An assessment of metabolic bone disease in the skeletal remains of Chinese indentured mine labourers from the Witwatersrand

by

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DECLARATION

I declare that the dissertation that I am hereby submitting to the University of Pretoria for the MSc degree in Anatomy is my own work and that I never before have submitted it to any other tertiary institution for any degree.

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Anja Meyer

This day 28 of the month January 2014

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ABSTRACT

An essential part of bioarchaeology is the study of diet and nutrition and its effects on the general health of a person. Interpretation of nutritional and metabolic disease related pathologies often provide additional insight into the daily social and cultural practices of people. It is therefore also an essential part of understanding differences amongst past populations from archaeological contexts and provides an alternative means for cross referencing historical accounts.

In this study the skeletal remains of 36 Chinese indentured mine labourers, who worked and died on the Witwatersrand mines during the period AD 1904-1910, were assessed for any signs of metabolic or nutritionally related signs of disease. Historical information suggests that these indentured Chinese labourers came from poverty stricken communities in China where disease and malnutrition were often encountered. Once in South Africa they were again subjected to the harsh living and working conditions associated with mining. Analyses suggest that all 36 individuals were males between the ages of 16 and 45 years, with the majority being of young adult age (20-34 years). Pathology that could be observed included a high prevalence of nutrition-related changes and linear enamel hypoplasia which suggests that the Chinese miners had been subjected to long periods of malnutrition and illness throughout childhood continuing into adulthood. Nevertheless, a large proportion of lesions associated with malnutrition showed some degree of healing. A high frequency of traumatic lesions, specifically peri-mortem fractures, was observed and may have contributed to the death of many of the Chinese miners. It therefore seems that even though the healing of pathological lesions associated with malnutrition indicated a period of improved nutritional intake, possibly during their time on the Witwatersrand mines, the high prevalence of peri-mortem fractures attests to the hazardous working conditions associated with deep-level mining.

In order to aid in the interpretation of skeletal pathology associated with metabolic and nutritional diseases non-specific signs of disease observed in a cadaver skeletal sample with known causes of death (related to specific metabolic or nutritional diseases) were compared to pathology observed in the Chinese miners. This provided pathological patterns which enabled a better interpretation of the pathology observed in the Chinese skeletal remains.

Keywords: Beriberi, bioarchaeology, cadaver skeletal remains, Chinese indentured labour, deep-level mining, malnutrition, metabolic bone disease, nutritional deficiencies, palaeopathology, Witwatersrand mines

ABSTRAK

Die bestudering van dieet, voedingswaardes sowel as die effekte daarvan op die mens is 'n uiters noodsaaklike deel van bioargeologie. Die interpretasie van voedingsgebonde- en metaboliese siekte-verwante patologie kan insig verleen tot die daaglikse sosiale en kulturele aktiwiteite van die mens. Om hierdie rede is dit ook ‘n essensiële manier om populasie verskille in ‘n argeologiese konteks te verstaan en kan ook dien as ‘n alternatiewe metode om historiese inligting te verifiëer.

In hierdie studie was die skeletale oorskot van 36 Sjinese kontrak-mynwerkers, wat in die tydperk van 1904-1910 in die goudmyne van die Witwatersrand gewerk en daar gesterf het, geassesseer vir enige tekens van metaboliese of voedingsverwante siektes. Geskiedkundige bronne dui aan dat hierdie Sjinese kontrakwerkers van baie armoedige gemeenskappe in Sjina afkomstig was; waar siekte en wanvoeding aan die orde van die dag was. In Suid-Afrika was hulle weer aan die ongenaakbare lewens- en werksomstandighede blootgestel eie aan die toestande in die mynbedryf in daardie tyd. Analises dui aan dat al 36 manlik was, tussen die ouderdom van 16 tot 45-jaar oud, met die meerderheid in die groep van jong volwassenes tussen 20 en 34-jaar oud. Patologie waargeneem sluit 'n prominente voorkoms van voedingsverwante veranderinge en lineêre emalje hypoplasie wat daarop dui dat hierdie mans lang periodes van wanvoeding en siekte deurstaan het in beide kinderjare sowel as in hul volwasse lewe. Ten spyte hiervan is daar bewyse van 'n groot persentasie van littekens wat tipies met wanvoeding verbind word wat tekens van herstel toon. 'n Hoë aanwesigheid van traumatiese beserings, meer spesifiek veral voordoodse frakture, is waargeneem en mag spesifiek bygedra het tot die dood van hierdie mynwerkers. Al dui die herstel van die patologiese littekens op 'n periode van verbeterde gehalte van voedselinname, moontlik tydens hulle verblyf op die Witwatersrand myne, is die hoë voorkoms van voordoodse frakture bewys van die lewensgevaarlike omstandighede wat met diepskag-mynwerk verbind word.

Om bystand te verleen aan die interpretasie van skeletale patologie waargeneem in die Sjinese is dit vergelyk met nie-spesifieke tekens van patologie waargeneem in kadawer skeletale oorskot, vir wie die oorsake van dood bekend was en verwant was aan metaboliese en voedingwaarde-gekoppelde siektes. Hierdie ondersteunende studie het patrone gelewer ten opsigte van patologie wat ‘n meer effektiewe interpretasie van patologie in die Sjinese tot gevolg gehad het.

Sleutel woorde: Beriberi, bioargeologie, diepskag mynbou, kadaver skeletale oorskot, metaboliese been siektes, paleopatologie, Sjinese kontrakwerkers, voedingsverwante siektes, wanvoeding, Witwatersrand myne

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CHAPTER 1: INTRODUCTION

"A persons' skeleton is remarkably informative about their health and well-being, dietary history, lifestyle (activity), ancestry, and key biological attributes (i. e., age and sex) that are used to construct demographic profiles of the population from which they originate" (Larsen, 2002: 119). The study of palaeopathology forms an integral part of bioarchaeology and can provide valuable information about a person's lifestyle and socio-economic status (Larsen, 1997; Buikstra and Beck, 2006). One of the subsets of palaeopathology is the study of diet and nutrition and its effects on the general health of a person. Interpreting such nutritional and metabolic disease related pathologies often provides additional insight into the daily social and cultural practices of people. It is therefore also an essential part of understanding differences amongst past populations from archaeological contexts and provides an alternative means for cross referencing historical accounts.

Central to the study of bioarchaeology is the understanding of the interactions between biology and human behaviour within a specific cultural and/or environmental setting (Larsen, 2002). The study of diet in bioarchaeological analysis of historic skeletal samples is traditionally concerned with the reconstruction of dietary patterns through studying the associated faunal and floral remains or more directly by isotopic analysis of human collagen (Larsen, 2002). Skeletal pathology, especially those related to metabolic diseases, can however aid in our understanding of the interaction between diet and health and how external factors such as environmental conditions and socio-cultural settings may influence this. This type of approach, termed the 'life course' approach by Brickley and Ives (2008), recognises that life is culturally construed, therefore interlinking culturally and behaviourally mediated influences on life and health with the surrounding environment and dietary practices (Brickley and Ives, 2008).

Some factors which can potentially limit the extent and accuracy of the use of skeletal pathology in interpretive studies should however be considered. Such factors include the accuracy of historical accounts in describing the environmental and socio-cultural conditions encountered by an archaeological population (Tosh and Lang, 2006); the reliability of palaeopathological and palaeodemographic data in being a true representation of the past population; and the accuracy with which investigators can classify and interpret skeletal pathology (Wood *et al.*, 1992; Ortner, 2003; Waldron, 2009). Wood *et al.* (1992) caution against the predisposed idea that a direct and indefinite relationship exists between the

frequency of skeletal lesions and the actual health of the populations studied. Wood *et al.* (1992) mention three fundamental theoretical problems that may influence the interpretation of palaeopathological and palaeodemographic results. The first theoretical problem identified by them is demographic non-stationarity which implies that age at death statistics does not necessarily reflect the mortality rate of the population studied. Instead it refers to the population's fertility measures (Wood *et al.,* 1992). This is based on the notion that no population is static and stationary. Constant change of their natural and cultural environment will have small effects on the fertility rate which in turn will have greater consequences on the life expectancy of the population (Wood *et al.,* 1992). The second theoretical problem is that of selective mortality; implying that all skeletal samples, irrespective of the sample size, are subject to selection bias (Wood *et al.,* 1992). This is due to the fact that the only individuals studied in skeletal samples are those who have died, therefore not taking into account those individuals who survived the same condition. Survivors will automatically be excluded from the skeletal series resulting in a misrepresentation of the true population health. Skeletal lesions observed will therefore be an overestimation of the true incidence amongst the larger population (Wood *et al.,* 1992). The third and last theoretical problem deals with the matter of hidden heterogeneity in the risk of disease and death in an individual (Wood *et al.,* 1992). Hidden heterogeneity refers to the variability of disease prevalence and susceptibility to disease and death in each human being. This may arise as a result of genetic causes, variations in the microenvironment, differences in the socioeconomic conditions, or even from trends in health (Wood *et al.,* 1992).

Since palaeopathological studies are based on the prevalence of skeletal lesions it should ideally be able to control for individual differences, yet due to nature of the discipline this becomes problematic as only individuals with skeletal pathology end up in palaeopathological studies. Due to the variability of individual immune system responses to pathogens and ante-mortem trauma it becomes problematic to interpret skeletal lesions solely as an indicator of ill health. The presence of skeletal lesions indicates that the person lived long enough for the pathogen to affect the skeleton, or as in the case with trauma provided enough time for the wounds to heal. Therefore skeletal lesions can also suggest a relative good health or at least a strong immune response which enabled the person to survive the pathogen or traumatic onslaught. The absence of skeletal lesions may therefore also be interpreted as an indicator of poor health as the persons immune system was not strong enough to fight off the pathogen and therefore succumb before the appearance of skeletal lesions.

Another problem stemming from this is the manifestation of skeletal lesions themselves. Not all diseases will necessarily affect the skeleton. Most diseases, especially the more fatal ones, will only affect the soft tissues at most, leaving no indication of its presence on the skeletal remains (Ortner, 2003; Waldron, 2009). Except in cases where organs and soft tissues are preserved, for example in mummified remains, palaeopathological studies are largely limited to those conditions affecting the skeletal system. Some diseases may present as diagnostic pathological lesions on the skeleton (for example treponemal diseases) whereas others may only present as non-specific markers of stress (for example subperiosteal bone growth) (Roberts and Manchester 1995; Ortner 2003; Waldron, 2009).

Even when considering all the shortcomings of palaeopathological studies, its usefulness in understanding past population health is still unequivocal. Some of the problems mentioned here can however be overcome by redirecting the focus of the study. The problem of heterogeneity as mentioned by Wood *et al.* (1992) for instance may be addressed, at least to some extent, by incorporating bioarchaeology's multidisciplinary strengths (Wright and Yoder, 2003). Aspects of socioeconomic differences and varying environmental conditions can be identified and understood through close consideration of the archaeological context and/or associated historical sources (Steckel *et al.*, 2002; Wright and Yoder, 2003). New advances in the field of bioarchaeology hope to overcome some of the issues mentioned here by looking at new avenues to understanding population dynamics and its influences on population health. These studies include advances made in the field of demographics, population movement, palaeodiets, skeletal growth, and ancient DNA analysis (Wright and Yoder, 2003).

For this reason the collective assessment of all pathological lesions observed on the skeletal remains of an individual is necessary to better interpret aspects of diet and health. Comparing the incidence of skeletal pathology in the archaeological remains studied with that of well documented modern-day medical collections may also aid in the interpretation of skeletal pathology and its diagnosis (Wood *et al.*, 1997).

1.1. Aim and objectives

This study is aimed at assessing the health of a historic skeletal sample of Chinese indentured mine labourers, who worked and died on the Witwatersrand mines during the early parts of the 20th century. The study specifically focuses on identifying skeletal pathology indicative of the nutritional health of the individuals in question. In order to get to this aim this study had four main objectives:

1. Standard physical anthropological analyses of the Chinese skeletal remains to assess their demographic features as well as any pathology and trauma which provided insight into their diet and health as well as their living conditions prior to and during their time on the mines.
2. Combine primary data obtained through the physical anthropological analysis with historical data about the importation and employment of Chinese labourers on the Witwatersrand mines. Historical accounts provided additional information pertaining to the diet and health of the Chinese miners and were used in combination with skeletal pathology to make inferences about life on the mines in general.
3. Study skeletal pathology in cadaver skeletal remains from the Pretoria Bone Collection (L'Abbè *et al.,* 2005) and the Raymond A. Dart Collection (Dayal *et al.,* 2009) for whom the cause of death was known and related to some form of malnutrition or nutritional deficiency in order to be able to recognize and interpret signs of disease possibly associated with nutritional deficiencies in this group.
4. Information obtained through the three above mentioned objectives will aid in the identification, interpretation and possible differentiation of pathology associated with metabolic bone diseases and nutritional deficiencies, including the possible identification of skeletal pathology associated with beriberi or thiamine deficiency.

CHAPTER 2: LITERATURE REVIEW

2.1. Historical background of Chinese mine labourers in South Africa

During the period 1904 to 1910, 63 695 Chinese were imported to South Africa to work on the Witwatersrand mines (Richardson, 1982; Harris, 2006). Gold was first discovered on the Witwatersrand in 1886 on the farm Langlaagte (Richardson, 1982). This discovery led to the influx of hundreds of prospectors from across the country as well as the rest of the world in an attempt to gain personal fortune. Subsequently large mining companies were established which eventually gained control over the large scale extraction of gold. Due to the low grade nature of the gold ore found in the Witwatersrand, deep level mining soon became a necessity and large quantities of golden ore had to be extracted in the shortest possible time in order to make a profit (Richardson, 1982). This facilitated a need for a large unskilled working force. This working force almost exclusively consisted of African labourers, mostly migrant labourers from rural areas across South Africa and even beyond South African borders.

Yet, this working force soon became insufficient following the second Anglo-Boer War 1899-1902 with the growing increase of wages alongside the large scale desertion or death of many of the African labourers. In 1903 an average of 62 056 Africans were employed on the Witwatersrand mines and in one year only a total of 47 323 men resigned, whereas 3164 were lost through desertion and an appalling 5022 due to death by disease or accident (Richardson, 1982). Unable to find a satisfactory alternative local workforce, the Chamber of Mines focused on China, known for its large, hardworking and dedicated unskilled working forces (Richardson, 1982; Harris, 1998). In May 1904 Britain and China successfully negotiated on an international convention, therefore completing the legal apparatus on which the introduction of this labour was based (Richardson, 1982; Yap and Man, 1996; Harris, 2006). Under the command of Lord Milner, governor of the Transvaal at this point, Chinese men were imported to South Africa as indentured labour to relieve the work shortage on the Witwatersrand mines (Harris, 1998).

2.1.1. Recruitment

The recruitment and importation of unskilled Chinese labour began in July 1904 (Richardson, 1982; Yap and Man, 1996). Initially the Chamber of Mines focused their attention towards southern China when recruitment of Chinese labourers began (Richardson, 1982; Yap and Man, 1996; Harris, 1998). The reason for this was that it was believed that the southern Chinese were already accustomed to the idea of working away from home, in countries abroad (Yap and Man, 1996). It was however soon realized that the southern Chinese did not present as a feasible labouring group to recruit from, mainly as a result of their health (Meyer, 1946; Richardson, 1982; Yap and Man, 1996). The SS Tweedale was the first ship to arrive at Durban in 25 May 1904 with new recruits from southern China and already the apparent poor health of these people was realized (Richardson, 1982; Yap and Man, 1996). "*Of the 1055 labourers embarked in China on this first shipment, only 1005 were actually distributed to the mines, and several more were to be rapidly struck down on arrival to the Rand*" (Richardson, 1982: 95). Their ill health was directly attributed to a disease called beriberi (Meyer, 1946; Richardson, 1982; Harris, 1998). The aetiology of beriberi was still poorly understood at that point in time. It was commonly perceived to be the result of an infectious disease rather than a vitamin deficiency, which meant that there was no actual treatment for the disease (Richardson, 1982; Carpenter, 2000).

As a result of the extensive loss of labourers due to disease the Chamber of Mines turned its attention to north China. Originally, recruitment from north China was not considered as the Northern Chinese were perceived to be much more rural and therefore wary of moving away from home to work in a foreign country (Yap and Man, 1996). "*In Northern China the idea of emigration to a far distance has to be brought home to the Coolie, but when this has been done (a process taking time and money) many fine men will be obtained from the North, especially from the province of Shantung*" (Yap and Man, 1996: 105). In the end only 2000 of the 63 695 labourers recruited to work on the Rand were from southern China. The majority of the labourers came from northern areas of Chihli (also known as Zihli, modern day Hebei Province), Shantung and Honan (also known as Shandong and Henan Provinces) (Richardson, 1982; Yap and Man, 1996; Harris, 1998). Most of the Northern Chinese who were recruited were peasant farmers and small scale tradesmen as suggested by the following quote; "*Far the greater proportion are peasants fresh from the plough or petty traders*" (Richardson, 1982: 126). Recruitment from these areas, however, even though more expensive, were more successful owing to the fact that the northern regions were suffering

the effects of the Russo-Japanese War (1904), and the economic stagnation and collapse of the silk-spinning industry (Yap and Man, 1996).

Chinese women were not employed as labourers and it is reported that only six Chinese wives came to South Africa during the period of 1904 to 1907 (Harris, 1998). "*It has never been anticipated that large numbers of labourers would bring their families, nor is it likely that those registered as married will bring their wives over… Suitable accommodation is provided, and every care is shown them on the ships, but the fact remains, that it is not their custom to bring their women folk with them on such occasions*" (Richardson, 1982: 127).

The age distribution of recruits ranged between 20-55 years, with a marked concentration in age group 20-35 years (Richardson, 1982). Very few recruits were younger than 20 years. However, it seems likely that discrepancies in the actual age given by recruits must have occurred as the low numbers of under aged labourers recorded do not correspond with predicted numbers (Richardson, 1982). "*In terms of age, the task was simpler and very few men seem to have been rejected on grounds of age, despite the fact that the contract stipulated a minimum age of twenty years without parental consent*" (Richardson, 1982: 128).

2.1.2. Terms of employment

Each Chinese labourer had to work for a minimum contract period of 3 years after which an additional 2 year contract could be issued by the Chamber of Mines (Richardson, 1982; Yap and Man, 1996). Prior to embarkation each recruit had to pass a medical examination and would then be issued a metal disc with his number engraved on it, along with a passport, some clothes and a salary advance of about £2-£10 (Yap and Man, 1996). They were also allowed to fill in an allotment book which allowed part of their salary to be paid directly to their relatives in China (Yap and Man, 1996). Chinese cooks and translators were also employed on the ships and on the mines to assist with the daily tasks. It is uncertain whether the Chinese recruits were fully aware of the circumstances surrounding their employment as none of the Transvaal emigration agents could speak the northern dialects (Yap and Man, 1996). This can be corroborated by the fact that so many complaints were lodged by Chinese miners once they were employed on the Witwatersrand, stating that they were not made aware of the fact that they had to work underground (especially deep underground) as mining in China was mainly done above ground or at very shallow depths (FLD 240, 76/7).

The Chinese labourers were provided with on-site accommodation in the form of compounds (Fig. 2.1a&b) specifically built for them, and were not allowed to venture outside the mining grounds without permission (Meyer, 1946; Richardson, 1982; Harris, 1998). Large brick buildings enclosing a rectangular shape were constructed to serve as compounds. These buildings provided 'bunk rooms', communal bathrooms and outside latrines (Yap and Man, 1996). Accommodation varied between compounds with some labourers having low sleeping platforms divided by curtains and wire-mesh bunks and others were confined to rooms for 40 men, consisting of two tiers of concrete sleeping bunks (Yap and Man, 1996). Medical facilities were provided in each compound and consisted of white and Chinese doctors and Chinese assistants (Meyer, 1946; Yap and Man, 1996). In some cases traditional Chinese medicine was also provided.

Every compound also had its own cooking house (Fig. 2.2). Chinese cooks were employed to prepare daily meals in accordance with a traditional Chinese diet (Meyer, 1946; Yap and Man, 1996). Meals were served in large dining rooms, however most labourers preferred to retreat to their sleeping quarters (Meyer, 1946; Yap and Man, 1996). The daily food rations consisted of one and a half pounds of rice, half a pound of dried or fresh fish or meat, half a pound of vegetables, half oz (ounce) of tea, half oz (ounce) of nut oil and salt (Meyer, 1946; Yap and Man, 1996).

