the health of the offspring in later life is now a feature of medical research (Barker, 1998), and its implications may well affect the study of archaeological populations in the future. Low birth weight babies (under 2500g) may result from chronically, that is long-term, malnourished mothers, and congenital malformations may also be linked to maternal diet (Passmore & Eastwood, 1986:575-6). Studies have shown that the nutritional state of women prior to becoming pregnant is “as important a predictor of birth weight as diet during pregnancy” (Allen, 1986:265). Low birth weight is associated with higher than average risks of congenital abnormalities (Czeizel, *et al.*, 1994) and immune-response deficiencies (Chandra 1975). Pre-natal development is recognised to be influenced by various factors including the biology, environment, and socio-economic status of the mother, all of which are represented to the foetus through her nutritional status (Saunders & Hoppa, 1993:139). Women suffering from chronic low-grade infection and parasitic infestation of the intestinal tract, for example, will be nutritionally impaired, even if their dietary intake is adequate (Jaffe *et al.*, 1976). Inadequate uptake of micronutrients such as iron, zinc, calcium, folic acid, and Vitamin B6 are all linked to increased risk of foetal abnormalities ( Jaffe *et al.*, 1976; Allen, 1986:267-70).

Conversely, dietary excesses may also cause problems: hypervitaminoses, especially of vitamin A, are suspected human teratogens (Hogue, 1984:47).

#### **7.10 EPIDEMIOLOGICAL PERSPECTIVES ON BIRTH DEFECT AETIOLOGY**

By applying the biocultural approach to human skeletal remains, it has been possible to show the effects of environmental factors acting on the development of the axial skeleton in a way which would have probably been impossible to do using living populations. The popular approach to investigation of birth defect aetiology still focuses largely on occupational exposures, or proximity to radiation. Of course, these can also be heavily culturally dependent, for example, the poorer members of society tend to work in factories, processing plants, or similar, and are likely to live in inner-city, run-down areas. In this way, exposures tend to affect specific socio-economic groups, with professional-grade families tending to be affected by different environmental and cultural factors.

Perhaps too great an emphasis has been placed so far on exposure to chemicals, radiation and drugs. Of course, these are recognised to have potentially lethal effects on the foetus, and must be controlled adequately in the interests of society. However, this study has shown that more subtle environmental effects may also have a significant role in defect aetiology, as has been suggested before (Goldsmith, 1986).

For example, Saxen (1983) and Garcia & Fletcher (1998) found significant associations between maternal employment in the leather and fur industries,



but failed to consider the implications of infection from untreated pelts and hides rather than the chemical processes involved in tanning and treating the leather. The urban populations of York and Chichester showed a higher risk of skeletal birth defects than their rural counterparts. It does not seem reasonable to believe that these populations were *all* subject to chemical exposures or other factors which are today commonly associated with defect risk. On the other hand, infection levels are arguably the most obvious variable affecting whole populations in differing environments, and must now be considered as a subject of further investigation.

#### **7.11 GENETICS AND ENVIRONMENTAL FACTORS**

The homeobox genes are recognised as the “blueprint” for vertebral formation, instigating the development of vertebral elements with the appropriate segmental characteristics (Weiss, 1993). Homeobox genes, upon the onset of some trigger action, produce proteins which “tell” somitic cells which type of vertebral element they are to develop into (Weiss, 1993). Variations in this mechanism have been recognised as causing developmental defects in vertebral formation, for example, producing a border shift by causing elements to take on the characteristics of the adjacent segment (Saegusa, 1996).

Somitic cells are unable to trigger the action of Hox genes without the help of *morphogens* or *Hox gene enhancers/promoters*. These morphogens are not yet all identified, and neither is their origin or action fully understood. However, it is known that they act in the area of the cells where *morphogen receptors* are located, and that they bind to these receptors in the cells. From this it is

possible to see that there are various loci in this mechanism where problems could occur:

- The morphogen itself may be absent or faulty
- The receptor may be absent or faulty
- The somitic cell may be faulty
- The Hox gene itself may be faulty
- The conditions required for the effective action of morphogen and receptor may not be present or correct
- A teratogen may mimic the morphogen and trigger Hox at the wrong time
- A teratogen may bind a morphogen receptor and block the action of a morphogen at the appropriate time.