During their stay in South Africa the Chinese labourers were not permitted to leave the compound without a permit. A red permit allowed absence until sundown on the day of issue whereas a white permit allowed overnight leave or for a period not exceeding 48 hours (Fig. 2.3; Meyer, 1946; Richardson, 1982; Yap and Man, 1996). Strict measures were taken to exercise control over the labourers. Each shipload had been accompanied by a 'headman', policemen, Chinese-speaking whites who would be Chinese Controllers at the mines, and interpreters (Yap and Man, 1996). This caused many problems and resulted in constant complaints from the labourers. Chinese Controllers often could not communicate adequately due to the fact that they were not really fluent in the dialect (Richardson, 1982; Yap and Man, 1996). This meant that effective control could not be enforced which indirectly lead to bribing and other illicit activities such as gambling, opium smoking and illegal trading (Richardson, 1982; Yap and Man, 1996). Ill treatment of Chinese labourers by mine management and fellow workers was often reported. One of the most contentious issues surrounding the treatment of labourers was 'floggings'. This form of corporal punishment was used to circumvent legal proceedings as these were time-consuming and expensive (Yap

and Man, 1996). A public outcry eventually led to the abolishment of floggings in June of 1905 (Yap and Man, 1996).

Policemen or 'police boys', as they were referred to, often abused their power and severely mistreated the labourers they were responsible for. Part of their responsibilities was to deal with complaints and grievances on the labourers' behalf, therefore acting as a middle man between labourers and white officials (Meyer, 1946; Richardson, 1982; Harris, 1998). Bribes were commonly accepted to keep illicit activities quiet and as a result many labourers had little hope of gaining justice through appeals to white officials (Kynoch, 2010). Powerful syndicates, controlling the gambling activities and to some extent the opium supplies, came into place and were often directed or at least sanctioned by the Chinese police force (Kynoch, 2010). This led to violence within the compounds and often resulted in murder and/or suicide (Meyer, 1946; Yap and Man, 1996; Harris, 1998; Kynoch, 2010). Some of the hostilities between Chinese police and Chinese labourers may have derived from previous antagonisms in their home areas (Kynock, 2010). The most successful recruiting grounds were those areas affected by the Boxer Rebellion (1900) and as a result some of these tensions may have made their way into the mines and compounds (Richardson, 1982; Kynoch, 2010).

2.1.3. Working conditions and health

Even though the Chinese labourers were employed on a contract basis and strict measures were taken to ensure their health and safety, many still lost their lives on the Witwatersrand mines. The Chinese labourers were employed underground (Fig. 2.4) whereas the few remaining African labourers were employed above ground (Meyer, 1946; Richardson, 1982; Yap and Man, 1996; Harris, 1998). Not only did underground work present with obvious hazards, but many of the Chinese labourers employed here had no prior experience of mining underground and had never dealt with explosives before, causing many work-related accidents (Richardson, 1982). Inexperience contributed to many accidents like labourers jumping into moving cages, drilling into unexploded charges and not knowing how to use explosives (Yap and Man, 1996). "*It was estimated that 3192 Chinese labourers died on the Rand between 1904 and 1910, a mortality rate of nearly one in twenty. Of these, 986 died as a result of causes directly attributable to their conditions of work*" (Yap and Man, 1996: 117). A further 611 Chinese labourers sustained injuries that left them partially or totally disabled resulting in the repatriation of 523 disabled Chinese labourers (Richardson, 1982; Yap and Man, 1996). Chinese miners often drilled into unexploded or misfired holes

from previous blasting shifts which would set off these explosives, killing and injuring many in the process (Richardson, 1982). "*Between 1904 and 1910, 212 Chinese died and a further 283 sustained serious permanent injury from drilling into unexploded holes – an annual average percentage of total Chinese casualties of 21.4 per cent*" (Richardson, 1982: 172).

Most of the Chinese indentured labourers employed on the mines were from the low income peasant society of China and suffered from disease and malnutrition (Harris, 1998; Harris, 2006). Most of the people screened for recruiting and even those who were eventually recruited were not in a good state of health. "*Major causes of death among the labourers included beri-beri, dysentery, phthisis, opium-poisoning, pneumonia, tuberculosis and other respiratory diseases, suicide and murder"* (Yap and Man, 1996: 118). This first became evident in screening periods during recruitment. Medical examinations were conducted prior to consideration to ascertain whether the men were healthy and fit enough to be able to cope with the work demands posed by the Witwatersrand mines. During this first medical screening period large numbers of interested labourers were rejected as being medically unfit. This was mainly due to the fact that so many suffered from beriberi (Meyer, 1946; Richardson, 1982). The wide-spread occurrence of beriberi was again confirmed when many recruits, originally cleared during the first medical screening, were rejected during the medical examination prior to embarkation and again at the departure in Durban (Richardson, 1982). This can be reflected in that 63 938 Chinese recruits originally left from China to work in South Africa (63 695 arriving in Durban), but that in the end only 62 654 were actually employed on the mines (Richardson, 1982). Although some of the losses can be attributed to desertion, a large proportion was due to the death or illness to such an extent that the recruits had to be returned to China immediately.

The deceased ended up being buried in mine cemeteries, sometimes alongside African mine workers or in separate areas specifically allocated for Chinese mine labourers (Dart, 1952). With the commencement of the repatriation of Chinese indentured labourers from 1907 to 1910, some of the graves were exhumed and the remains cremated to be taken back to China (Fig. 2.5; Richardson, 1982; Yap and Man, 1996). Graves were dug up under municipal supervision and funeral pyres were made out of stone and flammable material and the coffin placed on top (Yap and Man, 1996). Afterwards the ashes were collected in two-foot square bags and taken back to China (Yap and Man, 1996). For safety reasons this was only allowed for graves that were older than one year (Yap and Man, 1996) and as a result many of the graves were left in place. These graveyards, although classified as official mine cemeteries, were unmarked and presented what can be termed as paupers graves. Today these

mining cemeteries remain scattered throughout the Witwatersrand area, most no longer visible on the surface.

2.1.4. Witwatersrand Deep Ltd.

Witwatersrand Deep gold mine was owned by S. Neumann & Co. and was first registered in 1899 (Richardson, 1982). This company employed Chinese labourers from 1904 to 1908. In October of 1906 it was recorded that 2942 Chinese were employed on Witwatersrand Deep Ltd. (Richardson, 1982).

Medical records recorded for the year 1905 to 1906 reflected the following causes of death for Chinese employees for this period (FLD 90, 13/7; FLD 91. 13/7; FLD 127, 18/7; FLD 166, 32/7);

Table 2. 1: Causes of death recorded for the Chinese employees from Witwatersrand Deep Ltd for 1905 to 1906

*Shock: Fractures obtained through rock falls, explosions, falls from skip and down mine shafts, and mine accidents in general.

Cause of death	Total number	Cause of death	Total number
Septicaemia	17	Chronic diarrhoea	4
Dysentery	10	Malaria	1
Acute gastritis	2	Electric shock	1
Enteric fever	9	Inhalation of gas	1
Opium poisoning	14	Beriberi	11
Peritonitis	3	Syphilis	4
Abscess of brain	5	Necrosis of skull	1
Chronic bronchitis	2	Shock*	58
Wounds	2	Fracture (skull/neck)	21
Haemorrhage	2	Murder	4
Insanity	1	Execution	3
Phthisis	3	Shot by SAC (South African Corps)	1
Cardiac disease	2	Suicide	5
Erysipelas	2		

It is clear from these numbers that by far the most deaths could be attributed to injuries sustained as a result of work related accidents. There is also clear evidence for the presence of treponemal disease as well as for nutritional deficiency as indicated by the presence of beriberi.

2.1.5. Archaeological history of remains

In 1951 the Boksburg Municipality uncovered graves during pipe laying activities (Dart, 1952). It was determined that the remains uncovered during construction work belonged to Chinese miners employed and buried on the Witwatersrand Deep mine grounds during the importation and employment of Chinese labourers in the Witwatersrand area during the period of 1904 to 1910 (Dart, 1952). With the permission of the Transvaal Provincial Administration and the concurrence of the Boksburg Municipality, the skeletal remains were exhumed and transferred for permanent custody to the Anthropological Collection of the Anatomy Department at the University of the Witwatersrand (Dart, 1952). These skeletons are today still housed at the University of the Witwatersrand as part of their Raymond A. Dart Collection. These skeletons represent the only known Chinese indentured labourer sample in South Africa and is representative of a very short, but important period in the history of the Transvaal gold mines.

Other than the obvious historical significance these remains may also provide additional insight into the living and working conditions encountered by these people on the Witwatersrand mines. It is known that Chinese labourers migrating from China were already subjected to less than ideal living conditions and that most of those recruited came from low income peasant societies across China (Richardson, 1982; Yap and Man, 1996; Harris, 1998). "*Poverty was endemic in these regions* [Northern China] *as inhabitants were subjected to floods, droughts and severe famines*" (Harris, 1998: 153). Furthermore the large number of 'medical rejects' identified during the preliminary selection period in China in the recruitment phase attests to a background of considerable distress (Richardson, 1982; Harris, 1998). These conditions were brought on by the prevailing poverty amongst the peasant societies, and also the political instability and oppression caused by the Boxer rebellion in 1900 followed by the Russo-Japanese War of 1904-1904 (Richardson, 1982; Harris, 1998). During this period the Chinese were subjected to severe conditions of poverty which resulted in famine and the overall decline of their health. For this reason this skeletal sample becomes very valuable in understanding the overall health of these Chinese indentured mine labourers;

not only as a direct result of their socio-economic status in China, but also as a result of the conditions presented by mine work and compound living in South Africa. Once employed on the Witwatersrand mines, their conditions did not always improve and Chinese labourers were further subjected to the hazards of underground mining as well as infectious diseases and violence commonly associated with compound living conditions (Harris, 1998). Other than the traumatic injuries associated with deep level mining, dietary health also played a role in the overall health and survival of these labourers. As has been shown, a large number of deaths sustained amongst the Chinese labourers were brought on by beriberi, a thiamine deficiency (Yap and Man, 1996; MacDonald, 2008). Coming from a poverty-stricken background, most of the Chinese labourers employed on the Witwatersrand mines may already have suffered from several nutritional deficiencies, possibly worsened by their prevailing nutritionally deficient diet of mainly white rice. Dietary deficiencies are often visible on skeletal remains and therefore this sample provided an opportunity to study some of the possible dietary deficiencies suffered by these Chinese labourers.

2.2. Metabolic bone diseases

Metabolic bone diseases include an assemblage of diseases which pathogenesis is quite varied and still incompletely understood (Brickley and Ives, 2008; Kozłowski and Witas, 2012). These disorders include many vitamin-related diseases like vitamin C and D deficiency, as well as disorders related to changing hormonal balances such as post-menopausal osteoporosis, and disorders caused by insufficient intake of crucial dietary components and trace elements like fluorine (Allgrove, 2007; Brickley and Ives, 2008; Waldron, 2009). The term ‘metabolic bone disease’ has been used since 1948, when Albright and Reifenstein first used it to describe conditions that affected the processes of bone formation and remodelling involving the whole skeleton (Brickley and Ives, 2008; Kozłowski and Witas, 2012). Metabolic bone diseases in the palaeopathological context do not, however, take into account the various metabolic related diseases that do not directly affect the skeleton. Recently the term, ‘metabolic syndrome’ which is considered a subset of metabolic disease, has received great attention (Brickley and Ives, 2008; Kozłowski and Witas, 2012). Disorders labelled under the metabolic syndrome now focus on diseases like cardiovascular disease, hypertension and high cholesterol levels which do not directly affect the skeleton (Brickley and Ives, 2008; Kozłowski and Witas, 2012). Yet, some of the diseases labelled

under this term, for instance anaemia and obesity, may have secondary manifestations that can become apparent as pathological alterations on the skeleton.

2.2.1. Disorders of bone formation

The osteological manifestations of metabolic bone diseases can respond either as bone resorption, bone depositioning, or a combination of both these processes (Allgrove, 2007; Brickley and Ives, 2008; Waldron, 2009). Metabolic bone diseases are characterised by those osteological responses that reflect disruptions in bone formation, bone remodelling, or a combination (Brickley and Ives, 2008; Waldron, 2009). The cells responsible for these processes are known as osteoclasts, osteoblasts, and osteocytes. Osteoclasts are responsible for the removal of bone through a process of resorption, whereas osteoblasts are responsible for synthesising new bone through secreting organic bone matrix and regulating its subsequent mineralization (Brickley and Ives, 2008; Waldron, 2009). Osteocytes do not play a direct role in bone absorption and bone formation, but rather act as communicating cells and help recognise bone fatigue which will then initiate the remodelling of bone in such areas (Brickley and Ives, 2008; Waldron, 2009).

Remodelling of bone takes place after bone growth has ceased and entails the removal of existing bone to create new bone. Remodelling will only take place in areas where the bone has been identified as being structurally weakened and needing replacement or to release calcium ions required by other metabolic processes (Brickeley and Ives, 2008; Waldron, 2009). The older bone is, therefore, the more remodelling it will undergo in order to maintain the integrity and functionality of the skeleton as a whole (Brickley and Ives, 2008; Waldron, 2009). Osteological responses can therefore be useful in identifying underlying diseases as affected areas will present with distinctive remodelling. In children where bone growth is still apparent, remodelling may not be the only means of identifying underlying diseases. Osteological responses may also present in the form of growth stunts or the altering of bone shapes due to mechanical loading in response to such underlying diseases and in some cases may prevail throughout adulthood (Larsen, 1997; Brickley and Ives, 2008).

Harris lines have always been interpreted as non-specific signs of stress in children (Harris, 1933; Larsen, 1997; Aufderheide and Rodríguez-Martin, 1998; Ortner, 2003). Harris lines present as transverse radiopaque lines in the long bones and have been correlated with episodes of temporary arrest of longitudinal growth caused by stress factors like malnutrition, illness and psychogenic stress on long bones (Larsen, 1997; Ameen *et al.*, 2005;

Papageorgopoulou *et al.*, 2011; Weston, 2012). Alfonso-Durruty (2011) and Papageorgopoulou *et al.* (2011), however, indicated that Harris lines could not be directly associated with episodes of stress during childhood and related growth stunts. Their results suggested that Harris lines rather, are suggestive of normal growth and growth spurts. This is indicated by the high frequency of Harris lines observed during periods of peak growth as a result of hormonal secretion (Alfonso-Durruty, 2011; Papageorgopoulou *et al.*, 2011). Also no correlation with the presence of enamel hypoplasia and Harris lines could be observed, further suggesting that Harris lines may be a factor of normal growth rather than an indicator of nonspecific stress (Alfonso-Durruty, 2011; Papageorgopoulou *et al.*, 2011).

Enamel hypoplasia, on the other hand, has been reported as an indicator of stress during childhood, either as a result of malnutrition or illness, or a combination thereof. Enamel hypoplasia is an enamel defect that occurs while tooth enamel is deposited during development (Hillson 1998; Aufderheide and Rodríguez-Martin, 1998; Ortner, 2003). It may be caused by periods of malnutrition or illness (Hillson 1998; Aufderheide and Rodríguez-Martin, 1998; Ortner, 2003). Goodman and Rose (1991) describe enamel hypoplasia as a deficiency in enamel thickness visible as transverse grooves or pits on the outer enamel surface. These defects occur as a result of disrupted ameloblast function and reduced secretion of enamel matrix during amelogenesis (Goodman and Rose, 1991; Aufderheide and Rodríguez-Martin, 1998). Enamel does not remodel, causing permanent visual indicators for stress that remains visible throughout adulthood (Roberts and Manchester 1995; Larsen 1997; Aufderheide and Rodríguez-Martin, 1998).

2.2.2. Trace element and vitamin deficiencies

2.2.2.1 Vitamin D deficiency

One metabolic disease responsible for the altering of bone shape in juvenile skeletons is rickets or vitamin D deficiency. Vitamin D is a pro-hormone rather than a traditional vitamin since it requires synthesis to become actively utilised in the body (Brickley and Ives, 2008). Vitamin D (calciferol) plays an important role in the maintenance of skeletal health and calcium and phosphorous homoeostasis, and is necessary for the proper mineralization of osteoid formed during bone growth and remodelling (Aufderheide and Rodríguez-Martin, 1998; Ortner, 2003; Brickley and Ives, 2008; Kozłowski and Witas, 2012). Osteoid mineralization strengthens the bone and prevents deformation during bone modelling in childhood or remodelling during adulthood (Aufderheide and Rodríguez-Martin, 1998;

Ortner, 2003; Kozłowski and Witas, 2012). Vitamin D therefore aids in intestinal mineral absorption and regulating renal re-absorption and excretion to ensure a sufficient amount of calcium and phosphorous in the blood serum, which in turn enables bone mineralization and maintenance of the skeleton (Jowsey, 1977; Aufderheide and Rodríguez-Martin, 1998; Brickley and Ives, 2008; Kozłowski and Witas, 2012).

- **Synthesis of vitamin D**

When the skin is exposed to solar ultraviolet B radiation it converts 7-dehydrocholesterol to pre-vitamin D3, which in turn is rapidly converted to vitamin D3 (Aufderheide and Rodríguez-Martin, 1998; Holick, 2004; Holick, 2007; Waldron, 2009). The vitamin D3 is then metabolized in the liver to 25-hydroxyvitamin, which is alternatively metabolized in the kidneys by the enzyme 25-hydroxyvitamin D-1α-hydroxylase (CYP27B1) to its active form, 1,25-dihydroxyvitamin D (Jowsey, 1977; Holick, 2004; Holick, 2007; Waldron, 2009). The metabolically active form of vitamin D can now be used throughout the body for gastrointestinal, cellular, musclular, and skeletal functions (Brickley and Ives, 2008). It is estimated that sun exposure of 5 to 15 minutes each day is necessary for adults wearing sleeveless clothes, whereas children fully clothed, but without a hat, would need at least 2 hours exposure every week for sufficient vitamin D synthesis (Holick, 2005). Yet, it should be kept in mind that sunlight exposure varies between different geographical locations and fluctuates with the seasons (Holick, 2005). About 90% of the vitamin D requirements come from daily exposure to sunlight (Holick, 2004), yet some foodstuffs are also known to contain a form of vitamin D which can be synthesised by the body into an active form of vitamin D. Calciferol or vitamin D2 is produced by irradiated food sources whereas other foodstuffs naturally contain vitamin D3 (Holick, 2007). Both are metabolized in the liver and kidneys, in the same way as vitamin D synthesis through sun exposure, to a form that can be readily used by the body (Jowsey, 1977; Holick, 2007; Brickley and Ives, 2008; Waldron, 2009).

- **Pathogenesis of vitamin D deficiency**

Several factors play a role in the development of Vit D deficiency, including the exclusive breast-feeding of infants, maternal vitamin D deficiency, calcium deficiency, living in temperate climates, lack of sunlight exposure, darkly pigmented skin in areas with less ultraviolet rays, or insufficient intake of vitamin D from the diet in areas where sun exposure

is limited (Pettifor, 2004; Brickley and Ives, 2008; Waldron, 2009; Kozłowski and Witas, 2012). Vitamin D synthesis is, therefore, dependent on the exposure of the skin to the ultraviolet rays of the sun or dietary intake of vitamin D from eggs, fortified milk, liver and oily fish (Klein, 1993; Brickley and Ives, 2008, Waldron, 2009; Kozłowski and Witas, 2012). It has also been suggested by Özgür *et al.* (1996) that high levels of strontium may inhibited the parathyroid glands, leading to a reduction in the production of active vitamin D metabolites by the kidney. They concluded that in populations where nutrition is mainly based on grain cereals the presence of strontium in the soil will increase the prevalence of rickets significantly (Özgür *et al.*, 1996).

Aspects of social behaviour also play a role in the prevalence of vitamin D deficiency amongst specific population groups. For instance in some cultural groups it is customary to adopt specific clothing practices which limits their skin exposure to a large extent and therefore makes them much more susceptible to the deficiency (Brickley and Ives, 2008; Kozłowski and Witas, 2012). Amongst some African populations it is also customary for mothers to carry their young children on their backs, almost completely covering their bodies with blankets or clothes (Holick, 2007; Brickley and Ives, 2008). Dietary preferences may also lead to a deficiency in vitamin D and calcium. Children and adults consuming strict vegan and vegetarian diets are more prone to developing vitamin D deficiency (Curtis *et al*., 1983; Messina and Mangels, 2001; Murphy and Allen, 2003; Pettifor, 2004). Such strict diets are devoid of any animal products and without supplementation drastically limit the amount of vitamin D and calcium being consumed (Curtis *et al*., 1983; Messina and Mangels, 2001; Murphy and Allen, 2003; Pettifor, 2004). Furthermore, the elderly, specifically elderly women, as well as pregnant or lactating women are also extremely susceptible to developing a deficiency due to the higher demand of calcium needed during such conditions (Andiran *et al.*, 2002; Ortner, 2003; Van Schoor *et al*., 2007). An aspect not so readily thought of is the modern day concern surrounding skin cancer and as a result the routine use of sunscreen (Holick, 2004; Brickley and Ives, 2008). By constantly preventing the absorption of ultraviolet light from entering the skin, vitamin D synthesis is largely diminished.

Without vitamin D synthesis calcium and phosphorus, two main components of bone mineral, cannot be adequately metabolised, which will then severely affect the modelling and remodelling of bone (Aufderheide and Rodríguez-Martin, 1998; Ortner, 2003; Waldron, 2009; Kozłowski and Witas, 2012). *"Without vitamin D, only 10 to 15% of dietary calcium and about 60% of phosphorus is absorbed"* (Holick, 2007). The skeletal features associated with vitamin D deficiency include more than 60 pathological conditions (Ortner, 2003).

Similarly, the manifestation of gross skeletal pathologies may largely hamper the specific and indefinite identification of the various causes; be it either nutritional or due to underlying pathology (Ortner, 2003). These causes include all the conditions giving rise to the inadequate intake and synthesis of vitamin D and its precursors, an inadequate intake of calcium, as well as any genetic problems influencing mineral absorption and retention (Ortner, 2003; Pettifor, 2004; Waldron, 2009). Vitamin D deficiency affects both children and adults and is alternatively described as rickets in children and osteomalacia in adults (Jowsey, 1977; Klein, 1993; Ortner, 2003; Brickley and Ives, 2008). The reason for the differential naming is mainly attributed to the age differences with major overlap and similarity seen within either's pathological features (Brickley and Ives, 2008). Additionally, severe and/or reoccurring conditions of childhood rickets may deform long bones to such an extent that some degree of deformation remains even after healing has occurred. This occurrence is referred to as residual rickets and may be observed in children or adults (Brickley *et al*., 2010).

- **Rickets**

Rickets is seen as a systematic disease of early childhood which severely affects the skeleton, yet has no direct mortality (Ortner, 2003; Waldron, 2009). The vitamin D deficiency prevents calcium from being deposited in the developing cartilage or newly formed bone osteoid, impeding bone mineralization (Ortner, 2003; Brickley and Ives, 2008; Waldron, 2009). The result of deficient mineralization of osteoid is that bone has inadequate mechanical strength and therefore deforms easily when subject to weight bearing and muscular tension (Ortner and Mays, 1998). Rickets rarely begins before 4 months of age due to the fact that vitamin D passes from the mother to the foetus through the placenta and is eventually stored in the liver of the infant and can still be used for several months after birth (Ortner, 2003). The highest incidence rate of rickets is, however, seen at an age of between 3 months and 2 years, with a peak around 18 months and is usually more common in infants that are not yet mobile enough to move around freely in the sun (Ortner, 2003; Pettifor, 2004; Brickley and Ives, 2008).

Very few new cases of the developments of rickets have been observed at an age older than 4 years (Ortner, 2003). Some cases of active rickets have, however, been reported in South Africa in 4-16 year old children, yet the cause here was attributed to a severe calcium deficiency rather that a vitamin D deficiency (Pettifor, 2004). It was noted that a major

characteristic of diets in both rural South Africa and Nigeria is a high concentration of especially unrefined cereals (Pettifor, 2004). These cereals have a high content of phytate which in turn impairs calcium absorption. It was therefore suggested that the major cause for rickets among children outside the infant group was a dietary calcium deficiency, rather than a lack of sun exposure (Pettifor, 2004). So even though adequate sun exposure produced enough vitamin D, the body did not have enough calcium available for the proper osteoid mineralization. The age of onset may, therefore, vary considerably depending on the context, be it the maternal health or socio-cultural practices influencing diet and mobility of juveniles (Brickley and Ives, 2008).

The physical effects of the deficiency include a temporary cessation of breathing, involuntary muscle contractions and convulsions particularly affecting the face, hands and feet (Brickley and Ives, 2008). Severe muscle weakness may also be associated with the deficiency and can be significant enough in some cases that it prevents movements to a large degree (Truswell, 2002; Brickley and Ives, 2008). Vitamin D deficiency amongst children not only causes overt rickets, but can also prevent children from reaching their genetically programmed height and peak bone mass (Holick, 2004; Kozłowski and Witas, 2012). Bone deformity in rickets depends on the age at onset and behaviour relevant to the age of occurrence (Brickley and Ives, 2008). Skeletal manifestation of rickets is first seen and most marked on the rapidly growing areas of the skeleton which includes the osteocartilaginous junctions of the ribs, the distal metaphysis of the femur, radius, and ulna, and the proximal humerus (Aufderheide and Rodríguez-Martin, 1998; Ortner, 2003; Brickley and Ives, 2008; Kozłowski and Witas, 2012). The changes at the growth plate include failure of mineralization of the cartilage and the newly deposited osteoid on the cartilage septae (Ortner, 2003). The growth plates may appear widened and there is a widening and cupping of the metaphysis (Ortner, 2003; Waldron, 2009). In severe cases the accumulation of unmineralized osteoid and the weakened bone causes bending deformities in the long bones as a direct response of weight-bearing, and includes the forearm during crawling and the legs during walking (Ortner, 2003; Brickley and Ives, 2008; Kozłowski and Witas, 2012). Young infants (specifically in some African populations) that are tightly swaddled on the mothers' back may exhibit bowing and angulation deformities and even metaphyseal fractures in the long bones and ribs (Brickley and Ives, 2008). In ribs this can be presented by round nodular swelling of the osteocartilaginous junction, also referred to as the rachitic rosary, and in long bones it may be presented by a broadening and cup-shaped depression of the metaphyseal areas (Ortner, 2003; Waldron, 2009; Kozłowski and Witas, 2012). The skeletal changes

observed in rickets result from three mechanisms, namely the direct effects of the metabolic disturbance, deformities secondary to the vulnerability and pliability of the poorly mineralized skeleton, and the retardation of growth (Ortner, 2003).

Skull

The rapidly growing cranial bones of an infant suffering from rickets are increasingly replaced by non-mineralized osteoid and this causes the vault to develop thin or soft areas (Ortner, 2003; Brickley and Ives, 2008). This is especially prevalent in the posterior lateral portions of the parietal bones as well as the occipital squama (Ortner, 2003). This is due to the fact that the skull is rapidly remodelled in order to accommodate the growing brain, and therefore replacing mineralized bone with osteoid (Ortner, 2003). This may result in the delayed closure of fontanelles and during the constant process of remodelling, the outer and often the inner table disappears, so that the entire thickness of the cranial vault has a porous appearance of diploë and may present as porotic hyperostosis (Aufderheide and Rodríguez-Martin, 1998; Ortner and Mays, 1998; Ortner, 2003; Kozłowski and Witas, 2012). These porotic lesions may also occur on facial bones and a weakened skull base which may result in the deformation of the foramen magnum (Ortner, 2003; Kozłowski and Witas, 2012).

Long bones

Bending deformities of the long bones and metaphyseal swelling are some of the most characteristic features of rickets (Ortner, 2003; Brickley and Ives, 2008). There can be considerable variation in the manifestations of bending between affected limbs and between individuals (Brickley and Ives, 2008). The early stages of skeletal manifestation of rickets may not include bending deformities, instead the initial changes at the metaphyses will include fraying and flaring of the growth plate margins and metaphyseal junction as the cartilage structure starts to lose the organized vertical arrangement (Aufderheide and Rodríguez-Martin, 1998; Brickley and Ives, 2008; Kozłowski and Witas, 2012). These changes may progress to marked swelling in severe cases. Overall active rickets can be recognised by increased porosity of bone surfaces (Brickley and Ives, 2008). This porotic form of rickets is usually associated with a vitamin D deficiency and a general malnutrition (vitamin C, iron, and calcium deficiency) which results in a combination of rickets and osteoporosis (Ortner, 2003; Kozłowski and Witas, 2012). In relatively well-nourished infants the deposition of massive amounts of osteoid on the endosteal and periosteal surfaces results

in “plump” bones, which have narrowed medullary spaces and is characteristic of hyperplastic rickets (Ortner, 2003). In the porotic form of the deficiency, stress fractures especially in the diaphysis are a common occurrence and usually lead to axial deformities (Aufderheide and Rodríguez-Martin, 1998; Ortner, 2003; Brickley and Ives, 2008). Bending deformities are also associated with porotic rickets and may present with or without additional stress fractures (Ortner, 2003).

The epiphyseal closure times are not affected by active rickets, yet growth is delayed with the most marked shortening seen in the femur (Aufderheide and Rodríguez-Martin, 1998; Ortner, 2003; Kozłowski and Witas, 2012). Periosteal deposition on the femur is also more severe on the posterior side than on the anterior surface. Bending of the femur occurs in the shaft usually at the lower metaphyses with an anterolateral convexity (Ortner, 2003). On the tibia periosteal deposits locate on the posterior and medial surfaces, leaving free the lateral surfaces that face the fibulae (Ortner, 2003; Waldron, 2009). The tibiae also bend anteriorly at the lower metaphyses which result in the fibulae following the similar deformity due to the fixation of the fibula to the tibia (Ortner, 2003; Waldron, 2009). Deformities that develop in the long bones may remain static for the rest of that individual’s life should vitamin D intake increase and osteoid formed during rickets mineralize. When the deformities develop during the active phase, compensatory alterations of the distribution of subperiosteal bone are observed (Ortner, 2003). This includes deposition on the concavity of the deformity in response to the altered stresses (Ortner, 2003). Additional callus formation may also be observed in such areas caused by stress fractures, mainly observed on the diaphysis (Ortner, 2003). In infants bending deformities of the humerus and forearm may occur before walking as a result of crawling (Ortner, 2003; Brickley and Ives, 2008; Kozłowski and Witas, 2012). The humerus head is depressed medially downward and can bend the shaft laterally and forward and is referred to as *humerus varus* (Ortner, 2003). If, however, rickets only start after walking then these deformities may only be limited to the lower extremities. When healing occurs minor deformities may disappear whereas the more severe alteration will remain throughout adulthood.

Ribs, vertebrae and pelvis

The ribs may illustrate flattening of their curves once bending of the rib cartilage at the costochondral junction has occurred (Aufderheide and Rodríguez-Martin, 1998; Ortner, 2003). The sternum has to respond to this deformity and bends forward causing the pigeon

breast appearance also called *pectus carinatum* (Aufderheide and Rodríguez-Martin, 1998; Ortner, 2003). The vertebrae are only affected in severe rickets and present with the compression of the vertebral bodies, often combined with a deeper scalloping of the endplate (Ortner, 2003). After the active phase kyphoscoliosis may develop (Aufderheide and Rodríguez-Martin, 1998; Ortner, 2003; Kozłowski and Witas, 2012). The pelvis, on the other hand, is more affected by altered growth than by mechanical deformation and is much smaller and plumper than normal (Ortner, 2003). The pelvic canal narrows anteroposteriorly which creates the typical postrachitic flat appearance (Ortner, 2003). The acetabulum is also affected to some extent and protrudes more into the pelvic canal with the acetabulum facing much more forward (Ortner, 2003).

- **Residual rickets**

In case of persistent childhood vitamin D and calcium deficiency residual rickets may occur (Brickley and Ives, 2008; Brickley *et al*., 2010). Very few of the more subtle osteological changes, like the widening and cupping of metaphyses, associated with rickets will survive as recognisable changes in the adult skeleton (Brickley and Ives, 2008; Brickley *et al.*, 2010). In contrast, the more gross deformities of the long bones, for example the medio-lateral and anterior-posterior bowing, will be more likely to persist throughout adulthood and would therefore present as a marker for residual rickets in adult skeletal remains (Brickley and Ives, 2008; Brickley *et al*., 2010).

Other macroscopic changes that can be observed in residual rickets include changes in the skull, dentition, vertebrae, ribs and sternum, pelvis and sacrum, and the long bones. The skull may present with frontal or parietal bossing, the formation of a large square shaped head, and mandibular ramus medial angulation (Brickley and Ives, 2008). Dentition may present with enamel hypoplasia and dental caries (Hillson, 1998; Brickley and Ives, 2008). The vertebrae may deform to create kyphoscoliosis and vertebral body collapse (Ortner, 2003; Brickley and Ives, 2008). These lesions may be more prone to occur in T9-L3 (Ortner, 2003; Brickley and Ives, 2008). The ribs and sternum may present with the characteristic pigeon breast appearance also associated with rickets (Brickley *et al*., 2005; Brickley and Ives, 2008). The pelvis may be narrowed laterally with bulging pubic symphyses and a narrowed pelvic inlet with the acetabulae pushed dorsally (Ortner, 2003; Brickley *et al*., 2005; Brickley and Ives, 2008). This is also seen in osteomalacia. The sacrum becomes ventrally projected and anteriorly angulated (Ortner, 2003; Brickley and Ives, 2008). Finally,

the long bones of the lower extremities may present with residual bending (Brickley and Ives, 2008). The femoral neck may present with heightened angulation as well as the angulation of the knees (knock knees), with medial and lateral widening of the proximal and subtrochanteric areas (Brickley *et al.*, 2005; Brickley and Ives, 2008). Overall the long bones may become shortened and may become deformed (anterior-posterior and medio-lateral bowing) with increased thickness in the areas of bowing (Brickley *et al.*, 2005; Brickley and Ives, 2008).

- **Osteomalacia**

"*The term "osteomalacia" originally referred to generalized softening of bone leading to crippling deformities*" (Parfitt, 1998: 327). In adults the vitamin D deficiency and the associated pathology may be very non-specific (Ortner, 2003; Brickley *et al.*, 2005; Brickley and Ives, 2008; Kozłowski and Witas, 2012). Symptoms associated with vitamin D deficiency in adults include generalised or local muscle pains with muscle weakness, whereas in prolonged cases muscle and bone deformity in the pelvis and hip may lead to the impediment of locomotion resulting in a '*waddling gait*' (Brickley and Ives, 2008). In adults the trabecular and compact bone undergo constant remodelling and the bone matrix formed during the disease will remain as uncalcified osteoid for as long as the deficiency persists. The first skeletal manifestation is a diffuse diminished density of the skeleton that is not distinguishable from osteoporosis (Ortner, 2003; Van Schoor *et al.*, 2007; Kozłowski and Witas, 2012). If the vitamin D deficiency persists for very long periods of time, depending on the remodelling rate, the skeleton can become sufficiently weakened to show radiolucent zones or Looser's zones also referred to as Milkman syndrome in mechanically stressed areas (Aufderheide and Rodríguez-Martin, 1998; Ortner, 2003; Waldron, 2009). In very severe cases mechanical deformity of the skeleton due to fractures and pliability may occur (Ortner, 2003; Brickley *et al.*, 2005; Kozłowski and Witas, 2012).

The deficiency usually affects the whole skeleton, however, the most marked changes are observed in the bones that contain mostly cancellous bone and, therefore, have the highest remodelling rate (Ortner, 2003; Brickley *et al*., 2005). Such areas include the scapula, ribs, sternum, vertebrae, pelvis and sacrum (Ortner, 2003; Brickley *et al*., 2005) with the skull and long bones presenting with a lower remodelling rate, therefore deformities in these areas are quite rare (Brickley and Ives, 2008). The specific changes of osteomalacia are the presence of unmineralized osteoid on the trabeculae of old bone and the gradual replacement of the

normal cortical bone with osteoid that does not mineralize during the active disease stage (Ortner, 2003; Brickley et al., 2005; Brickley and Ives, 2008).

The skull may present with porous lesions on the cranium in the form of fine pitting/diffuse porosity of the cortical surface (Ortner, 2003; Brickley and Ives, 2008). Affected vertebrae appear flattened and display accentuated cupping of the endplates with fewer and more dense trabeculae (Ortner, 2003; Brickley and Ives, 2008). The scapulae may present with pseudo fractures (Looser's zones) affecting the lateral border and inferior-lateral margins of the spinous process (Brickley *et al*., 2005; Brickley and Ives, 2008). This may also include an increased posterior curvature of the scapular blade with buckling and collapsing of the superior border (Brickley *et al*, 2005; Brickley and Ives, 2008). The vertebral bodies may present with buckling, folding and a loss of body height (Brickley and Ives, 2008). Biconcave compression can occur on the inferior and superior surfaces of the vertebral bodies (Ortner, 2003; Brickley *et al.,* 2005; Brickley and Ives, 2008). In severe cases angulating kyphosis and scoliosis may be followed by the deformation of the ribs and sternum (Aufderheide and Rodríguez-Martin, 1998; Ortner, 2003). The ribs show decreased curvature and the sternum is angulated and pushed forward causing the deep and laterally narrowed appearance to the thorax (Ortner, 2003; Brickley and Ives, 2008). Stress fractures can occur in the ribs, the medial cortex of the neck of the femur and the humerus, the pubic rami, and the lateral margin of the scapula (Brickley *et al*., 2005; Brickley and Ives, 2008). These fractures may present as multiple fractures within the same area and in healing internal and external callus is formed.

The most obvious and most characteristic deformity occurs in the pelvis and is referred to as puerperal osteomalacia (Ortner, 2003). Deformation of the pelvis begins with the fourth and fifth lumbar vertebral bodies descending and protruding into the pelvic canal (Ortner, 2003; Brickley and Ives, 2008). The iliac wings folds inward with the pubic rami pushed together and causing anterior projection of the pubic symphysis. The ischial tubera are bent medially and the acetabular floors protrude inward with the sacrum bent angularly forward in the lower portion (Ortner, 2003; Brickley and Ives, 2008). These changes narrow and partially obliterate the pelvic canal, reducing its normal cloverleaf-shaped space (Ortner, 2003). This may lead to difficulties in parturition.

Deformation of the long bones in adults is much less common with the downward bending of the neck of the femur (*coxa vara*) or the humerus (Ortner, 2003). Deformation of the long bone shafts are even less common and are usually affected by anterior-lateral

bending coupled with pseudo fractures (Ortner, 2003; Brickley *et al*., 2005; Brickley and Ives, 2008).

2.2.2.2 Vitamin C deficiency or scurvy

Scurvy is a metabolic disease caused by a vitamin C (ascorbic acid) deficiency (Aufderheide and Rodríguez-Martin, 1998; Ortner, 2003; Brickley and Ives, 2008; Waldron, 2009; Kozłowski and Witas, 2012). Humans are unable to synthesise vitamin C and must acquire it solely through their diet (Mays, 2007; Brickley and Ives, 2008; Waldron, 2009). Vitamin C is available from a large range of foods, especially fresh fruits and vegetables, but also to some extent in milk, fish and meat (Brickley and Ives, 2008; Kozłowski and Witas, 2012). Scurvy affects many organs in the body and has a high mortality rate if the condition is severe. Scurvy is also linked with anaemia as a result of severe haemorrhaging (Ortner, 2003; Brickley and Ives, 2008).

- **Role of vitamin C in the body**

Ascorbic acid acts as a highly effective antioxidant and as an electron donor for enzymes that are essential for the formation of collagen, the main organic matrix of bone (Ortner, 2003; Maat, 2004; Mays, 2007; Waldron, 2009). The exact role that vitamin C plays in collagen synthesis is still unclear, yet it is known that defects in the synthesis have a number of serious health consequences including haemorrhage, delayed wound healing, petechiae (dark spots caused by bleeding under the skin), purpura (rash of dark spots under the skin) and lack of bone formation in juveniles (Aufderheide and Rodríguez-Martin, 1998; Ortner, 2003; Brickley and Ives, 2008). One of the symptoms associated with scurvy is pain and weakness in the limbs which could be related to reduced vitamin C dependent carnitine synthesis which is required to produce energy in the muscles (Brickley and Ives, 2008). Vitamin C also plays a role in blood formation and the metabolism of iron and folate and therefore individuals suffering from severe scurvy might also present with anaemia (Brickley and Ives, 2008). Vitamin C is essential in maintaining the immune system and assists in neutralising or destroying pathogens as well as producing various protective antioxidants (Brickley and Ives, 2008; Waldron, 2009).

Scurvy has a number of effects, yet the severity of these is dependent on the age of the affected individual and the length of the deficiency. Some of the associated symptoms found

in both children and adults include tiredness and lethargy, accompanied by musculoskeletal pain and weakness (Brickley and Ives, 2008; Kozłowski and Witas, 2012).

- **Osteological responses to scurvy**

A vitamin C deficiency, also known as Moller-Barlow's disease, causes general weakness of connective tissue as well as weakness in the walls of blood vessels (Aufderheide and Rodríguez-Martin, 1998; Ortner, 2003; Maat, 2004; Mays, 2007; Kozłowski and Witas, 2012). These weaknesses cause haemorrhaging and if it occurs adjacent to bone this might provoke an osteological response (Aufderheide and Rodríguez-Martin, 1998; Ortner, 2003; Mays, 2007; Brickley and Ives, 2008). Subperiosteal haemorrhaging can occur in a variety of locations throughout the skeleton and can result in the stripping of the periosteum from the bones (Ortner, 2003; Brickley and Ives, 2008; Kozłowski and Witas, 2012). Subperiosteal haemorrhaging may also lead to the formation of ossified haematomas which may present as subperiosteal bone growth on the affected bone surfaces (Van der Merwe *et al.*, 2009). Scurvy can furthermore cause depressed osteoblastic activity resulting in the reduced deposition of osteoid and eventual osteopenia (Brickley and Ives, 2008). The expression of osteological responses will differ between children and adults due to the fact that juvenile skeletons still undergo a large amount of modelling whereas adult skeletons only undergo remodelling (Brickley and Ives, 2008).