With regard to the point on conditions required for the effective action of morphogens and receptors, Bennett (1972) found that heat variation induced vertebral formation anomalies in the offspring of pregnant mice, and more recently, Nifuji *et al.* (1997) investigated the action of *bone morphogenetic proteins* (BMP) on the development of the axial skeleton. They observed that the addition of BMP to the dorsal regions of chick embryos during somitogenesis (development of the somites) resulted in morphological anomalies in the dorsal areas of the vertebral elements, and ribs. They concluded that BMP signalling (between genes and cell-based receptors) is involved in the early development of the axial skeleton, particularly affecting the dorsal regions of the vertebrae (Nifuji *et al.*, 1997:340). This is borne out by the high rates of neural arch defects which were observed among

archaeological human populations, and the relative lack of anomalies in the vertebral bodies.

Looking at the possible causes of disruption outlined above, one may consider the environmental factors which affected the populations observed in this study. There must have been various factors at work in the Medieval period which could have had an effect on the developing embryo via the mother's environment. For example, these could include seasonal food fluctuation (in type, amount and in quality), pathogens related to variable food storage and preparation, domestic and industrial/craft related exposures to chemicals, dust or fumes, not to mention the hazards of open sewers, and damp, smokey houses. All of these could be the cause of some perturbation in the signalling/trigger mechanism between genes and receptors in the developmental stage of the early axial skeleton.

The results obtained from the populations in this study showed significant variation in patterns of border shifting. There were also variations in *type* of border shifting, with some populations tending to express more sacralisation than lumbarisation, for example. Lumbarisation appears to have been particularly different between urban and rural sites, although sacralisation differences were noted only between certain individual sites. Why should this be is uncertain.

Perhaps certain disruptive factors produce specific morphological defects due to their action on morphogens/receptors. Also, there appears to be a sex-based

issue, with males tending to be affected more frequently than females at most sites, raising questions about sex-mediated sensitivity. Yet, at Wharram Percy, this was reversed, and in addition to this reversal, the Wharram females had the highest rate of sacralisation among *all* the observed females. Was there some specific local factor at work at Wharram that produced this phenomenon? There may, of course, be some straightforward genetic answer to this question.

The results suggest that certain environmental factors produce certain types of shifting. Whilst the *timing* of the perturbation in signalling/triggering may well have a role, these results show that environmental factors (urban or rural) do seem to be having some sort of quantifiable effect at a population level.

Whether males are more susceptible to these effects than females is unclear. It may be that males are simply more successful at surviving to birth than females are, following exposure and developmental disruption. However, this would not necessarily explain the pattern at Wharram, in which females were more commonly affected than males.

## 7.12 SUMMARY OF DISCUSSION

- Nutritional variation in terms of quantity and quality of maternal diet both prior to and during pregnancy have been implicated in the frequency and type of birth defects in the medical literature
- The efficiency of the immune response is closely associated with nutritional status, and a reduction in quality and/or quantity of diet will

impair the immune response rendering the individual more susceptible to infection

- Increased population density is associated with an increase in disease rates, including infectious diseases/episodes
- Urban populations, living in conditions of higher population density, are more exposed to pathogenic agents than rural populations in terms of their overcrowded housing and sanitary arrangements, as well as their exposure to open sewers, polluted water and adulterated food and drink and other hazards of raised population density
- In addition, psychological stress, such as experienced by those removed from the support of family and kinship networks, has been shown to reduce the strength of the immune response
- Observations on five English Medieval populations in this study have shown that the York and Chichester urban populations had higher rates of axial skeletal birth defects than did the Raunds Furnells and Wharram Percy rural populations; the Hull population is unusual in respect of the stress indicator prevalence and could possibly be regarded as anomalous in terms of urban/rural defect prevalence as there may be some burial pattern

occurring there which does not reflect the local population (further research into the Hull population by other workers may elucidate this situation)

- the author suggests that the observed higher urban rates of birth defects are related to the increased population density in the two cities, and largely resulted from generalized nutritional and immunological deficiencies in those populations when compared with the rural populations
- the author suggests that the observed birth defects result from either the teratogenic action of the active pathogenic agents themselves, or the teratogenic action of maternal fever when infectious episodes occurred during organogenesis, or some nutritionally-related deficiency caused by impairment of maternal immune/nutritional status
- the differences in patterns observed in defects originating in the same developmental field, for example, between lumbarisation and sacralisation, may be due to some unrecognised environmentally-mediated genetic behaviour, perhaps affecting the expression of genes (for example, the homeobox genes)
- apparent changes in frequency over time, for example, with the rates of numerical variation, may be an adaptive reaction as a biological response to a changing environment (e.g. increasing urbanisation of society)