- **Scurvy in children**

Prenatal scurvy cannot occur as vitamin C or ascorbic acid passes freely from the mother through the placenta to the foetus, except if the mother suffers from scurvy which can then be passed on to the unborn child (Ortner, 2003). After birth, even if almost no vitamin C is ingested, it takes a few months before the deficiency manifests as a recognizable disease. Infantile scurvy is seldom observed before 4 months of age and reaches its maximum prevalence between 8 and 10 months (Ortner, 2003). Changes in the gums may occur with severe haemorrhaging around newly developed teeth (Brickley and Ives, 2008; Waldron, 2009; Kozłowski and Witas, 2012). Subperiosteal haemorrhaging may be very marked in children due to the fact that the periosteum is not yet firmly attached. Inflammation associated with bleeding will cause bone surface porosity in many areas throughout the skeleton followed by cessation of new bone formation with weakening of bone structures in

turn resulting in metaphyseal fractures (Aufderheide and Rodríguez-Martin, 1998; Ortner, 2003; Brickley and Ives, 2008).

Skeletal changes associated with juvenile scurvy are most marked in the bones that grow most rapidly and include the costochondral junctions of the ribs, the distal metaphysic of the femur, radius and ulna, and the proximal metaphysic of the humerus (Ortner, 2003; Brickley and Ives, 2008; Waldron, 2009). These skeletal changes can either be due to the direct effect of the vitamin C deficiency or due to secondary changes caused by trauma in the vulnerable scorbutic bone and associated blood vessels. The primary lesion consists of the combined effect of severely diminished osteoblastic activity and continued osteoclastic and chondroclastic activity. Complete exclusion of vitamin C from the human diet is quite rare and very small amounts are required to produce a considerable reaction and new bone formation at the periosteum (Brickley and Ives, 2008; Kozłowski and Witas, 2012).

The skull is often involved with subperiosteal haemorrhaging on the frontal bone and particularly on the portion that forms the orbital roof (Ortner, 2003; Waldron, 2009). These haemorrhages are found more often on the orbital rather than the endocranial surfaces and may lead to deposition of porous, hypertrophic bone that covers the thin cortex (Ortner, 2003; Brickley and Ives, 2008). Due to the inflammation of the gums, antemortem tooth loss may be associated with scurvy (Ortner, 2003; Brickley and Ives, 2008). Enamel hypoplastic defects can also be observed as a result of growth stunts and or a diminished immune system resulting in frequent infection and/or illness. The bones adjacent to the osteochondral junction are often transversely fractured, causing an inward dislocation of the sternum and rib cartilage (Ortner, 2003; Brickley and Ives, 2008). The osteochondral joint on the rib side tends to enlarge forming an abnormal flair also referred to as the scorbutic rosary (Ortner, 2003; Brickley and Ives, 2008). The long bones, on the other hand, present with cortical thinning and deposition of reactive periosteal pumice bone coupled with metaphyseal fractures (Ortner, 2003; Brickley and Ives, 2008). In severe cases the proximal metaphysis of the femur caves in only beneath the head and not beneath the greater trochanter, as a direct cause of the weight bearing stresses of the hip. This then results in the depressed angle of the femur neck. When healing occurs no major deformities remain, yet, collapsed metaphyses and dislocated epiphyses may become reunited in an abnormal position which will remain so throughout adult life (Ortner, 2003). Secondary ossification centres of the epiphyses can show marked osteoporosis surrounded by a layer of increased calcified cartilage at the growing periphery. The flat bones rarely present with subperiosteal haemorrhaging and if the

pelvis and scapula become involved lesions will occur in the form of reactive porosity (Ortner, 2003; Brickley and Ives, 2008).

- **Adult scurvy**

Scurvy can also be observed in adult skeletal remains although the potential of observing skeletal lesions of scurvy diminish with increasing age (Ortner, 2003; Maat, 2004). Osteological responses of scurvy tend to be minor in adolescents and even more so in adults. In adults skeletal changes are mainly restricted to transverse fractures at the osteocartilaginous junctions of the ribs with some inflammatory change in the alveolar bone of the jaws due to chronic bleeding of the gums which might also present as periodontal disease (Hirschmann and Gregory, 1999; Ortner, 2003; Brickley and Ives, 2008). New bone formation may be observed in the orbits in the form of cribra orbitalia and the ends of the long bones as subperiosteal bone growth (Aufderheide and Rodríguez-Martin, 1998; Ortner, 2003; Brickley and Ives, 2008; Kozłowski and Witas, 2012). The colour of bones affected by scurvy may be darker and sometimes greyish towards the ends of the long bones (Kozłowski and Witas, 2012). Black symmetrical spots have also been observed at the ankle and knee joints (Maat, 2004; Kozłowski and Witas, 2012). Vertebrae may present with osteopenia and possible biconcave compression and vertebral collapse which in turn could result in kyphosis (Aufderheide and Rodríguez-Martin, 1998; Brickley and Ives, 2008).

Adult scurvy has been recognised worldwide as a serious disease that has caused high mortality amongst people throughout history, usually taking place during times when people were deprived of fresh fruit and vegetables due to war, famine, or in some cases prolonged trips at sea (Maat, 2004; Mays, 2007). Furthermore, because vitamin C is destroyed by high temperatures and by exposure to air, populations which consume mainly cooked foods as opposed to fresh foods will be more likely affected. Two cases where adult scurvy has been positively diagnosed in osteoarchaeological material include a study by Maat (2004) and Van der Merwe *et al.* (2010c). These two studies clearly diagnosed the presence of adult scurvy in osteoarchaeological human remains by linking the osteological manifestations observed with that of the clinical symptoms associated with the deficiency. The first study, done on the remains of 50 Dutch whalers who had been buried during the 17th and 18th centuries on an island of Spitsbergen Archipelago, indicated that dark stains observed on the long bones where synonymous with haemorrhaging clinically associated with scurvy (Maat, 2004). The second study, done on a 19th century mining population from Kimberley, South Africa,

indicated the presence of subperiosteal lesions on the long bones (Van der Merwe *et al.*, 2010c). These lesions were histologically diagnosed as being ossified haematomas, again supporting the clinical literature in the disease manifestation.

2.2.2.3. Iron deficiency anaemia

As has been mentioned before, many of the skeletal pathologies associated with metabolic diseases are not easily distinguishable in terms of their causes and in many cases an individual may present with more than one deficiency. Anaemia often goes hand in hand with other metabolic diseases, especially scurvy. Dietary iron's extensive involvement of bone marrow tissue in order to regenerate blood cell supply results in secondary effects on bone cells and structures (Brickley and Ives, 2008; Kozłowski and Witas, 2012). The specific diagnosis of iron deficiency anaemia in the past is complex and has a wide range of potential causative factors, which includes genetic mutations which affect red blood cell production, shortages of iron in the diet, intestinal parasitic infections compromising iron absorption in the intestinal tract, infections which triggers the body to reduce serum iron levels, intensive haemorrhage and/or excessive blood loss (Stuart-Macadam, 1989; Steckel, 2005; Brickley and Ives, 2008; Kozłowski and Witas, 2012).

Iron is essential for many bodily functions and is especially an essential element in haemoglobin, thus enabling the sufficient transport of oxygen throughout the body tissues (Larsen, 1997; Steckel, 2005; Kozłowski and Witas, 2012). Iron absorption from the diet is dependent upon its source within the foods consumed, either being heme or nonheme (Larsen, 1997). Generally, heme sources of iron are efficiently absorbed, with meat being the best source (Larsen, 1997). Iron in meat does not require processing in the stomach and the amino acids from the digestion of meat help to enhance iron absorption (Larsen, 1997). Nonheme sources include most of the plant sources and the iron contained in it is generally poorly absorbed with various substances found in plants inhibiting iron absorption. These include phytates found in many nuts, cereals, and legumes (Larsen, 1997). A number of foods are known to enhance the absorption of iron, the most common being vitamin C enriched foods (Larsen, 1997). For this reason vitamin C and iron deficiency may often occur simultaneously.

Iron deficiency can also be caused by non-dietary factors. Children with low birth weights can be predisposed to iron deficiency whereas blood loss, haemorrhage, parasitic

infections and chronic diarrhoea, and genetic diseases like sickle cell anaemia may also result in the appearance of the disease (Stuart-Macadam, 1989; Larsen, 1997).

- **Osteological responses to anaemia**

Skeletal manifestation of the disease is not always prevalent, but where it is these changes are part of a generalized syndrome which may include porotic hyperostosis (lesions on the outer table of the cranial vaults), and cribra orbitalia (lesions found in the roof areas of the orbits) (Aufderheide and Rodríguez-Martin, 1998; Larsen 1997; Ortner, 2003; Kozłowski and Witas, 2012). These lesions are usually caused by marrow hyperplasia and present as areas of pitting and/or porosity on the external surfaces of bone as a result of the expansion of the diploë (Ortner, 2003; Walker *et al.,* 2009). Severe cases of cribra orbitalia present with deep pores in the orbital roofs whereas porotic hyperostosis may have a "hair-on-end" appearance which is especially observable in radiographs (Ortner, 2003). It is argued that cribra orbitalia is one of the earliest manifestations of anaemia with porotic hyperostosis only occurring as a secondary lesion. Active porotic hyperostosis is usually found in juvenile remains whereas in adult remains it mostly already underwent remodelling and healing (Stuart-Macadam, 1985). This is due to the fact that marrow spaces in young children are completely occupied with red marrow and with an expansion due to an increase in marrow cells (a direct response to anaemia) increased stress will be placed on the bone creating the osteological reaction (Stuart-Macadam, 1985). It may therefore be possible that anaemia acquired after childhood will affect the bone to a lesser extent. New research suggest (Walker *et al.,* 2009; Oxenham and Cavill, 2010) that other possible causes for this condition should also be considered, which in turn may help to explain why differences in frequencies observed in adults and juveniles occur.

Even though cribra orbitalia and porotic hyperostosis have repeatedly been ascribed to anaemia (Stuart-Macadam, 2005) it has recently been suggested by Walker *et al.* (2009) that the causes might rather be due to a combination of deficiency in vitamins B12 (cobalamin) and B9 (folic acid), poor sanitation, and infectious diseases. Walker *et al*. (2009) argued that this condition decreases the production of mature red blood cells and therefore cannot cause bone marrow hyperplasia. However, Oxenham and Cavill (2010) suggested that even though iron deficiency decreases the production rate for functional erythrocytes the actual erythropoietic activity is increased. This suggests that bone marrow hyperplasia and the hypertrophic lesions associated with it can indeed occur as a result of iron deficiency

anaemia. These authors go on to provide alternative reasons for the appearance of hypertrophic lesions suggesting that it may either be caused by haemolytic anaemia (thalassemia, sickle cell anaemia, and malaria) or megaloblastic anaemia (chronic dietary deficiency, malabsorption, deficiencies of the vitamins B12 and B9, and gastrointestinal parasite infections (Walker *et al.*, 2009; Oxenham and Cavill, 2010).

It should, however, be emphasized that iron deficiency anaemia is but one of many possible causes of cribra orbitalia (Wapler *et al.*, 2004). A wide variation in the frequencies of orbital versus nonorbital lesions exists across human populations (Larsen, 1997). It has also been suggested that other metabolic diseases, infection, and cancer can produce porous and hypertrophic lesions in bone, yet porotic hyperostosis of the skull is most commonly linked to some type of anaemia (Ortner, 2003). A study by Wapler *et al.* (2004) showed that in at least 56.5% of the cases where cribra orbitalia could be observed there were no histological features indicating changes as a result of anaemia. They provided several alternative reasons for the wide-spread occurrence of cribra orbitalia. These include inflammation (osteitis, periostitis and hypervascularisation) which can be caused by the spreading of other infections (this may include sinusitis, tooth abscesses and other oral infections, nasophyrangeal infections and suppurating skin inflammations); other undetermined causes such as other underlying metabolic diseases (scurvy, rickets and osteoporosis), localised pressure as a result of an enlarged organ (for example the lacrimal gland); and postmortem erosion as a factor of taphonomic alterations (Wapler *et al.*, 2004).

2.2.2.4. Osteoporosis

Osteoporosis is another metabolic disease, characterised by a decrease in bone mass and a constant increase in skeletal frailty with a heightened risk of fractures (Brickley and Ives, 2008; Kozłowski and Witas, 2012). Osteoporosis poses serious health issues, even today, and has been called one of the most important skeletal stress markers of the past (Kozłowski and Witas, 2012). Osteoporosis occurs due to an imbalance between the processes of bone formation and resorption resulting in a net loss of bone density and integrity (Aufderheide and Rodríguez-Martin, 1998; Kozłowski and Witas, 2012). "Until early adult life the skeleton is in positive balance, that is to say, a larger amount of bone is formed than is lost and the total skeletal mass increases until it reaches a maximum at about the age of 25-30" (Waldron, 2009: 118). Osteoporosis and its precursor osteopenia can occur in a wide range of circumstances and can either be classified as being 'primary', 'secondary'

or 'post-menopausal' (Brickley and Ives, 2008; Kozłowski and Witas, 2012). Primary osteoporosis usually refers to the age-related form of the disease whereas the secondary refers to the underlying pathology, trauma or dietary insufficiency which gives rise to osteoporosis (Brickley and Ives, 2008; Kozłowski and Witas, 2012).

- **Primary and post-menopausal osteoporosis**

Bone apposition takes place on the endosteal and subperiosteal surfaces well into the third and fourth decades of life, peaking at about 25-35 years of age (Larsen, 1997; Waldron, 2009). Yet, at an age of around 40, bone commences resorption endosteally while continuing deposition periosteally. The imbalance between the amount of bone loss and bone gain on these surfaces results in an overall reduction of bone tissue during and following the fifth decade of life (Larsen, 1997; Aufderheide and Rodríguez-Martin, 1998; Kozłowski and Witas, 2012). Adult bone loss or osteoporosis increases the risk of bone fractures during older age (Aufderheide and Rodríguez-Martin, 1998; Ortner, 2003; Waldron, 2009; Kozłowski and Witas, 2012). There are two types of bone loss due to osteoporosis that are identified clinically; these include Type 1 and Type 2 osteoporosis (Aufderheide and Rodríguez-Martin, 1998; Ortner, 2003; Waldron, 2009; Kozłowski and Witas, 2012). Type 1 refers to osteoporosis following menopause in women, whereas Type 2 refers to osteoporosis as a result of old age in both men and women (Aufderheide and Rodríguez-Martin, 1998; Ortner, 2003; Waldron, 2009; Kozłowski and Witas, 2012). Overall women are more at risk since they may suffer from both Type 1 and Type 2 osteoporosis at the same time. The rate of bone loss in human populations is highly variable and nutritional status plays an integral role in the prevalence of the disease (Larsen, 1997; Waldron, 2009; Kozłowski and Witas, 2012). Clinical evidence indicates that individuals with low calcium intakes, alcohol abuse, smoking, high protein consumption, caffeine excess, and unfavourable body weights are more prone to bone loss (Larsen, 1997; Waldron, 2009).

- **Causes of osteoporosis**

Increased age results in an imbalance in bone remodelling and bone loss with both males and females losing 20-30% of trabecular and cortical bone (Brickley and Ives, 2008). Increases in osteoclastic resorption depths can perforate trabecular elements contributing to structural weakness and removing the bone surface necessary for bone formation, preventing the means of improving skeletal structural support (Brickley and Ives, 2008). Overall very

little is known of the effect of age on bone cell function. Aging may adversely affect osteoprogenitor cells via reducing the number of existing cells as well as limiting the capacity to differentiate new bone cells (Brickley and Ives, 2008). Aging may also hinder the detection and response to accumulating fatigue damage, adding to the increasing fragility in the skeleton. A reduced intake of calcium, vitamin C, amino acids, and synthesis of vitamin D further exacerbates adverse aging effects on skeletal health (Brickley and Ives, 2008; Waldron, 2009; Kozłowski and Witas, 2012).

In menopausal women oestrogen can restrain bone turnover and limits the resorptive capability of mature osteoclasts. With loss of oestrogen bone remodelling increases and bone resorption increases to 90% compared to bone formation at a meagre 45% (Brickley and Ives, 2008). Bone loss can occur following a decrease in ovarian function within the decade prior to the final cessation of menses. Oestrogen loss may further remove cell sensitivity to strains from physical activity potentially further contributing to bone loss (Brickley and Ives, 2008).

- **Osteological responses to osteoporosis**

Reduction of bone quantity as well as quality from primary and post-menopausal osteoporosis increases bone fragility and consequently require only minimal trauma in order to result in fracture (Aufderheide and Rodríguez-Martin, 1998; Brickley and Ives, 2008; Waldron, 2009; Kozłowski and Witas, 2012). Three sites in the skeleton are prone to osteoporosis-related fractures and include the distal radius, vertebrae and the femoral neck (Aufderheide and Rodríguez-Martin, 1998; Brickley and Ives, 2008; Kozłowski and Witas, 2012). Nevertheless, age-related osteoporosis can increase the susceptibility to fracture throughout the skeleton with post-menopausal women presenting with an even greater risk (Brickley and Ives, 2008; Kozłowski and Witas, 2012).

The distal radius is largely composed of cortical bone, yet the proportions of trabecular bone increase towards the epiphysis in order to protect the joint (Brickley and Ives, 2008). Osteoporosis causes cortical thinning; intra-cortical porosity and trabecular bone loss severely hampers the strength of the distal radius, therefore predisposing this area to easy fracturing (Brickley and Ives, 2008; Kozłowski and Witas, 2012). Fractures to the distal radius are referred to as Colles' fractures and are typically derived from falls onto an outstretched hand, resulting in posterior displacement of the distal part of the radial shaft (Ortner, 2003; Brickley and Ives, 2008; Kozłowski and Witas, 2012). These fractures are not solely limited to individuals suffering from osteoporosis; although osteoporotic condition

increases the changes of such a fracture should an injury to this area occur. Colles' fractures present a metaphyseal fracture to the distal radius and can result in dorsal displacement, trabecular compression and in severe fractures reduced function of the wrist (Mays, 2006).

Vertebral fractures are an important manifestation of osteoporosis in both post-menopausal females and aging men. Trabeculae in the vertebral body provide most of the bone strength and a decrease in bone mass will result in significantly reduced bone strength (Brickley and Ives, 2008). In osteoporotic individuals activities such as lifting, coughing, laughing and jumping as well as falling can increase vertebral compression and may result in vertebral fracturing (Brickley and Ives, 2008). Three vertebral fractures can occur, namely wedge factures where the lower thoracic and lumbar vertebrae show either an anterior or lateral collapse which in some cases may result in kyphosis; compression fractures which entail the severe compression of the whole vertebral body and are usually found in single vertebrae; and concave ballooning fractures which result in the compression of the central portion of the superior and inferior surfaces of multiple vertebrae (Melton *et al.*, 1988; Aufderheide and Rodríguez-Martin, 1998; Brickley and Ives, 2008; Kozłowski and Witas, 2012).

Fractures of the femur occur in two locations affecting both cortical and trabecular bone in the form of intra-capsular fractures affecting the femoral neck and extra-capsular fractures affecting the trochanteric region (Melton *et al.*, 1988; Brickley and Ives, 2008; Kozłowski and Witas, 2012).

- **Secondary osteoporosis**

There are three broad mechanisms that can result in the skeletal manifestations of secondary osteopenia with osteological responses similar to that found in primary osteoporosis (Brickley and Ives, 2008). These include trauma or pathology which affect a specific limb or the spinal cord limiting mobility and leading to disuse and eventual atrophy; diseases like polio, diabetes, hyperthyroidism and kidney disorders; and dietary inadequacy which disrupts mineral metabolism (Brickley and Ives, 2008; Kozłowski and Witas, 2012). One such manifestation can be seen in the uncommon occurrence of juvenile osteoporosis. Juvenile osteoporosis is the result of a combination of factors which includes heritability of the condition, pathological or traumatic injuries, and overall deficiency of vitamins and minerals including calcium, vitamin C and vitamin D (Jowsey, 1977; Norman, 1993). The

osteological effects of the disease usually present in the vertebral column and include vertebral fractures and Scheurmann's kyphosis (Jowsey, 1977; Norman, 1993).