- age-at-death profiles appear, in the case of some defects, for example, sacralisation, to reflect the presence/absence of the defect, suggesting that the defects or associated soft-tissue anomalies may have some effect on longevity
- stress indicators appear to be more likely to occur in an individual who has also got a birth defect of the axial skeleton, suggesting that there are some other health-mediating issues present than simply a bone defect
- sex-based frequency of defects shows that males were more commonly affected than females.

## CHAPTER EIGHT

### Conclusions

#### 8.0 THE HYPOTHESIS AND THE RESULTS

The results have supported the hypothesis, namely, that urban populations appear to have a higher risk than their rural counterparts of suffering birth defects of the axial skeleton. Overall, the populations of the Chichester hospital/almshouse, and of St Helen-on-the-Walls, York, had the highest prevalence of defects. The Anglo-Saxon, early medieval population of Raunds had the lowest prevalence and the agricultural community of late Medieval Wharram Percy had a slightly higher rate. The Augustinian Friary site had a pattern that mirrors the rural sites, falling between Raunds and Wharram in terms of mean prevalence. There are statistically significant differences between urban and rural populations in the prevalence of the majority of defects observed, and in many cases, between males and females. The anomalous pattern observed at Hull is difficult to interpret. As mentioned in Chapter 7, there may be a different pattern of burial occurring at the Hull site, making it an unrepresentative cemetery (as far as investigation of the urban/rural theme is concerned). As there appears to be a relatively clear urban/rural divide between Chichester and York (53.5% and 58.5% mean affected population respectively) on the one hand and Raunds and Wharram (39% and 43.5% respectively) on the other, the apparent rural pattern observed at Hull (42.5%) has to raise questions about who was granted burial there. This is beyond the scope of this study, but warrants further investigation.



## 8.1 MECHANISMS IN BIRTH DEFECTS

Congenital defects of the skeleton may actually be an *adaptation* to environmental changes. For example, restricted blood flow to the foetus is an adaptation to maternal undernutrition - it makes the foetal body smaller, but the head and the brain are unaffected (Barker, 1994:128). Are we therefore seeing a similar adaptation of the body when vertebral defects occur? Such adaptation may occur in response to an effective lack of nutrients (specific or non-specific), protein-energy malnutrition, and/or to a reduced immune response, allowing pathogens to act on the body relatively unchallenged. However, the key issue regarding biological responses may be simply that of *time*. Do populations react differently to *sudden* or *gradual* changes of environment? For example, biological acclimatization involves modification of mechanisms such as the immune response. Observed biological reactions must result from the innate ability of such mechanisms to respond to environmental stimuli, be it in a beneficial or a detrimental manner. Arguably, gradual changes may produce fewer, or at least different, effects than sudden changes, which may initially result in the deaths of large numbers of people. Such occurrences are well documented, for example the decimation of native Americans post-1492 (Crosby, 1972). However, work following up the surviving remnants of such populations has been noticeably lacking. How did they adapt in the long term to accommodate the new environment? By observing birth defect frequencies of groups drawn from *within* the population of Medieval England, but who were *subject to* different environments (rural and urban, both pre-industrial), it is suggested that biological responses will be seen to have been modified in the long term (Bogin, 1988; Coleman, 1995; Sever, 1995). This would require examination of a well-excavated and/or well-documented cemetery in which early, middle and late

time periods could be identified. Statistical analysis could then be applied to the observed data set and comparison through time achieved. The author suggests that populations in demographic transition take time to immunologically adapt to their new environments. In the case of migration, this could include the migrants and the host population. Such adaptation is already documented by other authorities (Bogin, 1988; Coleman, 1995), but usually focuses on epidemiological changes in diseases such as cancer. The results of this study suggest that the prevalence of birth defects of the axial skeleton may be used as an indicator of the degree to which a population is in equilibrium with its environment, in terms of an efficient balance between nutritional status and immune response. Once pathogen load and/or infection rates significantly increase, as a function of rising population density, then any such equilibrium may be disturbed, triggering adverse biological responses such as an increase in the risk of skeletal birth defects. In addition, the medical literature shows that many skeletal defects are associated with soft tissue anomalies of varying degrees of severity, which may be considered by biological anthropologists when trying to reconstruct past health patterns and particularly disability.

## **8.2 THE NUTRITION-POPULATION DENSITY-INFECTIOUS TRIAD**

The medical literature clearly indicates the hazards of disruption to the nutrition/immune status equilibrium (for example, Chandra, 1978, 1979, 1980; Reeves & Todd, 1996; Scrimshaw, 2000), and these are all associated with an increase in population density (Roberts & Manchester, 1995). Living conditions including the domestic and occupational environments have been shown to be associated with certain health issues, such as respiratory disease, and exposure to poisonous agents

(see Lewis *et al.*, 1995; Palmer & Coggan, 1997), all of which would have affected individuals in Medieval England. In addition, and this has not been adequately appreciated to date, the psychological aspects of health have been recently shown to have a significant effect on the immune response (see Ursin, 1994), which the study of psychoneuroimmunology is only just beginning to elucidate. The health-mediating effects of psychological stresses on populations undergoing transition, or in conditions of separation from family and traditional kinship networks, must not be underestimated.

Deficiencies in diet in the past would have commonly included reduced micro-nutrient intake or uptake, often on a fluctuating seasonal basis, with foodstuffs being unavailable or ineffective as nutrient sources once they were past their best or all consumed. Over-cooking of food would also have resulted in micro-nutrient deficiencies as vitamins were leached into cooking water, and poor storage or preservation techniques would also have resulted in loss of nutritional value. As well as vitamin and mineral deficits, protein-energy malnutrition would also have been a seasonal or even semi-permanent phenomenon, particularly in the spring and early summer when agricultural communities have to expend huge amounts of energy on the land to ensure a good autumn harvest. These effects would have been particularly harmful when combined with extant immune deficiencies, and the hazards of increased population density experienced in towns. In addition, the consumption of various non-foods in the diet, such as illegal additives, residues of preserving techniques, pottery glazes, over-salting, and the taking of "quack" medicines and, unwittingly, other poisons, would have had a significant role in the reduction of both nutritional and

immune states, as well as those agents possibly acting as teratogens at embryonic critical periods. Further evidence of nutritional deficits is found in the generally reduced nature of adult stature in urban groups, particularly as evidenced at York. The higher mean stature of the Chichester population probably may not indicate a better diet or greater nutritional efficiency, but rather a focus of the sick of the area, who were suffering because of having reached something approaching their genetic potential height. The alternative explanation of the greater mean stature at Chichester may be as discussed in Chapter 7, that is, due to the inmates not being the poorest sick, but simply the sick who could afford to pay for their residence in the hospital/almshouse and who had not suffered undue nutritional deficiencies during their growing years.

Overall, the author suggests that rural populations were subject to the ill-effects of seasonal or economically-related nutritional deprivation or impairment, but that urban groups suffered similar nutritional deficits *in the context of relatively greater population density*. Together, those impairments of nutritional and immune status rendered urban populations at greater risk of suffering more frequent and/or more severe infections, along with greater exposure to a wider variety of pathogens, and the carrying of a greater pathogen load. Through this reduction in their health status, individuals were unable to adequately fight against the effects of this raised pathogen load. A raised frequency of birth defects in urban populations thus resulted from either:

- unchallenged infectious agents acting, directly or indirectly, as teratogens on unborn babies,

- or maternal fever associated with more frequent infectious episodes acted as a teratogen
- or the reduction in micronutrients and other food requirements had a teratogenic effect on such babies.