2.2.2.5. Paget's disease of bone

Paget's disease of bone is a chronic disease that results in disruption of bone remodelling in affected bones, characterised by gross deformity and enlargement of parts of the skeleton (Brickley and Ives, 2008; Ralston, 2008; Kozłowski and Witas, 2012). The exact causes of the disease remain unknown and several infectious diseases (possibly viral infections), genetic predisposition, environmental conditions and a number of metabolic diseases are usually discussed as possible causes (Siris, 1993; Roodman, 1996; Brickley and Ives, 2008; Kozłowski and Witas, 2012). The condition may present in single bones (monostotic), or between two and four bones (oligostotic), or in at least five bones (polyostotic) (Brickley and Ives, 2008, Ralston, 2008). Any bone can be affected but the most commonly involved bones are those of the pelvis, lower spine, cranium and the long bones of the leg (Brickley and Ives, 2008; Kozłowski and Witas, 2012). Three stages are associated with the disease. In the first phase, bony changes are characterised by an increase in resorption caused by a massive increase in osteoclastic activity and is referred to as the osteolytic phase (Brickley and Ives, 2008). The second phase of the condition is characterised by marked bone formation and this has been referred to as the intermediate phase. This phase includes a very characteristic pattern of cement lines developing within the bone tissue, creating a mosaic appearance. The third phase is marked by the development of sclerotic bone and a decrease in vascularity and is also referred to as the sclerotic phase. During the final stages of this phase complete cessation of osteoclastic activity and only minimal osteoblastic activity occurs (Brickley and Ives, 2008; Kozłowski and Witas, 2012).

- **Osteological responses in Paget's disease**

The pelvis is an early skeletal site for the development of lesions with the ilium most frequently affected, specifically in the region of the sacroiliac joint (Ortner, 2003; Brickley and Ives, 2008; Kozłowski and Witas, 2012). Involvement of the pelvis is not bilateral and the right side is more often affected than the left side. Any bones of the skull can be involved but areas most commonly affected are the cranial vault and the skull base (Ortner, 2003; Brickley and Ives, 2008; Kozłowski and Witas, 2012). Lesions can be single or multiple and is characterised by prevailing bone resorption resulting in marked thinning of the inner and

outer table which is referred to as osteoporosis circumscripta (Brickley and Ives, 2008; Ralston, 2008). The thin tables present a porous surface with the outline of the lesion presenting a wavy line. These lesions may cross the cranial sutures and have a sharp interphase against adjacent uninvolved bone (Ralston, 2008). With advanced stages of the disease the cranial vault may exhibit distinct thickening of the calvarium by endocranial and external bone formation and may reach up to several centimetres in thickness. In the skull base thickening occurs at the endocranial surface with encroachment upon the cranial cavity. The auditory bones can also become affected and may lead to eventual deafness (Ralston, 2008). The jaws are not commonly affected by the disease, yet abnormal deposit of radiodense cementum around the dental alveoli has been reported (Ortner, 2003).

Deformities in the long bones follow characteristic patterns with the femur bowing laterally whereas in the tibia bowing occurs anteriorly (Siris, 1993; Brickley and Ives, 2008). The earliest forms of the disease can be seen in the femur and tibia as purely lytic osteoclastic resorption involving the entire thickness of the cortex (Ortner, 2003). Later stages of the disease is characterised by the bowing of the femur and tibia and is a direct result of complete or incomplete transverse pathological fractures which is an expression of the mechanical inferiority of the abnormal bone formed in the lesions (Ortner, 2003; Kozłowski and Witas, 2012). The epiphyses and metaphyses can also become involved and may show coarsening and focal deposition of pumice bone. Secondary osteoarthritis may also affect these areas in severe cases. The spine is also often involved and present lesions that gradually decrease from the sacrum to the cervical vertebrae. The bodies of the vertebrae are most commonly involved however lesions may spread to the adjacent neural arches as well (Ortner, 2003).

Paget's disease should not be confused with osteopetrosis, which is a genetic condition in which osteoclasts do not reabsorb bone normally and as a result bone formation and remodelling are impaired (Brickley and Ives, 2008). Unlike Paget's disease which only affects one bone at a time or up to five bones in the case of polyostotic Paget's, osteopetrosis affects the whole skeleton (Aufderheide and Rodríguez-Martin, 1998; Ortner, 2012).

2.2.2.6. Beriberi

A disease labelled under the 'metabolic syndromes', which does not have any known direct skeletal manifestations, is beriberi. Beriberi is the Singhalese word in the Sri Lankan language for "*I cannot, I cannot*" and is caused by a vitamin B1 or thiamine deficiency (Saunders, 1974; Thurnham, 1978; Gibney *et al.*, 2002; Truswell and Milne, 2002). This

nutritional disease was especially common in historical times throughout south-east Asia following the introduction of the steam-powered mill that made highly polished (therefore thiamine-depleted) white cereals available (Thurnham, 1978; Carpenter, 2000; Gibney *et al*., 2002). A diet mainly consisting of polished white rice or wheat flour and little to no fresh meat and whole-grain cereals meant that large numbers of Asian people suffered from beriberi (Ball, 1998; Carpenter, 2000; Truswell and Milne, 2002; Doung-ngern *et al.,* 2007).

Thiamine is a water soluble vitamin found mainly in the skeletal muscles with lesser concentrations in the heart, liver, kidneys and brain (Murphy, 2005). Its main function is the breaking down of carbohydrates and it is therefore essential for normal glucose metabolism (Murphy, 2005). Thiamine can be found in food sources like yeast extracts, legumes, pork, unpolished rice or brown rice, peas, wheat flour etc. (Ball, 1998; Suskind, 2009). On the other hand, consumption of certain food sources may actually inhibit the bioavailability of thiamine (Ball, 1998). Such foods can be divided into two categories; thermolabile enzymes (thiaminases) and thermostable substances such as tannins in plants (Ball, 1998). Thermolabile enzymes can be found in raw shellfish and raw or fermented freshwater fish (these enzymes are destroyed once the food sources are cooked), whereas thermostable substances are found in especially teas and coffees, as well as rice bran, nuts and a variety of fruits and vegetables (Ball, 1998). Food preparation techniques using a high pH or high temperatures may also denature the thiamine molecule making it less available for uptake (Carpenter, 2000; Suskind, 2009).

Beriberi can develop within two to three months and can be fatal without treatment (Doung-ngern *et al.,* 2007). Today isolated cases of beriberi can still be observed amongst individuals consuming a diet rich in carbohydrates and low in thiamine and is especially prevalent amongst patients suffering from chronic alcoholism (Ball, 1998; Smith, 1998; Gibney *et al*., 2002; Truswell and Milne, 2002; Thomson and Marshall, 2006; Doung-ngern *et al.,* 2007). Cases of beriberi have also been reported in patients who have undergone bariatric or gastric bypass surgeries (Towbin *et al.,* 2004). Beriberi can result in three distinct syndromes that may present with distinct symptoms or as mixed symptoms (Smith, 1998; Gibney *et al*., 2002). Firstly, it can present as a chronic peripheral neuritis which in the case of "wet" beriberi (cardiovascular) is associated with heart failure and oedema and in the case of "dry" beriberi (neurological) as muscular weakness and atrophy (Ball, 1998; Smith, 1998; Carpenter, 2000; Truswell and Milne, 2002; Thompson and Marshall, 2006). Secondly, it may manifest as acute pernicious beriberi or shoshin beriberi (a severe form of 'wet' beriberi), in which heart failure and metabolic abnormalities predominate, with little evidence

of peripheral neuritis (Smith, 1998; Carpenter, 2000; Bates, 2007). Thirdly, Wernicke's encephalopathy with Korsakoff's psychosis, a thiamine-responsive condition associated with alcohol and narcotic abuse, may occur (Carpenter, 2000; Thomson and Marshall, 2006; Bates, 2007).

It has already been mentioned that a large number of the Chinese employed on the Witwatersrand mines suffered from beriberi upon arrival and that many eventually succumbed to this nutritional disease. Although there is no direct association with this nutritional disease and skeletal pathology seen in metabolic bone diseases, secondary skeletal pathology might still be observed. This is especially the case with dry beriberi where muscular atrophy and disuse of limbs (Carpenter, 2000) are some of the common symptoms suffered by patients that may manifest as secondary osteological lesions.

- **Alcoholism**

In the case of beriberi acquired as a result of chronic alcoholism, secondary manifestations of the disease can include direct effects on mineral and vitamin absorption and secretion (except for thiamine deficiency which is readily associated with severe cases of alcoholism and the manifestation of Wernicke-Korsakoff's encephalopathy). Alcoholism in itself can cause deficiencies of multiple vitamins and minerals. In patients suffering from chronic alcoholism the basal metabolic rate is increased, resulting in an inadequate energy intake and availability (Truswell, 2002). In heavy drinkers, 10-30% of the total energy intake comes from alcohol itself, yet alcoholic beverages contain no protein and very few micronutrients. Appetite may also be suppressed in heavy drinkers as a result of alcoholic gastritis. Effectively a diet that would normally provide all the essential nutrients becomes displaced with a nutrient-poor source of calories (Truswell, 2002). Secondary or conditioned nutritional deficiencies are therefore readily observed in alcohol dependant individuals.

Some of the vitamin and mineral deficiencies seen in chronic alcoholism may have indirect influences on skeletal health. Such vitamins include B-vitamins (B1 (thiamine), B2 (riboflavin), B3 (niacin), B6 (pyridoxine), and B12 (cobalamin); folates (specifically folic acid); iron; zinc; calcium; and vitamin D (Ishii and Nishihara, 1981; Mobarhan *et al*., 1984; Rodriguez-Moreno *et al.*, 1997; Ball, 1998; Truswell, 2002; Groenen *et al.*, 2004a; Groenen *et al.*, 2004b; Suskind, 2009).

- **B vitamins (B1, B2, B3, B6, B9 and B12)**

As discussed above, vitamin B1 or thiamine is a water soluble vitamin responsible for glucose metabolism by breaking down carbohydrates. A deficiency in thiamine, as already mentioned, can result in beriberi or Wernicke-Korsakoff's encephalopathy (Ball, 1998; Carpenter, 2000; Truswell and Milne, 2002; Bates, 2007; Suskind, 2009). Vitamin B2 or riboflavin is part of two important coenzymes, flavin mononucleotide and flavine dinucleotide, both of which act as oxidizing agents (Truswell and Milne, 2002; Rivlin, 2007). Deficiency in vitamin B2 causes minor symptoms in humans as the human body has the ability to conserve riboflavin relatively effectively (Truswell and Milne, 2002). Vitamin B2 deficiency is therefore more commonly seen alongside other nutrient deficiencies, especially pellagra (Truswell and Milne, 2002). Dietary riboflavin deficiency leads to both a decrease in iron absorption and an increase in iron loss from the intestine (Rivlin, 2007). As a result of this, one of the clinical symptoms of vitamin B2 deficiency is anaemia, which if very severe may result in skeletal manifestations such as porotic hyperostosis and cribra orbitalia (Truswell and Milne, 2002; Rivlin, 2007).

Vitamin B3 or niacin, or its nicotinamide nucleotide coenzymes, function as proton and electron carriers in a wide variety of oxidation-reduction processes like energy release from carbohydrates, fatty acids and amino acids, as well as the synthesis of amino acids, fatty acids and pentose for nucleotide and nucleic acid production (Ball, 1998; Truswell and Milne, 2002; Suskind, 2009). A deficiency in niacin will result in pellagra which is associated with dermatological lesions, diarrhoea and dementia (Ball, 1998; Truswell and Milne, 2002; Brenton and Paine, 2007; Suskind, 2009). Deficiencies in niacin are most commonly observed in poorer communities who subsist mainly on maize (Brenton and Paine, 2007; Kirkland, 2007). Niacin in itself is in a "niacytin" state which means that it is not biologically available for absorption (Ball, 1998; Truswell and Milne, 2002; Brenton and Paine, 2007). Only when tryptophan is present will the human body be able to absorb niacin effectively. Cereals do contain small amounts of tryptophan of which maize contains the least. However, most proteins contain enough tryptophan to maintain an adequate niacin status if the rest of the diet is completely devoid of niacin (Ball, 1998; Truswell and Milne, 2002). Low levels of niacin will, however, create a demand for more tryptophan which in turn means that the body will have to have access to more iron (Brenton and Paine, 2007). People consuming a staple diet of maize, with very little or no protein, will therefore be more prone to developing niacin deficiency and therefore pellagra (Brenton and Paine, 2007). Skeletal pathologies associated with pellagra include cortical bone loss in the ribs, periostitic lesions, dental caries, extreme

alveolar bone loss, cribra orbitalia and cranial pitting (Brenton and Paine, 2007). A deficiency in tryptophan, on the other hand, may lead to low plasma zinc levels and low bone zinc which may further inhibit the tryptophan to niacin conversion (Brenton and Paine, 2007). As will be discussed below, zinc is mainly responsible for the effective growth and development of humans. It is for this reason that a deficiency in niacin during pregnancy has been suggested to result in spina bifida in the foetus (Groenen *et al.*, 2004b).

Vitamin B6 (pyridoxine) works as a coenzyme for many enzymes involved in amino acid metabolism, including the biosynthesis of niacin from tryptophan. A deficiency in vitamin B6 only may lead to B6 responsive anaemia but because of its interlinking functions may also therefore lead to a deficiency in vitamin B3 (Ball, 1998; Truswell and Milne, 2002; Dakshinamurti and Dakshinamurti, 2007; Suskind, 2009).

Vitamin B9 (folic acid) is essential for the metabolism of certain amino acids and DNA synthesis (Ball, 1998; Bailey, 2007). Folic acid works in combination with vitamin B12 and therefore a deficiency in vitamin B12 will often result in a deficiency in folic acid as well (Ball, 1998; Suskind, 2009). The most common cause of folic acid deficiency is a lack of dietary intake (Suskind, 2009). Although many foodstuffs, like liver, greens and yeast contain folic acid it can easily be destroyed by heat during cooking (Bailey, 2007; Suskind, 2009). The consumption of a cereal based diet can also predispose an individual to developing a folic acid deficiency as carbohydrate type foods only contain trace elements of folate and iron (Rose, 1982). Excessive alcohol consumption further accelerates megaloblastic changes therefore increasing the need for iron and folic acid availability (Rose, 1982). The body also stores very small amounts of folic acid in comparison with vitamin B12, meaning that a deficiency of folic acid can develop much more easily (Suskind, 2009). A deficiency in folic acid leads to a lack of adequate DNA replication which results in impaired cell division, especially in the haemopoietic tissue of bone marrow and epithelial cells of the gastrointestinal tract (Ball, 1998; Bailey, 2007). A deficiency can therefore develop into megaloblastic anaemia within 4 to 5 months (Ball, 1998; Bailey, 2007; Suskind, 2009). A poor folate status during early pregnancy has also been related to the occurrence of neural tube defects (Ball, 1998; Groenen *et al.*, 2004a; Groenen *et al.*, 2004b; Bailey, 2007; Suskind, 2009). Skeletal manifestation of folic acid deficiency can therefore appear in the form of hypertrophic bone lesions in the case of megaloblastic anaemia being present and/or developmental defects such as notochord defects, cleft neural arches, sacralisation, and spina bifida (Ball, 1998; Groenen *et al.*, 2004a; Groenen *et al.*, 2004b; Bailey, 2007). Some authors also attribute spondylolysis to developmental defects which may be related to a folic acid and

vitamin B12 deficiency of the foetus during early pregnancy as a result of maternal deficiency in these vitamins (Aufderheide and Rodríguez-Martin, 1998; Haun and Kettner, 2005; Ward and Latimer, 2005; Ward *et al*., 2007).

Vitamin B12 or cobalamin, and its coenzymes methylcobalamin and deoxyadenosylcobalamin, is mainly responsible for proper neurological functioning and blood formation. It is also responsible for DNA synthesis and regulation, fatty acid synthesis, folate metabolism, and overall cell metabolism (Ball, 1998; Truswell and Milne, 2002; Green and Miller, 2007). An inadequate intake is not the usual cause of vitamin B12 deficiency as vitamin B12 reserves can remain in the body for up to 6 years (Truswell and Milne, 2002). A dietary deficiency in vitamin B12 can however arise in adults consuming a strict vegan diet or in non-vegetarian subjects consuming grossly inadequate diets, primarily due to poverty (Truswell and Milne, 2002; Green and Miller, 2007). A more common cause of vitamin B12 deficiency is malabsorption due to gastric atrophy (causing pernicious or Addisonian anaemia), gastric diseases, parasites and gastric bypass surgery (Ball, 1998; Truswell and Milne, 2002; Green and Miller, 2007). A vitamin B12 deficiency results in either the manifestation of megaloblastic anaemia and/or neurological dysfunction or neuropathy (Ball, 1998; Truswell and Milne, 2002; Green and Miller, 2007; Suskind 2009. Skeletal manifestation may therefore appear as hypertrophic bone lesions and developmental defects such as spina bifida and notochord defects (Truswell and Milne, 2002; Groenen *et al.*, 2004a; Groenen *et al.*, 2004b; Green and Miller, 2007; Walker *et al.*, 2009; Oxenham and Cavill, 2010).

- **Iron**

It has already been mentioned that iron is responsible for many metabolic functions and is especially an essential element in synthesis of haemoglobin which is necessary for the sufficient transport of oxygen throughout the body tissues (Larsen, 1997; Steckel, 2005; Kozłowski and Witas, 2012). The supply of iron is greatly influenced by the composition of the diet and can be derived from either heme- or nonheme foods (Larsen, 1997; MacPhail; 2002). Heme iron are mainly derived from haemoglobin and myoglobin in meat whereas non-heme iron are found in iron salts, iron in other proteins (plant proteins), and iron derived from processing or storage methods (MacPhail, 2002). In the past an iron deficiency was thought to be largely due to an abnormal loss of iron through, for example blood loss and loss as a result of intestinal parasites, rather than as a result of insufficient iron supply. It is now

perceived that the poor bioavailability of iron in largely cereal based diets is a major cause of iron deficiency, especially in developing countries (MacPhail, 2002). Non-heme iron can be more readily absorbed when promoters like ascorbic acid (vitamin C) are consumed in association, whereas inhibitors like phytates and polyphenols (contained in most cereals) can result in a decreased absorption of the non-heme iron available (MacPhail, 2002). Ethanol has been shown to increase the urinary excretion of iron (Rodriguez-Moreno *et al.*, 1997) which in combination with malnutrition and additional vitamin deficiencies, often observed in chronic alcoholics (Truswell, 2002) may result in iron deficiency anaemia which may invariable present as hypertrophic lesions on the cranium and orbital roofs (Walker *et al.*, 2009; Oxenham and Cavill, 2010).

- **Zinc**

It has been shown that chronic alcoholism and patients suffering from liver cirrhosis have increased levels of zinc and iron excretion (Rodriguez-Moreno *et al.*, 1997; Samman, 2002). Zinc is an essential micronutrient responsible for human growth, development and immune system function (Suskind, 2009). The primary dietary sources containing zinc include animal product such as meat, seafood, and milk (Suskind, 2009). Zinc absorption may be inhibited by the consumption of large quantities of dietary phytates and fiber as zinc bonds with these elements, decreasing absorption and/or bioavailability of the vitamin (Carpenter, 2000; Samman, 2002; Suskind, 2009). Zinc deficiency has also shown to increase rates of infection and diarrhoea as a result of a diminished immune function (Samman, 2002; Suskind, 2009). Furthermore, zinc deficiency may result in the abnormal development and growth of a child, and has been shown to result in developmental defects such as spina bifida and notochord defects (Groenen *et al.*, 2004a; Groenen *et al.*, 2004b).

- **Calcium and vitamin D**

Calcium and vitamin D are both essential for the formation of bone osteoid and therefore play an important role in the modelling and remodelling of bone (Aufderheide and Rodríguez-Martin, 1998; Ortner, 2003; Waldron, 2009; Kozłowski and Witas, 2012). It has been shown that persons suffering from chronic alcoholism exhibit a decrease in plasma 25(OH)D3 levels, intestinal calcium absorption as well as a decrease in bone mineral content (Mobarhan *et al.*, 1984; Norman and Henry, 2007). Bone abnormalities may, however, be caused by a combination of factors including; malabsorption of calcium and vitamin D as a

result of a diminished liver and kidney function and gastrointestinal complications; poor dietary intake in general; lack of sunlight exposure due to constant intoxication; and the direct effects of ethanol on the metabolism of vitamin D (Mobarhan *et al.*, 1984; Truswell, 2002). Ethanol is known to supress osteoblast activity and is also suggested to increase osteoclast activity and therefore induces osteopenia and in severe cases osteoporosis (Chakkalakal, 2005). As has been mentioned already, a deficiency in vitamin D and calcium may invariably lead to rickets/osteomalacia and/or osteoporosis. Each of these diseases may result in the deformation of bones and/or the appearance of pseudo-fractures which may be observable in the skeleton.