### 8.3 EPIDEMIOLOGY AND THE BIOCULTURAL APPROACH

The epidemiological approach has been shown to be compatible with the study of archaeologically-derived human material. Much information has been obtained through a relatively simple, non-destructive study of axial skeletal birth defects. By combining the epidemiological, biocultural, morphogenetic and clinical diagnostic approaches, this study has shown that amongst English medieval populations, birth defect prevalence can be associated with environmental variations. Given the continuum-like nature of teratogenic effects on the human body (within and around critical periods), and the medically-defined biological responses to stimuli, the defects observed in this study are highly likely to also be present in modern-day populations in similar frequencies. This is probably especially true of populations in transition. For example, those populations experiencing urbanisation in developing countries, and those groups moving to the West, as well as their host populations in the area of destination, must be at greater risk of adverse birth outcomes than their cultural neighbours who remain in the environment to which they are adapted. Migrant studies are useful in addressing the possibility that correlations observed in ecological studies are due to genetic factors. We already know that migrant populations “migrating from an area with its own pattern of cancer incidence acquire rates characteristic of their new location” (Willett, 1990:9). Therefore genetic factors cannot be primarily responsible for the large-scale differences in cancer rates among the observed

countries. Some migrant adaptation rates only occur in later generations, especially cancer types. This may be what is happening with high urban rates of birth defects observed in this study.

This raises some important questions. For example, what effect is the vaccination of immigrants having on those populations? As many females arrive in the West with a view to starting a family, what is being done to their immune systems by suddenly loading them with unfamiliar pathogens? Of course, vaccination is a vital disease-prevention measure, and cannot be abandoned, but it may be that an adverse effect occurs when such women then have babies before they are immunologically adapted to their new environment. Also, what of the effects on the host population? May they not also be exposed to unfamiliar pathogens from incoming groups? Obviously, these are extremely sensitive and potentially explosive cultural and political, as well as health, issues. They would require highly delicate handling and investigation, but this may be an area that requires research and some preventative action in the future, particularly with today's trend towards the globalisation of many aspects of life.

The epidemiological approach is essentially the application of the biocultural approach to modern, living populations. There is, however, arguably, a greater emphasis and scope in data collection and an increased element of interpretation of the data, in the epidemiological approach. This has been particularly appropriate to the study of birth defects and their aetiology. If biological anthropologists are to effectively "sell" their work to health research foundations, thereby utilising the wealth of information to be gleaned from extant sample populations from archaeological contexts, they will

probably find their task easier if they refer to the epidemiological aspects of that work. It is to be hoped that as biological anthropologists increasingly relate their published work to the medical and epidemiological literature, medical practitioners will become more aware of the need to share information about skeletal anomalies which they themselves consider insignificant (e.g. vertebral anomalies, which are rarely mentioned, or are not specified). This would of course assist biological anthropologists greatly in their own research areas.

#### 8.4 DISABILITY IN THE PAST

Overall, with regard to disability, this study has shown that congenital anomalies of the axial skeleton may be used as a signpost indicating the likely presence of some moderate to severe soft tissue involvement. This is particularly useful as many of these secondary effects are usually invisible to the osteologist. Among the vertebral defects observed, several have been associated in the medical literature with significant lower back problems, and potentially serious disruption of nerve supply to the arms or legs (see, for example, Castellvi *et al*, 1984; Pizzutillo *et al*, 1994). Spondylolysis, transitional vertebrae, and block vertebrae all contribute to pain-inducing conditions that can produce secondary osteo-arthritis, scoliosis, kyphosis, and reduced or impaired mobility and dexterity. Clearly, such conditions could have had a significant effect on the work capability, and therefore, the economic outlook for affected individuals and their dependants. As the greatest frequencies of these conditions were observed among the urban populations, one would expect this to be the socio-economic environment most affected. Unfortunately, the socio-economic prospects for affected individuals living and working in an urban setting, possibly removed from

family networks or support, would have been perhaps even more problematic than for rural individuals. In addition, if, for example, a carpenter were affected, and could not work, then there would be plenty of others in the market ready to take on the work and displace him (although this situation could have been alleviated by various guild practices, including provision of charitable support and control of work practices in some towns and cities). At least in a rural, village setting, affected individuals would have had a greater chance of being helped in a *quid pro quo* manner, with domestic/farm work shared out according to ability. Such considerations may explain the high concentration of such defects at the hospital/almshouse in Chichester.

An important issue raised by the observation of cleft defects in this study is probably the question of society's attitudes towards the "imperfect" individuals. Visible facial deformities were observed among the Medieval populations, suggesting an element of acceptance and/or tolerance in the past. Affected infants were obviously not all subject to an early death due to neglect or direct infanticide. On the other hand, no sub-mucosal clefts were observed. These would have been invisible. Their apparent absence suggests that affected infants did not stand a high chance of survival to childhood. This probably would be due to the associated infections that are likely to have struck them, which, combined with reduced nutritional status, would have been frequently life-threatening. With regard to the issue of infanticide, the author still holds the view that this would have been a relatively common practice in Medieval England, not least as a form of family-size control in times of stress (see Razi, 1980).