2.2.3 Dental pathology

2.2.3.1. Dental caries

Dental caries is the most common dental disease affecting humankind (Roberts and Manchester, 1995; Waldron, 2009) and is more frequently reported for archaeological populations than any other (Roberts and Manchester, 1995). Dental caries occur via a process that is characterised by centralised demineralization of dental hard tissue by organic acids produced by bacterial fermentation of dietary carbohydrates, especially sugars (Roberts and Manchester, 1995; Larsen, 1997; Lukacs, 2012). If the right combination of plaque bacteria and sugars, especially sucrose, occurs, then the acids produced will demineralise the teeth and leave cavities (Roberts and Manchester, 1995; Hillson, 1998). Dental caries can manifest in different stages of severity from small enamel opacities to extensive cavitations involving partial or complete loss of tooth crowns and roots (Roberts and Manchester, 1995; Larsen, 1997; Hillson, 1998; Ortner, 2003). This disease is considered to be a multi-factorial disease that is affected by diet, oral hygiene, bacteria, dental morphology, trace minerals such as fluoride, and even sex, genetic and socio-economic differences (Lukacs, 2012). Dental caries can be used as an indicator of diet and subsistence economy (Lukacs, 2012). Intensified agriculture has seen a dramatic increase in the frequency of dental caries as a result of increased consumption of carbohydrate rich cereals (Roberts and Manchester, 1995; Hillson, 1998, Ortner, 2003; Lukacs, 2012). Dental caries has also been associated with vitamin D and calcium deficiencies and may therefore also be an additional indicator for metabolic bone diseases and nutritional deficiencies (Brickley and Ives, 2008).

2.2.3.2. Ante-mortem tooth loss

Teeth may become lost due to processes before and after death. In cases where the alveolar bone (tooth socket) presents with remodelling, this may be attributed to ante-mortem tooth loss (AMTL; Waldron, 2009; Roberts and Manchester, 2010). AMTL loss may occur as a result of untreated dental diseases, such as caries, periodontal disease and abscesses, or due to trauma, dental attrition or even cultural practices (Hillson, 1998; Morris, 1998; Waldron, 2009; Roberts and Manchester, 2010). In cases where trauma and cultural practices of tooth extraction can be excluded as causes for tooth loss, the incidence of AMTL should reflect the incidence of dental disease within a population (Roberts and Manchester, 2010). The occurrence of AMTL can also be influenced by tooth attrition. In cases where severe dental wear occur as a result of attrition the destruction of enamel will lead to the eventual exposure of the pulp chamber. The pulp chamber would therefore be much more susceptible to bacterial infections which in turn may result in tooth loss (Hillson, 1998).

A general increase in the occurrence of AMTL has been noted in the shift from hunter-gatherer subsistence to the advent of agriculture (Hillson, 1998; Waldron, 2009). This condition has also been shown to increase with age and in some modern studies it has been more commonly observed in women than in men (Waldron, 2009). One of the major causes of AMTL is periodontal disease brought on by dental calculus build-up (Waldron, 2009; Roberts and Manchester, 2010). Dental calculus accumulates in the crevices between the tooth and the soft tissue and the alveolar bone, creating periodontal pockets (Roberst and Manchester, 2010). Periodontal disease commences with inflammation of the soft tissues of the jaw (referred to as gingivitis) and may become transmitted to the alveolar bone (referred to as periodontitis). Periodontal disease results in the resorption of the alveolar bone and the eventual loss of the periodontal ligament, responsible for holding teeth in place (Roberts and Manchester, 2010). The distances between the alveolar bone and the cemento-enamel junction increase and eventually tooth loss occurs (Roberts and Manchester, 2010). Periodontal disease may alternatively also be associated with scurvy as a result of the haemorrhaging of gums, which often leads to AMTL (Brickley and Ives, 2008; Roberts and Manchester, 2010).

2.2.3.3. Dental calculus

Dental calculus is mineralized plaque that accumulates at the base of a living plaque deposit, and is attached to the surface of the tooth (Hillson, 1998; Waldron, 2009). Dental plaque consists of micro-organisms which accumulate in the mouth, embedded in a matrix

partly composed by the organisms themselves and partly derived from proteins in the saliva (Roberts and Manchester, 1995). This process is sped up when diets containing large amounts of protein and/or carbohydrates are consumed (Roberts and Manchester, 2010). A high intake of protein and/or carbohydrate creates an alkaline oral environment which in turn makes mineralization of dental plaque possible. There are two types of dental calculus that can be observed namely supragingival calculus which accumulates above the gums and subgingival calculus which accumulates below the gums (Roberts and Manchester, 1995; 2010). The supragingival calculus is the more common of the two. Dental calculus develops more commonly on the teeth nearest the salivary glands, in other words on the tongue (lingual) side of the lower incisors and the cheek (buccal) side of the upper molars (Roberts and Manchester, 1995; 2010).

It has been argued that an inverse relationship exists between dental caries and calculus build up (Hillson, 1998). Whereas caries is the result of the net demineralization of tooth enamel as a result of an acidic oral environment, calculus formation is essentially a mineralization process favoured by an alkaline oral environment (Duckworth and Huntington, 2005). Tabacco smoking has been seen to enhance supragingival and subgingival calculus deposition (Van Reenen, 1954; Bergström, 2005). This apparent association was found to be independent of oral disease and oral hygiene conditions which suggest a direct dose-response relationship (Bergström, 2005).

Dental calculus may also be used to infer aspects of socio-economic status. This may be seen in cases where individuals from poorer communities do not have access to dental health care and therefore will not necessarily practice good oral hygiene (Waldron, 2009). Poorer communities may also be more prone to consuming cheaper cereal based staple diets which will have been shown to increase the occurrence of dental calculus (Larsen, 1997; Oliveira *et al.*, 2008).

2.2.3.4. Periodontal disease

Another dental disease which is closely associated with dental calculus is periodontal disease. Calculus accumulates in the crevices between the tooth and soft tissue and bone of the jaw, forming periodontal pockets that are a major influencing factor in the development of periodontal disease (Roberts and Manchester, 1995; 2010). This can cause inflammation of the gums (gingivitis or periodontitis), which in repeat occurrence can cause alveolar bone loss or the retraction of the alveolar bone from the tooth crown (Hillson, 1998; Waldron, 2009).

Periodontal disease is one of the most common dental diseases found in archaeological human skeletal remains, as well as present day human skeletal remains, and acts as a major cause of tooth loss (Roberts and Manchester, 1995; 2010; Waldron, 2009). Periodontal disease may also be associated with scurvy as a result of haemorrhaging and can result in severe tooth loss (Brickley and Ives, 2008).

2.2.3.5. Enamel hypoplasia

Enamel hypoplastic defects can also indicate factors of health and diet in a person's life, especially in the growing years when bone and teeth are still developing (Goodman and Rose, 1991; Roberts and Manchester, 1995). In bioarchaeology and biological anthropology, dental enamel defects have attracted the attention of many researchers (Roberts and Manchester, 1995). These enamel defects are also referred to as "indicators of stress". One of the most common defects is that of enamel hypoplasia (Roberts and Manchester, 1995). Like the production of skeletal tissue, the formation of enamel on growing teeth is a regular process that is subject to factors that may either slow or stop it (Larsen, 1997). Tooth enamel is especially sensitive to metabolic insults arising from nutritional deficiencies or disease, and because enamel does not remodel, it therefore provides permanent visual indicators for stress (Goodman and Rose, 1991; Larsen, 1997). Almost any environmental factor leading to metabolic disturbance will result in visible changes in the structure of enamel (Larsen, 1997). These changes can be observed as horizontal lines, pits or grooves on the enamel surface (Goodman and Rose, 1991; Roberts and Manchester, 1995). These defects can occur only while the teeth are developing, and remain as a permanent record into adulthood and beyond the grave (Roberts and Manchester, 1995; Hillson, 1998). Many factors play a role in the appearance of enamel hypoplasia but they can broadly be categorized as either due to nutritional deficiencies, or childhood illnesses (Roberts and Manchester, 1995).

2.2.4. Discussion

Some of the metabolic bone diseases, like scurvy and anaemia, may present with a higher mortality rate as well as a quick recovery rate which minimize the chances of skeletal manifestations and the eventual recognition of such diseases in past populations. Even where osteological manifestations of the disease can be observed, the analogous responses may hamper the definite diagnosis of the disease. The osteological manifestations of metabolic bone diseases can only present in a very limited number of ways and include either bone

resorption, bone depositioning, or a combination of both these processes. Furthermore, overlapping responses of metabolic diseases can become problematic in differentiating between specific metabolic diseases. Some metabolic diseases can coexist and therefore osteological responses of more than one disease may be presented within one individual. For this reason the collective assessment of all prevailing pathology observed in individual skeletons should be considered in order to better interpret the overall pathological pattern and the possible diagnosis of specific metabolic bone diseases and nutritional deficiencies.

2.3. Trauma and occupation

Trauma may be defined as injury to living tissue as a result of an external mechanical force or mechanism (Lovell, 1997). Fractures are the most frequent form of trauma observed in humans and occur as a result of forces such as tension, compression, flexion, and shearing on bones (Lovell, 1997; Berryman and Symes, 1998; Ortner, 2003). These forces lead to a break, either partial or complete, in the continuity of a bone (Lovell, 1997). Fracture timing is described as being either ante-, peri- or post-mortem. Ante-mortem fractures present fractures that have had time to heal (partially or completely) prior to death, whereas peri-mortem fractures refers to fractures obtained prior to or around the time of death (when bone is still fresh enough to react as live bone) and therefore does not present with healing. Post-mortem refers to the period well after death when bone no longer reacts as live tissue. These changes are associated with taphonomic alterations during the depositional phase.

Continued stress in the same area as well as additional underlying medical conditions may also result in trauma (Lovell, 1997). This is typically seen in stress fractures like spondylolysis, Schmorl's nodes and secondary fracturing as a result of underlying conditions like osteoporosis (Waldron, 2009).

Trauma can be influenced by intrinsic factors such as the person's age and sex as well as his/her general health (Ortner, 2003). The same is true for extrinsic factors associated with cultural practices, socio-economic status, and occupation. For this reason fracture patterns and frequencies can be useful in making inferences about health as well as the environment people lived and worked in. This is especially true for the 20th century mining industry where unskilled migrant labourers were often subjected to harsh working and living conditions. Compound settings often gave rise to interpersonal violence as tension rose in the confined spaces that compounds created whereas the hazardous working conditions lead to many work-related accidents and deaths (Richardson, 1982).

2.4. Comparative populations

It is difficult to compare the prevalence of disease and trauma with those of other groups reported on in the literature, as the Chinese miners' remains reflect very special circumstances and are not representative of a normal living population. The most appropriate comparative material is probably that from Gladstone, associated with diamond mining around Kimberley, South Africa (Van der Merwe *et al.*, 2010a-d), and Koffiefontein, also associated with diamond mining in the Free State, South Africa (L'Abbè *et al.*, 2003).

2.4.1. Gladstone, Kimberley

The remains of 107 individuals were recovered during rescue excavation at the Gladstone cemetery in Kimberley. These individuals were mostly migrant workers who came to Kimberley to work on the diamond mines and were thought to have died somewhere between 1897 and 1900. The sample comprised of 86 males, 15 females and six individuals of unknown sex. The majority of the individuals (71%) were between 19 and 45 years at the time of their death (Van der Merwe *et al.*, 2010a).

A high frequency of trauma was observed in this population with 27% of the individuals presenting with well-healed, healing or peri-mortem trauma. Skull fractures were most often observed and were identified in 49% of the individuals possibly suggestive a high occurrence of interpersonal violence (Van der Merwe *et al.*, 2010a). Long bone fractures (27%) and spondylolysis (7%) attested to the hazardous working conditions associated with an open-cast mine. Furthermore, 16 individuals presented with skeletal pathology associated with healed adult scurvy (Van der Merwe *et al.*, 2010b&c). These lesions included bilateral ossified haematomas, osteoperiostitic bone changes and periodontal disease (Van der Merwe *et al.*, 2010 b-d). Dental pathology was also recorded and included dental caries (57% males; 46.2% females); periodontal granulomata (17.7% males; 15.4% females), periodontal disease (39.5% males; 53.8% females), ante-mortem tooth loss (30.4% males; 23.1% females), and enamel hypoplasia was noted in 14 individuals (15.2%) (Van der Merwe *et al.*, 2010d).

2.4.2. Koffiefontein, Kimberley

The remains of 36 individuals were recovered during rescue excavations at the Koffiefontein Mine dump, Kimberley, South Africa. These individuals were mine workers

who died during a typhoid epidemic in 1896 (L'Abbè *et al.*, 2003). These individuals were also thought to be migrant workers performing unskilled labour on the mines.

The skeletal remains were assessed for any signs of trauma or disease. No signs of violent trauma could be observed, however stress fractures such as patellar avulsion were present (5.5%). A large proportion of the individuals presented with degenerative changes to the spine. These included vertebral osteophytosis (22.2%), intervertebral osteochondrosis (19.4%), and Schmorl's nodes (13.9%) (L'Abbè *et al.*, 2003). This reflected the hard physical labour associated with mining. Non-specific indicators of disease were also noted in the form of cribra orbitalia (8.3%), periostitis (11.1%) and enamel hypoplasia (61.1%) which suggests some type of 'systemic metabolic stress' during development, such as malnutrition, infectious disease, and/or weaning (L'Abbè *et al.*, 2003).

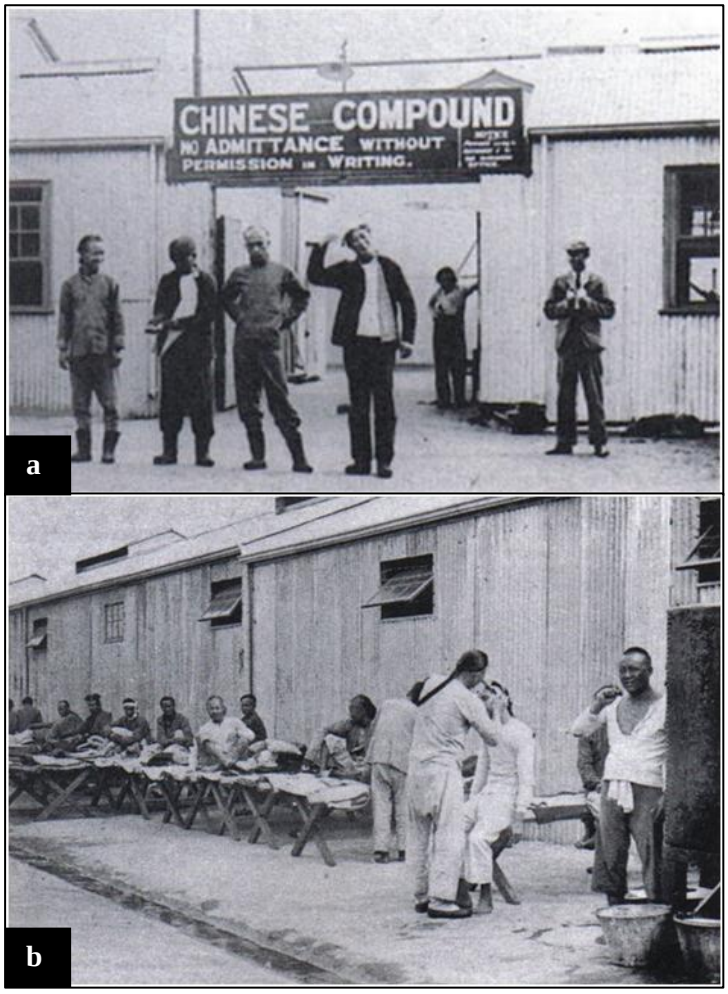
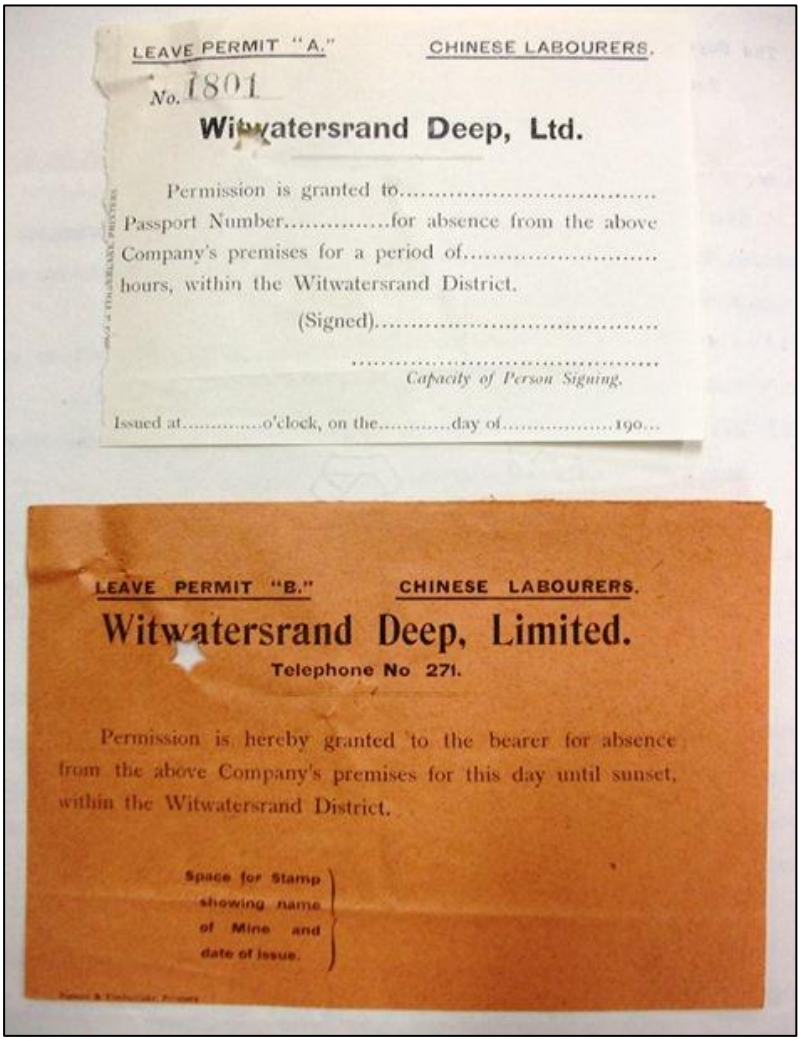


Figure 2. 1: Chinese miners in the compounds (Yap and Man, 1996)

Figure 2. 2: Chinese cooking house (Smith, 2006)



LEAVE PERMIT "A." CHINESE LABOURERS.

No. 1801

Witwatersrand Deep, Ltd.

Permission is granted to.....
Passport Number.....for absence from the above Company's premises for a period of..... hours, within the Witwatersrand District.

(Signed).....

.....
Capacity of Person Signing.

Issued at.....o'clock, on the.....day of.....190...

LEAVE PERMIT "B." CHINESE LABOURERS.

Witwatersrand Deep, Limited.

Telephone No 271.

Permission is hereby granted to the bearer for absence from the above Company's premises for this day until sunset, within the Witwatersrand District.

Space for Stamp showing name of Mine and date of issue.

Figure 2. 3: Leave permits for Chinese miners (FLD 214, 55/7)

Figure 2. 4: Chinese miners working underground (Smith, 2006)

Figure 2. 5: Chinese exhuming and cremating the remains of their fellows just before repatriation (Yap and Man, 1996)

CHAPTER 3: MATERIALS AND METHODS

3.1. Materials

For purposes of this study, the skeletal remains of 36 Chinese labourers housed in the Raymond Dart Collection, University of the Witwatersrand, have been studied. The background of these individuals was already discussed in the literature review. Standard physical anthropological analyses of the remains were conducted along with an in-depth recording of all pathology. Specific attention was given to the identification and diagnosis of any metabolic-related diseases. For the purpose of comparison, a sample of 100 individuals from the Raymond A Dart Collection (Dayal *et al.*, 2009), housed at the University of the Witwatersrand, as well as the Pretoria Bone Collection (L'Abbe *et al.*, 2005), housed at the University of Pretoria, for whom the cause of death were known and related to malnutrition, specific nutritional deficiencies or alcoholism, were assessed for pathological lesions.

The Raymond A Dart Collection comprises one of the largest documented cadaver-derived human skeletal assemblages in the world (Dayal *et al.*, 2009). Currently it houses over 2605 skeletons representing individuals from regional South Africa; including Africans (76%), Whites (15%), Coloureds (4%) and Indian populations (0.3%) (Dayal *et al.*, 2009). A very large proportion of the individuals in the collection are male (71%) (Dayal *et al.*, 2009). The recorded ages at the time of death range between new born to over 100 years, however, the majority of the individuals in the collection died between the ages of 20 and 70 years (Dayal *et al.*, 2009). Most of the skeletal remains are of cadaver-origin collected under the provision of South Africa's Human Tissues Act (No. 65 of 1983) and previous acts like the Anatomy Act (No. 20 of 1959). Acquisitioning already started in the early 1920s and is still continued today. Most of the individuals acquired for anatomical specimens and cadavers are unclaimed at their passing and in accordance with the Human Tissues Act No. 65 of 1983 their bodies become donated (L'Abbe *et al.*, 2005; Dayal *et al.*, 2009). The Dart Collection also houses a number of archaeologically derived skeletons which include the Chinese skeletons considered here (Dayal *et al.*, 2009).

The Pretoria Bone Collection is similar to the Raymond A Dart Collection and also comprises of cadaver-derived human skeletal remains. These remains have been acquired from 1942 with the inception of the Department of Anatomy and the Medical School at the University of Pretoria (L'Abbe *et al.*, 2005). The collection comprises of over 290 complete

skeletons, over 704 skulls and more than 541 complete postcranials. The skeletal material originates from unclaimed bodies (the majority) as well as donations (L'Abbe *et al.*, 2005). The majority of the collection comprises of black males (58.3%), with 17, 4% white males, 14. 2% black females and 7.6% white females (L'Abbe *et al.*, 2005). Black South Africans are represented in all age categories with a mean age of 52.9 (males 53.9 and females 47.4), whereas the few white individuals are mainly represented in the over 50 years age category with a mean age of 68.2 years (males 66.5 and females 70.6) (L'Abbe *et al.*, 2005).

For both collections specific information about the deceased is documented. This includes the sex, ancestry, age at death, approximate height and weight at the time of death, and cause of death as well as more personal information like the deceased's name, last known address and hospital where they passed away (L'Abbe *et al.*, 2005). This information is kept by the tissue bank associated with each institution. For research purposes personal information about the deceased, such as the deceased name and last living address, are not provided. Researchers are only provided with the demographic information and cause of death. For this reason all skeletal material sampled for research are of unknown identity.

For this study individuals from both collections were sampled. The sampling process was solely based on the cause of death and age, sex and ancestry were ignored. All individuals for whom the cause of death was given as 'nutritional deficiency', 'malnutrition', 'neglect' or as a specific nutritional or metabolic disorder for example 'scurvy', 'pellagra', 'beri-beri' and 'anaemia' were selected. Additionally, individuals noted to have died of 'alcoholism' and 'liver cirrhosis' were also selected. This reasoning stemmed from the fact that most alcoholics have severely nutrient deficient diets and would therefore possibly provide evidence of metabolic diseases (Truswell, 2002). It is also known that beriberi may be acquired as a result of chronic alcoholism resulting in Wernicke-Korsakoff's encephalopathy (Carpenter, 2000; Thomson and Marshall, 2006; Bates, 2007). One of the most common ailments and also a common cause of death documented amongst the Chinese indentured labourers was beriberi. For this reason possible comparable cadaver samples were selected to see whether it might be possible to identify and distinguish beriberi from other metabolic related diseases. Alcoholism-derived beriberi may further hamper mineral and vitamin absorption and secretion which may lead to additional vitamin deficiencies other than that of thiamine associated with beriberi (Ball, 1998; Smith, 1998; Gibney *et al.*, 2002; Truswell and Milne, 2002; Thomson and Marshall, 2006; Doung-ngern *et al.*, 2007). In cases where liver cirrhosis was not attributed to alcoholism it might still be possible to observe the

manifestation of metabolic bone diseases as the bioavailability of certain vitamins like vitamin D and calcium are severely influenced by liver function (Arteh *et al.*, 2010)

It should however be mentioned that the cause of death given for individuals may not always reflect the complete medical history of the person at hand. In some cases individuals may have suffered from multiple underlying conditions prior to death. These conditions would not necessarily have contributed to the person's death and would therefore not be noted once cause of death is given. In some cases it may also be possible that such underlying conditions would have contributed to bone pathology. This may influence the interpretation of bone pathology in cases where diagnoses are based on the cause of death. For this study the cause of death was used in order to create a known sample to which the unknown Chinese skeletal pathology could be compared to. Care was taken not to include pathology which clearly indicated other underlying conditions such as treponemal disease. The lack of more collective medical histories does however mean that less distinguishable pathology, suggestive of additional underlying conditions other than that given as the cause of death, may become included in the study.

The selected cadaver remains (Table 3.1) were assessed for any pathological lesions that may be related to metabolic bone diseases. Those cadaver skeletal remains known to have died as a result of alcoholism were assessed for any distinguishing pathology that may suggest the skeletal manifestation of beriberi and other possible related vitamin deficiencies like vitamin D and calcium deficiency. This provided pathological patterns which enabled a better interpretation of the pathology observed in the Chinese skeletal remains. This data also provided additional insight into the distribution and incidence rate of skeletal pathology associated with specific metabolic bone diseases.

Table 3. 1: Cadaver skeletal remains sampled from the Raymond A. Dart and Pretoria Bone Collection

*In some cases multiple causes of death were given and therefore the same individual may have been placed in more than one category

	RA Dart Collection		UP Bone Collection		TOTAL	
	Male	**Female**	**Male**	**Female**	**Male**	**Female**
Mean age at death	50.1	40.9	54.7	47.4	52.2	42.7
Number in Sample	50	24	18	8	68	32
Stated c.o.d. Nutritional Disease*	36	22	8	2	44	24
Stated c.o.d. Alcoholism*	17	5	11	6	28	11

3.2. Methods

3.2.1. Physical anthropological analysis

The analysis of the 36 Chinese skeletons entailed the standard physical anthropological analysis with specific attention given to the diagnosis and interpretation of pathological lesions. Here the "*Standards for data collection from human skeletal remains"* by Buikstra and Ubelaker (1994) was used as a basis for the analytical procedure. Standardised osteometric measurements and calculations, coupled with morphological characteristics were used to determine the demographics of each individual, where possible. The use of a wider array of skeletal characteristics and comparable measurements helped to improve estimate accuracies and precision, especially when dealing with archaeological remains which often present with incomplete and poorly preserved skeletal elements (Milner and Boldsen, 2012).

3.2.1.1. Sex and ancestry

The focus on estimation of sex and ancestry was limited since it was already known from historical and burial records that this sample almost certainly represents male Chinese indentured labourers (Yap and Man, 1996; Gibbon *et al.*, 2010). Therefore, estimations of sex and ancestry were done only to confirm the historical identification.

Sex of adult remains is based on either visual assessment of morphological characteristics for example those observed in the cranium and pelvis, or measurements of one or more bones, for example single long bone measurements which distinguish between

variation in size (Krogman and Íşcan, 1986; Loth and Íşcan, 2000a; Milner and Boldsen, 2012). Due to the presence of sexual dimorphism amongst *Homo sapiens*, males tend to be more robust with regard to muscle attachments and general size than females, enabling these characteristics to be used as a discerning factor in the determination of sex in the human skeleton (Ubelaker, 1989). It is however important to note that both morphological and metric differences between males and females follow a continuum from one extreme to another and that overlap should therefore always be considered (Milner and Boldsen, 2012). For this reason it is advisable to use more than one technique. Discriminant function analyses were therefore also used where possible. For this study population-specific anthropometric data of Chinese were used for comparison and included measurements obtained from the femur (Íşcan and Shihai, 1995) and humerus (Íşcan *et al*., 1998). Results obtained using the above-mentioned techniques were also compared to the results obtained from a study by Gibbon *et al.* (2010) reporting on the results of molecular sex identification through the amelogenin gene in the same Chinese miners' sample.

The determination of ancestry was not done here as it is already known that these individuals were of Chinese origin. The identification of ancestry was, therefore, not part of the objectives of the study.

3.2.1.2. Age

Age at death was estimated by the degree of epiphyseal closure (Krogman and Íşcan, 1986; Scheuer and Black, 2004; Schaefer *et al.*, 2009) and tooth development (Ubelaker, 1989; Loth and İşcan, 2000b; Scheuer and Black, 2004) for possible adolescent individuals. For adult individuals, age was estimated using the following methods: changes in the sternal ends of ribs (Loth and Íşcan, 2000b), changes in the face of the pubic symphysis of the pelvis (Brooks and Suchey, 1990), status of cranial suture closure (Acsádi and Nemeskéri, 1970; Krogman and Íşcan 1986), and changes in the auricular surface of the ilium (Lovejoy *et al*., 1985).

Due to the lack of population-specific data for the assessment of age in this population, age estimates were assigned in a wider than usual range. Age estimations were also given as suggested by Falys and Lewis (2011) to ensure that the data provided would be comparable with other population groups. The archaeological nature of this sample meant that most of the remains were in a poor state of preservation and were mostly incomplete.

Consequently the following age at death categories were assigned: adolescent (younger than 20 years), young adult (20-34 years), middle adult (35-45 years), and old adult (46+ years).

3.2.1.3. Stature

Adult stature was determined using long bone measurements where possible, based on the simple and multiple regression equations developed by Xiang-Qing (1989). This study was based on single and combined limb element measurements obtained from 472 Chinese males from North and South China (Xiang-Qing, 1989). 71 of these individuals came from the Shandong Province. Historical accounts suggested that the majority of the Chinese indentured labourers employed on the Witwatersrand mines actually originated from the Shandong Province. Measurements were obtained from males between the ages of 21 and 80 years and were taken before and after death (cadaver and macerated dried bone) (Xiang-Qing, 1989). Regression formulae are therefore provided in age categories for the youngest, middle and oldest age ranges (21-30; 41-50; 61-80). These age categories were used in this study where possible. If an age range could not be determined and age was only assigned as being adult the 21-30 age category was used to determine the stature. This age category was used since historical accounts suggested that most of the Chinese who were recruited and employed were young adults.

3.2.2. Assessment of pathology

An assessment of all pathology was done for the 36 Chinese skeletons whereas for the 100 cadaver skeletons, selected from the Pretoria Bone Collection and the Raymond A. Dart Collection, only pathology associated with metabolic bone and nutritional diseases was assessed. This enabled the researcher to compare results obtained from a ‘known’ sample (represented by the cadaver skeletal remains) to that of an ‘unknown’ sample (represented by the Chinese skeletal remains).

Each skeleton was visually assessed for any macroscopic pathological alterations. Where possible, diagnoses were made by comparing the observed bony characteristics and their distribution with standard palaeopathological textbooks (Aufderheide and Rodríguez-Martin, 1998; Ortner, 2003, Brickley and Ives, 2008; Waldron, 2009) and photographs. In cases where pathological lesions were less distinct or poor preservation hampered the macroscopic observation of possible pathology, X-rays were taken to help distinguish between pathological lesions and natural taphonomic changes. The location and frequency of

lesions were noted and possible aetiologies discussed. Various sources were used to aid in the identification and interpretation of such pathological lesions. They included the following; Barnes (1994), Larsen (1997), Aufderheide and Rodríguez-Martin (1998), Ortner (2003) and Brickley and Ives (2008), Waldron (2009).

The incidence of pathological lesions observed in the Chinese and cadaver skeletal remains were determined in relation to the number of individuals in each sample population as well as the number of skeletal elements investigated. Chi-squared analyses were used to determine whether statistical significant differences exist in the presence and frequency of pathological lesions observed in the Chinese skeletal remains with that in the cadaver skeletal material. Chi-square analyses were used here since it is one of the most common methods for comparing frequencies or proportions in two or more groups to determine whether there is a difference in proportions or independence between two factors (Dawson-Saunders and Trapp, 1994). Small sample sizes will however inflate the χ^2 value and effectively alter the P value. For expected values smaller than 5 Fisher's exact test was used to calculate the P value.

Since historical accounts indicated that the Chinese labourers came from areas struck by famine and since it is known that so many Chinese labourers died as a result of beriberi, higher frequencies of skeletal pathology associated with metabolic diseases were expected to be prevalent in this community. It was also expected that the frequency of specific pathological lesions associated with metabolic diseases might represent a pattern which may distinguish the presence of beriberi from other nutritional deficiencies.

3.2.2.1. Developmental defects

Developmental defects, especially those affecting the axial skeleton, were looked at as a possible indicator of development as well as early childhood health and nutrition. Even though many of the developmental defects occurring in the skeleton stem from a congenital predisposition (Ortner, 2003) it has been argued that some developmental defects such as spina bifida occulta and notochord defects may be representative of a poor maternal health during pregnancy and that this condition may be the result of vitamin deficiencies (vitamin B12, iron, magnesium, folic acid, and zinc; Groenen *et al.*, 2004a; Groenen *et al.*, 2004b). This would therefore provide information on the previous generation's health and if present may represent a long standing nutritional problem.

3.2.2.2. Metabolic and non-specific indicators of disease or stress

Conditions such as cribra orbitalia and porotic hyperostosis which are related to haemolytic anaemia (thalassemia, sickle cell anaemia, and malaria) or megaloblastic anaemia (chronic dietary deficiency, malabsorption, deficiencies of the vitamins B12 and B9, and gastrointestinal parasite infections) (Ortner, 2003; Stuart-Macadam, 2005; Mays, 2007; Walker *et al*., 2009; Oxenham and Cavill, 2010) were scored using techniques developed by Stuart-Macadam (1985) and Buikstra and Ubelaker (1994). Each lesion was scored according to its degree or severity and whether it was thought to be active or healed at the time of death. Subperiosteal bone growth or periostitis was also used as an indicator of health and diet. This condition may occur as a result of multiple afflictions including localised trauma (in most cases a unilateral response), infections, metabolic diseases and overall malnutrition (Ortner 2003; Waldron 2009; Weston 2012). Due to the macroscopic nature of this study subperiosteal lesions were only used as indicators of generalised bone disease associated with malnutrition and localised infection (Ortner, 2003; Weston, 2012), except in cases where a more definitive diagnosis could be made, for example in the case of scurvy where it may present in the form of ossified haematomas (Van der Merwe *et al.*, 2010b). Furthermore, the presence of any skeletal deformities, secondary fractures, and/or osteopenia was discussed as possible indicators of various metabolic related diseases.

Harris lines, which have always been interpreted as non-specific signs of stress in children (Harris, 1933; Larsen, 1997; Aufderheide and Rodríguez-Martin, 1998; Ortner, 2003) have not been looked at here. It has recently been argued that Harris lines may be a factor of normal growth rather than an indicator of nonspecific stress (Papageorgopoulou *et al.*, 2011) and for that reason it was not used in this study.

Dental pathology can also be used as a long term indicator of health, for instance in the form of enamel hypoplasia which represents periods of malnutrition and/or overall periods of illness during an individual's childhood. Enamel hypoplasia is an enamel defect that occurs while tooth enamel is deposited during development (Hillson 1998) Enamel does not remodel, causing permanent visual indicators for stress which may be retained throughout adulthood (Roberts and Manchester, 1995; Larsen, 1997; Ortner, 2003 Lukacs, 2012). For this reason enamel hypoplasia was recorded for both the Chinese and cadaver skeletal remains.

Enamel hypoplastic defects were recorded according to tooth type and whether it presented as linear or pitting defects. Enamel hypoplasia frequencies were calculated by using the individual count method created by Lukacs (1989) and Roberts and Manchester

(1995). This was done by dividing the total number of individuals presenting with the defect by the total number of individuals examined for the condition. Enamel hypoplasia intensity per tooth type was also determined. This was done by dividing the total number of a specific tooth type presenting with the defect with the total number of that specific tooth type examined. This provided frequencies for the defect for each tooth type.

3.2.2.3. Dental pathology

Along with skeletal pathology dental pathology was also included in this study and for this Roberts and Manchester (1995), Hillson (1998), Ortner (2003) and Lukacs (2012) were used. Dental pathology such as dental caries and dental calculus can be indicative of a person's diet and may provide additional information on any nutritional deficiencies suffered by a specific person (Hillson, 1998; Ortner, 2003). This is especially prevalent in individuals consuming large amounts of carbohydrates and may therefore be present in populations where the staple diet consists of cereals, such as rice or maize (Roberts and Manchester, 1995; Hillson, 1998).

The frequency of dental caries was calculated according to the methods set out by Lukacs (1989) and Roberts and Manchester (1995; 2010). Carious lesions were only recorded when a distinct cavity could be observed. This ensured that taphonomic alterations mimicking dental pathology were not scored erroneously. Carious lesions were recorded according to the tooth type and position (crown or root caries).

For dental caries the individual count method was used (Lukacs, 1989). This method determines the frequency of the lesion within the sample by dividing the frequency of individuals presenting with dental caries by the total number of individuals in the sample. This method however assumes that all the teeth were present for investigation, or that those teeth that were lost, either ante-mortem or post-mortem, did not have carious lesions. Due to the margin of error in this method the tooth count method (Roberts and Manchester, 1995) was also used to determine the caries intensity. This was done by dividing the total number of carious teeth with the total number of individuals presenting with caries. By calculating this the average number of carious lesions per mouth can be determined. Lastly, caries intensity was also calculated per tooth type. This was done by dividing the total number of a specific tooth type by the total number of that specific tooth type investigated.

Ante-mortem tooth loss (AMTL) frequency was also calculated using the individual count method (Lucaks, 1989). This was done by dividing the total number of individuals

presenting with AMTL with the total number of individuals (where the maxilla and mandible were preserved) in the sample. The AMTL per mouth in turn was calculated by dividing the total number of teeth lost ante-mortem with the total number of individuals in the sample. Lastly, AMTL intensity (tooth count) was calculated by dividing the total number of teeth lost ante-mortem by the total number of tooth sockets present in the sample.

3.2.2.4. Degenerative changes

Vertebral osteophytosis and peripheral osteoarthritis were recorded as possible indicators of activity or mechanical function and lifestyle (Larsen 1997; Jurmain *et al*. 2012). Some of the possible activity patterns associated with vertebral osteophytosis include physical activities that place strain on the spine, such as lifting or carrying heavy loads, whereas peripheral osteoarthritis of the calcaneo-talar joint may be the result of constant squatting or physical strain to this area (Larsen 1997). Jurmain *et al*. (2012), however, cautions the use of osteoarthritic changes in recreating activity and lifestyle patterns as there is to date no support to enable the simplistic assumption that these changes directly derive from habitual activity. Nevertheless, when such changes are observed in relatively young individuals it can suggest hard physical labour from a young age which may act as an indicator of socio-economic and socio-cultural status as well as occupation.

3.2.2.5. Infectious diseases

Specific infectious diseases such as treponematosis and tuberculosis were also noted where present. These conditions are often observed in mining populations where confined working and living conditions give rise to a higher infection rate. This is especially true for the 20th century Witwatersrand mines as compounds and barracks were generally overcrowded. Conditions like tuberculosis were also worsened by the high levels of dust and poor ventilation in mining shafts which caused silicosis in many of the workers (Roberts and Buikstra, 2003).

Additionally, the presence of osteomyelitis was also noted where present. Osteomyelitis refers to any form of infection of bone and bone marrow which results in the inflammatory destruction of bone (Ortner, 2003; Waldron, 2009). Infection of bone can result from three sources namely haematogenous spread, direct spread from an overlying or adjacent organ, or by direct implantation by penetrating injuries (Waldron, 2009). The latter

is especially significant as this is often seen in compound fractures and therefore has direct bearing on mining related accidents.

3.2.2.6. Trauma

Trauma may be defined as injury to living tissue as a result of an external mechanical force or mechanism (Lovell, 1997). Such forces include tension, compression, flexion, and shearing (Lovell, 1997; Berryman and Symes, 1998; Ortner, 2003). Continued stress in the same area as well as additional underlying medical conditions may also result in trauma (Lovell, 1997). Fracture timing is described as being either ante-mortem, peri-mortem or post-mortem. Ante-mortem fractures present fractures that have had time to heal (partially or completely) prior to death, whereas peri-mortem fractures refers to fractures obtained prior to or around the time of death (when bone is still fresh enough to react as live bone) and therefore does not present with healing. Post-mortem refers to the period well after death when bone no longer reacts as live tissue. These changes are associated with taphonomic alterations during the depositional phase.

For ante-mortem and peri-mortem trauma, criteria from Lovell (1997), Berryman and Symes (1998), Galloway and Zephro (2005), and Porta (2005) were used. Trauma was discussed as being ante- or post-mortem based on the degree of healing present. Peri-mortem fractures were classified according to the type of fracture (oblique, transverse, butterfly, spiral, and/or compression) as well as whether the fracture patterns were partial or complete.

3.3. Ethical considerations

When dealing with human remains some ethical considerations should be regarded. Research conducted on the Chinese indentured labourer skeletal sample as well as the cadaver bone samples followed legal and ethical directives to ensure the ethical handling of human remains throughout the study.