## **8.5 APPLICATIONS TO HEALTH CARE DEVELOPMENT**



The findings of this study may be applied to populations undergoing demographic transition today. Not only are millions of people in the developing world affected by the environmental changes associated with increasing urbanisation, but also, immigrants arriving in the developing world are exposing themselves to environments to which they are immunologically unadapted. Likewise, agencies specialising in emergency relief routinely encounter populations undergoing acute transition, who need many health care initiatives to ensure their long term as well as immediate wellbeing. Whilst prevention of birth defects may seem a relatively unimportant issue in emergency situations, the long term benefits of such planning will still have a significant socio-economic impact. This study has effectively shown that the epidemiological approach can be applied to archaeologically-derived human material, and that meaningful results may be obtained from such study. It is to be hoped that this approach will be accepted by other authorities, particularly in the field of medical research and health care planning, as excavated human material clearly has something to offer outside the field of archaeological research.

## 8.6 CURRENT TRENDS IN RESEARCH

Despite the current focus on exogenous agents as causes of congenital anomalies, Hahn's outline (1979) of the requirements for normal foetal development probably should be the environmental benchmark against which the majority of adverse birth outcomes should be considered. The issue of heat application to the foetus (Bennett, 1972; Kimmel *et al*, 1993) is particularly interesting, as it would have been a significant issue in populations affected by increased rates of maternal infection, with the accompanying fever associated with many of these episodes. When considered

alongside the findings of Nifuji *et al* (1997) in which the timing of Bone Morphogenetic Protein activity on the developing axial skeleton was shown to have detrimental effects if disrupted, it may be that variation in maternal temperature may act as a teratogenic effect at critical periods, or may influence the timing of BMP activity, producing vertebral anomalies such as those observed in this study.

### 8.7 FURTHER RESEARCH

Further research into teratological aspects of human health is warranted. Apart from the cost in human suffering and impairment, the economic costs of such outcomes are huge, both to the health and the social welfare services. Further identification of teratogenic agents and endogenous factors related to birth defects frequency will offer the prospect of improved health for people in both developing and developed countries. It seems likely that there will continue to be an increase in congenital defects world-wide, as modern treatment enables the survival of many affected individuals today who would not have lived long enough to reproduce in the past. Such individuals who carry genes altered by teratogens may then pass them on to their offspring (Nishimura, 1983). Appropriate research may help to prevent or reduce levels of physical impairment, enabling individuals who would have otherwise been affected by birth defects to have a quality of life, and a social and economic potential which would be seriously compromised if this issue remains unaddressed. Such research may include simple investigations. For example, as the nutrition/infection/immune response triad is so closely interwoven, treatment of one aspect may benefit the others. For example, diarrhoeal disease among transitional populations in the developing world, as a result of parasitic infestation, recurrent gastro-intestinal infection or contaminated water, may be implicated in a rise in birth

defects similar to those seen in the Medieval populations observed in this study.

Simple rehydration treatments with sugar and salt-based mixtures such as Dioralyte, if taken regularly, may actually have a significant effect in reducing the imbalance between the nutrition/infection/immune response triad, which may help to reduce rates of birth defects in babies conceived within those populations.

Apart from this aspect, several other avenues of further investigation could be pursued:

- Assessment of changes in defect prevalence over time, within an excavated population, to look for signs of adaptation to a changing environment;
- identification of tethered cord syndrome in juvenile cases of cleft sacrum through metric analysis of the legs looking for muscle wasting. This would enable a more accurate calculation of past rates of neural involvement in *spina bifida occulta*;
- identification of intervertebral disc problems in cases of transitional vertebrae and spondylolysis by analysis of osteoarthritis and Schmorl's nodes patterns in the vertebral column;
- identification of same by metric analysis of femora/tibiae/fibulae, to possibly identify sciatic involvement;
- identification of arm/hand wasting or hypoplasia in cases of cervical fused vertebrae by metric analysis of arms;
- identification of cleft palate/lip using the diagnostic criteria of Barnes (1994), as lack of understanding of cleft defects among biological anthropologists has probably led to cases being missed in collections;

- analysis of spondylolysis within archaeologically-derived samples, in order to define the likely aetiology;
- further analysis of stress indicators and stature variations to investigate the possible relationship between stature and susceptibility to environmental stresses
- the epidemiological/biocultural approach could be applied to the investigation of other diseases which can be identified on dry bone, for example, the analysis of osteo-arthritic changes in urban/rural contexts.

### **8.8 TO SUMMARISE**

Finally, the analytical study of birth defects among urban and rural populations has shown the potential value of archaeologically-derived human material as a tool in epidemiological research. Review of the medical literature has shown several soft tissue anomalies to be associated with skeletal defects, which are invisible to biological anthropologists. This has helped to form some ideas of related disability/impairment among affected populations in the past. Rather than simply presenting interesting case studies, (which do have a legitimate place in the research field), it has been shown that the epidemiological/biocultural approach can offer significant insight into the reproductive health of past populations, particularly in the long-neglected field of birth defects.

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## Appendix 1

## GLOSSARY

Definitions of medical terms stated below are taken from E.A. Martin (ed) (1994), *The Oxford Concise Medical Dictionary*.

<i>Blastemal</i>	of the blastemal stage, the earliest, pre-cartilage stage of embryonic development
<i>Blastocyst</i>	one of the earliest developmental stages after fertilization of the ovum, in which a hollow ball of cells forms; the inner mass develops into the actual embryo; the blastocyst implants in the uterine wall
<i>Biocultural</i>	to do with people in their social, technological, political, religious and economic contexts (i.e. any context which may affect their biological responses)
<i>Bone Morphogenetic Proteins (BMP)</i>	proteins involved in the development of the structure and form of bone material
<i>Branchial arch/pouch</i>	also known as pharyngeal arch/pouch; any of the paired and segmented ridges of tissue at the throat level of the embryo, which look like fish gills; each arch contains a cartilage, a cranial nerve and a blood vessel, which go on to develop into specific structures of the head and neck; between the branchial arches are the branchial pouches
<i>Central nervous system (CNS)</i>	the brain and the spinal cord
<i>Congenital</i>	present at birth; arising from a problem which was present at birth
<i>Congenital heart disease (CHD)</i>	defect(s) of the heart, often including defects such as hole-in-the-heart, or problems with valves or major blood vessels passing in and out of the heart.
<i>Cytomegalovirus (CMV)</i>	a member of the herpes group of viruses, causing cold-like symptoms; in individuals with a reduced immune status it may cause more severe illness; congenital defects may be found in infants if the mother contracts the virus during pregnancy

<i>Ecology</i>	the study of the relationship between people, plants, animals and environment, and how they may affect each other; the context in which people, plants and animals live together
<i>Embryo</i>	the products of conception within the uterus up until the 8 <sup>th</sup> week of gestation, in which time all the major organs are formed
<i>Epidemiology</i>	the study of disease processes and patterns within and between populations, with a view to finding causal relationships and associations with environmental factors; often with the aim of improving preventative planning
<i>Eustachian tube</i>	the tube connecting the oral cavity with the middle ear
<i>Foetus</i>	the unborn child from the 8 <sup>th</sup> week of gestation until birth
<i>Hyperplasia</i>	over-development of a body element by an increase in the number of normal cells forming it
<i>Hypoplasia</i>	under-development of a body element by a reduction in the number of normal cells forming it
<i>Hypospadias</i>	a urethral opening under the penis shaft, instead of at the end of the glans
<i>Idiopathic</i>	of unknown aetiology
<i>Inguinal hernia</i>	a herniation (extrusion) of the bowel through the site of the inguinal canal in males (the route by which the testes descend into the scrotum)
<i>Kyphosis</i>	an abnormal outward-bending of the spine causing a hunched-back appearance
<i>Kypho-scoliosis</i>	a combination of both scoliosis and kyphosis in the same individual
<i>Meiosis</i>	the process of cell division prior to the formation of ova and sperm, in which daughter cells are produced, each with half of the chromosomes of the original cell; the normal number of chromosomes is restored after fertilization; meiosis is the stage at which "crossing over" occurs, in which chromosomal material is exchanged between cells
<i>Meninges</i>	the three connective tissue membranes lining the skull and the vertebral (neural) canal, protecting the brain and the spinal cord; the inner layer is the <i>pia mater</i> , the middle layer the <i>arachnoid mater</i> and the outer layer, the <i>dura mater</i>



<i>Meningocele</i>	the protrusion of the meninges through a cleft in the vertebral arch in cases of spina bifida, covered with a layer of skin; may be associated with some weakening of the legs, and in tethered cord syndrome, with paralysis of the bladder and loss of use of the legs as the individual reaches adolescence
<i>Mesoderm</i>	embryonic tissue forming the middle germ layer of the embryo, out of which develop the cartilage, bone, muscle, blood, kidneys, testes, ovaries and connective tissues
<i>Mitosis</i>	the process of cell division by which one cell produces two identical daughter cells, for both body growth and repair
<i>Morphogenetic</i>	to do with the development of the structure and form of body parts
<i>Mutation</i>	an abnormal change in the genetic material of a cell, or the effects this causes in the characteristics of an individual
<i>Myelomeningocele</i>	the protrusion of the spinal cord and nerve roots through a vertebral cleft in spina bifida cystica; associated with paralysis of the legs and urinary incontinence
<i>Neural crest</i>	two bands of tissue which lie on either side of the neural plate in the embryo; the cells eventually become nerve cells of the autonomic nervous system
<i>Neural plate</i>	a strip of cells lying along the axis of the early embryo which develops into the neural tube and subsequently the central nervous system
<i>Neural tube</i>	the primitive structure from which the brain and the spinal cord develop
<i>Neural tube defect (NTD)</i>	a group of developmental defects resulting from failure in the normal development of the neural tube; includes spina bifida
<i>Neurological</i>	to do with the nerve supply to an element of the body
<i>Notochord</i>	a line of mesodermal tissue that lies along the back of the embryo below the neural tube; it disappears on formation of the vertebral column, and parts persist in the intervertebral disc tissue of children.

<i>Otitis media with exudate (OME)</i>	a middle ear infection, often associated with cleft defects at the back of the mouth, as infection enters the ear through the eustachian tube. Often associated with hearing loss if untreated.
<i>Paraxial mesoderm</i>	the cells which form the blastemal vertebral column around the notochord in the third and fourth weeks of gestation, starting cranially and progressing caudally
<i>Radiculopathy</i>	inflammation and/or irritation of the spinal nerve roots at the point of exit from the vertebral column, resulting in pain along the nerve pathway
<i>Renal</i>	to do with the kidneys
<i>Rubella</i>	German measles
<i>Sacral agenesis</i>	congenital absence of the sacrum, either in total, or partial
<i>Sclerotome</i>	the part of the segmented mesoderm which develops into the skeletal tissues
<i>Scoliosis</i>	an abnormal lateral bending of the spine
<i>Spasticity</i>	resistance to passive movement in a limb, usually accompanied by weakness in the affected limb, often resulting from injury or nerve supply impairment
<i>Spondylolysis</i>	a weakness, crack or break in the <i>pars interarticularis</i> of the vertebral arch, usually affecting the 4 <sup>th</sup> or 5 <sup>th</sup> lumbar vertebrae; may be bilateral or unilateral
<i>Spondylolisthesis</i>	a forward slippage of one vertebra over that below it, usually caused by the upper vertebra being affected by spondylolysis; may cause neurological symptoms and/or pain if the slippage is severe
<i>Talipes</i>	club foot, a deformity, in which all the elements of the foot are in place, but pressure of space <i>in utero</i> has caused the foot to form in an unnatural position
<i>Tarsal</i>	of the foot; or the bones of the proximal part of the foot (forming the ankle)
<i>Tetraplegia</i>	also known as quadriplegia, a paralysis affecting all four limbs

- Toxoplasmosis* a disease of mammals and birds caused by *Toxoplasma gondii*; transmitted via undercooked meat, contaminated soil, or direct contact (especially dead cats or cat excrement); congenital toxoplasmosis is passed via the placenta and can cause blindness and/or mental retardation in the fetus
- Treponemal disease* infection caused by the spirochaete of the genus *Treponema*; *T. pallidum* causes syphilis, *T. carateum* causes pinta, and *T. Pertenu* causes yaws