Skeletal data may be subject to biases and interpretive problems, especially when it comes to individual identities and views. Skeletal remains, nevertheless, encode information on genetic relationships and physiological processes related to growth, development and disease, which are a unique record of life and death; provided our interpretations are sensitive and contextual. Physical anthropological perspectives on the scientific value of human skeletal remains can cause offence and therefore practitioners should be sensitive towards the ethical issues governing the handling and studying of human remains. According to Larsen

and Walker (2005: 168) there are three such ethical principles that most people agree upon. First, human remains should be treated with dignity and respect. Secondly, descendants should have the authority to control the deposition of their relatives' remains. Thirdly, human remains need to be preserved when possible so that they are available for scientific research. Nevertheless, repatriation of human skeletal remains should always be a first priority, to ensure that scientists are responsive to descendants' wishes. This dissertation is no exception, and takes the respectful treatment of the Chinese human remains as well as the cadaver skeletal remains as a prime directive. All data obtained from this study was used purely for academic reasons and for the purpose of better understanding the pathological manifestations of metabolic bone diseases and malnutrition in the human skeleton.

The skeletal remains studied in this project are part of University collections and identities for these individuals are not known, therefore, permission from family members cannot be obtained. Nevertheless, data was used in a sensitive manner to ensure the ethical representation of these people and their remains.

Permission was gained from the University of Pretoria as well as the University of the Witwatersrand for the use of their bone collections. Awareness was given to the Human Tissue Act No. 65 of 1983 regarding the ethical handling of human remains, as well as the University of Pretoria's research ethics.

CHAPTER 4: RESULTS

In this chapter, results of skeletal analyses pertaining to the Chinese miners will be given, as well as a summary of pathological condition observed in a modern, comparative sample. Comparisons of these findings with other populations will be shown in the Discussion.

4.1. Demographic characteristics of the Chinese miners

Individual skeletal reports discussing the age, sex, stature, dentition, trauma and pathology are provided in Appendix 1. The preservation, cranial, postcranial and dental measurements are also noted in Appendix 1, Tables 1-4. For a summary of the results of the skeletal analyses, refer to Table 4.1.

4.1.1. Age distribution

Age at death assessment revealed that all 36 individuals had died between the ages of 17 and 45 years. The majority of individuals (58%) were of young adult age (n =21; 20-34 years). Two individuals were found to be of adolescent age (younger than 20 years) and nine of middle adult age (35-45 years). The remaining four were only scored as adults due to their poor preservation.

4.1.2. Sex distribution

Of the 36 skeletons analysed, a total of 24 individuals were assessed to be male using both morphological characteristics and metric analyses. Thirteen of these 24 individuals were also determined to be male by using DNA analysis (Gibbon *et al.*, 2010). For the remaining 12, sex could only be estimated by the morphological features of the skull and pelvis. These individuals presented with characteristics associated with masculine development and were therefore tentatively assigned to be male. The likelihood of any of these individuals being female is very slim since it is known from the historical records that a total of only six women were imported during the period of recruitment (Harris, 1998).

4.1.3. Stature

Ante-mortem stature was calculated for 30 of the 36 individuals. Due to the poor preservation of long bones in six of the individuals their stature could not be determined. Stature ranged between 152.9 cm and 170.8 cm with a mean of 163.2 cm. Living mean statures obtained from Northern Chinese males dating to the late 19th to early 20th centuries ranged between 166.0 cm to 169.6 cm (Morgan, 2004). There seems to be an overlap in the mean stature obtained from the 36 Chinese skeletons and the living data, although the average height of 163 cm observed in the current sample seems to be on the shorter side and may indicate some stunting in the growth of these individuals.

4.2. Pathology

A summary of all pathological changes is shown in Tables 4.1 and 4.2.

4.2.1. Developmental defects

Changes associated with developmental defects were observed in the form of sacralisation of vertebra L5 in three individuals (A1001, A1008, A1011). One of these cases presented as complete sacralisation (A1001) (Fig. 4.1), whereas in the other two it was only partial (A1008 and A1011). Spina bifida occulta was observed in two individuals (A1002 and A1030) (Fig. 4.2). Notochord defects were observed in the lumbar and thoracic vertebrae presenting as minor sagittal clefting at the midline in two individuals (A1030 and A1031) (Fig. 4.3) and as a cleft neural arch of L5 in two individuals (A1001 and A1002) (Fig. 4.4).

4.2.2. Metabolic bone diseases and non-specific signs of disease

- **Osteomalacia/residual rickets**

Bowing of the lower extremities, possibly associated with osteomalacia and/or residual rickets, was observed in nine individuals (A998, A1001, A1005, A1006, A1010, A1011, A1022, A1025, A1027) (Fig. 4.5). Four presented with bowing of the tibia (A1001, A1005, A1011, A1027), six of the fibula (A1001, A1005, A1006, A1010, A1011, A1022), and two of the femur (A998 and A1025). In none of these cases was bowing observed in any of the upper limb bones, but as can be seen many of the individuals with bowing of the tibia had concomitant changes in the fibula.

- **Porotic hyperostosis**

A high prevalence (78.3%) of porotic hyperostosis was observed (Fig. 4.6a&b). Eighteen out of 23 individuals that could be examined presented with the condition (A996, A997, A998, A1000, A1001, A1003, A1005, A1006, A1009, A1011, A1014, A1015, A1016, A1018, A1021, A1022, A1023, A1030). The majority of these lesions were concentrated on the parietal and occipital bones especially in the region of the lambdoid suture and presented as degree score 1 to 2 (healing/healed). X-rays of the skulls in lateral aspect view confirmed the diagnosis with the typical hair-on-end appearance (Ortner, 2003) (Fig. 4.6c).

- **Cribra orbitalia**

This condition was observed in nine individuals (34.6% of the individuals with orbits) (A998, A999, A1001, A1002, A1003, A1005, A1009, A1022, A1029). These lesions mostly presented as degree score 1 to 2 (healed) (Fig. 4.7a). A1009, however, presented with possible active cribra orbitalia (Fig. 4.7b). In all of these cases bilateral involvement was observed except in cases where one of the orbits could not be observed due to poor preservation.

- **Periostitis / subperiosteal bone growth**

Localised superficial infection was observed on the femora of A996, A997, A1004, A1013, A1019, A1020, A1022, A1028, and A1031, the tibiae of A996, A998, A1000, A1001, A1003, A1004, A1010, A1011, A1013, A1016, A1019, A1028, and A1031 (femur: 27.3%; tibia: 38.2%) (Fig. 4.8).

- **Enamel hypoplasia**

A high prevalence (93.1%) of linear enamel hypoplasia was found (Fig. 4.9). This condition was present in 27 of the 29 individuals with observable teeth. These lesions were observed in all the tooth types and presented as multiple hypoplastic transverse defects. Table 4.3 provides frequencies for enamel hypoplasia per tooth type. From this it is clear that the majority of defects could be observed on the canines (70.9%).

4.2.3. Dental pathology

- **Caries**

Carious lesions with and without abscess formation were observed in 10 out of 29 individuals, representing a total of 34.5% of the population. An average of 2.8 carious lesions per mouth was observed with a dental caries intensity of 4.1% (Table 4.4). The majority of the lesions could be identified as coronal caries and were mainly observed in the molars and premolars. Tooth type frequencies indicated that the posterior teeth (premolars and molars) were the only observable teeth affected by caries (Table 4.5). The anterior teeth (incisors and canines) did not present with any carious lesions. Of the posterior teeth the second molars were most often affected by dental caries (12.3%) followed by the third molars (8.1%; Table 4.5).

- **Ante-mortem tooth loss**

Ante-mortem tooth loss was observed in six of the Chinese miners (A997, A1000, A1003, A1014, A1022, A1029) presenting 22.2% of the population (Table 4.6). Per mouth the AMTL frequency was 33.3% whereas the AMTL intensity per tooth was 1.1%.

- **Dental calculus and periodontal disease**

Of all the individuals that could be assessed, 60.7% presented with dental calculus. Dental calculus was, however, not severe and presented as thin bands mostly on the buccal surfaces of the molars and premolars and on the lingual surfaces of the anterior teeth. Periodontal disease was observed in nine individuals or in 32.1% of the cases.

4.2.4. Degenerative changes

Osteophytic lipping at the vertebral endplates (vertebral osteophytosis) were observed in three individuals (A1000, A1007, A1008) and marginal osteophytes at the edges of the major synovial joints such as the elbow, knee and ankle were observed in six individuals (A1000, A1007, A1008, A1012, A1024, A1026). Almost all of these individuals were estimated to be between the ages of 35 and 45 at the time of their deaths except for A1024 which was estimated to be between 20 and 34 years and A1026 which could only be classified as an adult.

4.2.5. Infectious diseases

Pathology possibly associated with infectious diseases was observed and included possible osteomyelitis observed in two individuals (A1022 – calcaneus and A1023 – distal femur) (7.2% of the individuals). One individual (A1008) also presented with destructive lesions of his cranial vault which might be due to a treponemal disease, possibly syphilis (caries sicca), or a localised infection as a result of a scalp injury (Fig. 4.10). No additional signs of disease associated with treponemal disease could be observed.

4.2.6. Trauma

- **Ante-mortem fractures**

Three individuals (10%) had ante-mortem fractures, all of which showed complete healing except for A1003 which presented with a partially healed fracture to the distal epiphysis. A1003 also presented with an ante-mortem healed fracture of the right fibula (midshaft) (Fig. 4.11), whereas another presented with an ante-mortem healed depressed fracture of the left parietal bone (A1030) (Fig. 4.12). The third individual (A1031) had a fused tarsal and first metatarsal which may be related to trauma, but may also be the result of infection in that area.

- **Spondylolysis and Schmorl's nodes**

These two conditions are listed here as traumatic conditions, as it is said to represent typical fatigue fracture of the spine (Merbs, 1989; 1996). However it should be kept in mind that both spondylolysis and Schmorl's nodes may have alternative causes, such as developmental defects (Ward and Latimer, 2005; Ward *et al.*, 2007; Williams *et al.*, 2007) or degenerative changes (Aufderheide and Rodríguez-Martin 1998; Ortner 2003; Brickley and Ives 2008; Waldron 2009). Spondylolysis of the lumbar vertebrae was found in two individuals (A1007 and A1023). A1007 presented with bilateral spondylolysis of L5 whereas A1023 presented with unilateral spondylolysis also of L5 (Fig. 4.13). Schmorl's nodes were identified in three individuals (A997, A1023, A1031; Fig. 4.14). A997 presented with a Schmorl's node on T11, A1023 on T8-T10 and A1031 on L1 to L4.

- **Peri-mortem fractures**

Thirty percent of the individuals presented with peri-mortem fractures of especially the long bones. Two individuals (A997 and A1015) presented with peri-mortem fractures of the humerus - A997 had a complete transverse fracture of the right humerus (midshaft) (Fig. 4.15), whereas A1015 had a spiral fracture of the right humeral shaft (distal third). One radial fracture (midshaft) was observed (A1004) and presented with a butterfly fracture. Five individuals presented with vertebral fractures (A997, A1003, A1011, A1013, A1030). Two of these (A997 and A1030) were possible wedge fractures of T12, presented by a collapse of the anterior part of the vertebral body, whereas the rest were compression or burst fractures in the spinious process of T2 (A1003), T4 (A1011), and L3-L5 (A1013), presented by fracture lines throughout the vertebral body and extending into the spinious processes (Fig. 4.16).

Six femoral fractures were observed (A997, A1004, A1009, A1011, A1015, A1023). Three of these fractures presented as spiral fractures (A977 – distal third of the shaft, A1009 - subtrochanteric, A1023 – proximal third of shaft) (Fig. 4.17), whereas the other three presented as butterfly fractures (A1004 - midshaft, A1011 - midshaft, A1015 – proximal third) (Fig. 4.18). One possible compression fracture of a right os coxa was observed (A1003) and presented with a medially compressed ilium causing fracturing of the iliac wing. Four individuals presented with fractures of the tibia (A1004 - midshaft, A1009 - midshaft, A1015 - midshaft, A1030 – distal third) and two of the fibula (A1004 - midshaft and A1009 - midshaft). All of these fractures presented as butterfly fractures except for the tibial fractures of A1009 and A1030 which presented as a spiral fractures and the fibular fracture of A1009 which also presented as a spiral fracture.

Eight out of the nine individuals presenting with peri-mortem fractures presented with more than one fracture throughout the body (A997, A1003, A1004, A1009, A1011, A1013, A1015, A1030). This represented 26.6% of the total number of individuals assessed for trauma.

4.3. Pathology associated with metabolic and non-specific bone disease as observed in cadaver skeletal remains

A summary of all pathology associated with metabolic and non-specific diseases, observed in the modern skeletal collections, can be found in Table 4.7. A total number of 100 cadaveric skeletal remains were analysed. Cause of death overlapped in some cases as multiple (nutritional related) causes for death were sometimes provided per individual. Cause

of death was correlated to specific pathological conditions normally associated with metabolic bone diseases and nutritional deficiencies.

4.3.1. Porotic hyperostosis

The largest percentage of porotic hyperostosis (83.9%) was observed in individuals for whom cause of death was given as liver cirrhosis or alcoholism. This was followed by 72.7% for pellagra, and 55.8% for those who died as a result of general malnutrition. For scurvy, beriberi and anaemia respectively the number of individuals inspected was only one. All three, however, presented with porotic hyperostosis. Most of the cases presented as degree score 2, healing/healed.

4.3.2. Cribra orbitalia

For those individuals who died as a result of pellagra 81.8% showed signs of cribra orbitalia, which was the highest prevalence of all conditions assessed. Most of these cases presented as degree score 1 to 2, active and healing/healed. The second largest percentage (57.7%) was observed in individuals who died as a result of general malnutrition followed by liver cirrhosis or alcoholism at 51.5%. The two individuals who respectively died from scurvy and anaemia both presented with cribra orbitalia whereas the one individual who succumbed from beriberi did not present with any such lesions.

4.3.3. Periostitis (tibia and other)

By far the largest percentage periostitis was observed in the individuals who died of liver cirrhosis/alcoholism. Of the individuals who died as a result of liver cirrhosis/alcoholism 91.4% presented with periostitis to the tibiae and 55.6% to the other long bones. In the individuals who died of pellagra 70% presented with periostitis the tibiae and 63.6% for other long bones, whereas 53.8% of tibiae and 44.2% of other long bones were affected by periostitis in individuals who died from general malnutrition. The one individual for whom cause of death was given as beriberi also presented with periostitis in the tibiae and other long bones. No periostitis was observed in the anaemia case and for the individual who died of scurvy no postcranial remains were available.

4.3.4. Medio-lateral bowing (tibia and fibula)

The most frequent medio-lateral bowing of the tibia and fibula could be observed for the pellagra individuals at 20%, followed by liver cirrhosis/alcoholism at 11.4% and general malnutrition at 3.8%. No medio-lateral bowing was observed for the beriberi, scurvy or anaemia case.

4.3.5. Developmental defects (vertebrae)

Developmental defects were observed in 22.2% of the individuals who died of liver cirrhosis/alcoholism and 18.2% of the individuals who died of pellagra. Of the general malnutrition cases 7.7% presented with this type of pathology. The one beriberi case presented with these defects whereas the anaemia case did not. The individual who died of scurvy could not be assessed as no postcranials were present.

4.3.6. Enamel hypoplasia

The highest prevalence (45.5%) of linear enamel hypoplasia was observed in the individuals who died of pellagra followed by general malnutrition (20.4%) and liver cirrhosis/alcoholism (16.7%). The individual who died of scurvy also presented with enamel hypoplasia whereas the beriberi and anaemia cases respectively did not present with any such lesions.

Table 4. 1: Summary of physical anthropological analysis of the 36 Chinese individuals
R = right side
L = left side
- could not be determined due to poor preservation

Individual	Age (years)	Sex	Stature (cm)	Skeletal pathology	Dental pathology	Trauma
A996	20-34	Male	163.91±3.91	-Porotic hyperostosis -Periostitis femur and tibia	-Enamel hypoplasia	None
A997	20-34	Male	166.0±3.91	-Porotic hyperostosis -Periostitis femur -Schmorl's node on T11	-Enamel hypoplasia -Small carious lesion on lower L M1	-Peri-mortem fractures of R humerus, R femur, and possible wedge fracture of T12
A998	20-34	Male	170.84±3.91	-Porotic hyperostosis -Slight cribra orbitalia L orbit -Periostitis tibia -Slight lateral bending at subtrochanteric region of R and L femur	-Enamel hypoplasia -Dental calculus -Large carious lesion on upper L M1	None
A999	20-34	Male	165.56±3.91	-Cribra orbitalia	-Enamel hypoplasia -Dental calculus -Possible periodontal disease	Post-mortem autopsy cut marks
A1000	35-45	Male	162.62±3.25	-Porotic hyperostosis -Periostitis tibia -Osteophytic lipping of L5, calcaneus and talus	-Enamel hypoplasia -Dental calculus -Severe caries -Abscess upper L M2 -Periodontal disease -Ante-mortem tooth loss	None
A1001	20-34	Male	168.20±3.91	-Porotic hyperostosis -Slight cribra orbitalia -Periostitis tibia -Slight medio-lateral bowing of tibia and fibula -Sacralization of L5 -Cleft neural arch L5	-Enamel hypoplasia -Dental calculus -Carious lesions on upper and lower molars -Possible periodontal disease	None
A1002	20-34	Male	166.0±3.91	-Possible cribra orbitalia -Possible spina bifida	-Enamel hypoplasia -Small carious lesions	None

				occulta -Cleft neural arch L5		
A1003	35 – 45	Male	152.87±3.25	-Porotic hyperostosis -Possible cribra orbitalia -Periostitis tibia	None	-Possible peri-mortem fracture of T2 spinious process, R os coxa -Ante-mortem fracture of R fibula shaft and distal epiphysis
A1004	>20	Male	-	-Periostitis femur and tibia	-Enamel hypoplasia -Large carious lesions in upper L M2 and lower R M1 and M2	-Peri-mortem fractures of the L radius, R femur, R tibia and fibula
A1005	20-34	Male	163.91±3.91	-Porotic hyperostosis -Possible Cribra orbitalia -Medio-lateral bowing of tibia and fibula	-Slight enamel hypoplasia	None
A1006	35-45	Male	170.55±3.25	-Possible porotic hyperostosis -Medio-lateral bowing of fibula	None	None
A1007	35-45	Male	169.25±3.25	-Spondylolysis of L5 -Osteophytic lipping of ulna and lumbar vertebrae	-Enamel hypoplasia	None
A1008	35-45	Male	158.72±3.25	-Possible caries sicca -Osteophytic lipping of lumbar vertebrae -Osteophytic outgrowth L proximal tibia and L clavicle -Sacralisation of L5	-Enamel hypoplasia -Dental calculus -Periodontal disease	None
A1009	20-34	Possibly male	159.01±4.31	-Porotic hyperostosis -Cribra orbitalia	-Enamel hypoplasia	-Peri-mortem fractures of the L femur, tibia and fibula
A1010	20-34	Male	166.99±3.91	-Slight medio-lateral bowing of fibula -Slight periostitis tibia	-Enamel hypoplasia -Dental calculus	None
A1011	20-34	Male	158.16±3.87	-Porotic hyperostosis -Slight medio-lateral bowing of ulna, tibia and fibula	-Enamel hypoplasia -Dental calculus	-Peri-mortem fracture L femur -Possible peri-mortem fracture of T4

				-Slight periostitis tibia -Partial sacralisation of L5		
A1012	35-45	Possibly male	-	-Osteophytic lipping of proximal tibia and talus	-Enamel hypoplasia -Possible periodontal disease	None
A1013	20-34	Male	166.33±3.91	-Periostitis femur and tibia	-Enamel hypoplasia -Dental calculus -Possible periodontal disease	-Possible peri-mortem fractures of L3-5
A1014	20-34	Male	158.58±3.27	-Porotic hyperostosis	-Enamel hypoplasia -Possible periodontal disease	None
A1015	20-34	Male	166.07±4.31	-Possible porotic hyperostosis	-Enamel hypoplasia -Dental calculus	-Peri-mortem fractures of R humerus, and L femur and tibia
A1016	35-45	Male	168.60±3.25	-Porotic hyperostosis -Slight periostitis tibia	-Enamel hypoplasia -Dental calculus -Small carious lesions	None
A1017	20-34	Possibly male	161.49±3.87	None	-Enamel hypoplasia -Dental calculus	None
A1018	20-34	Male	164.64±3.48	-Porotic hyperostosis	None	None
A1019	20-34	Male	165.23±3.91	-Periostitis of femur and tibia especially R proximal tibia	-Enamel hypoplasia -Dental calculus	None
A1020	>20	Possibly male	-	-Periostitis L femur	-Enamel hypoplasia	None
A1021	35-45	Possibly male	-	-Possible porotic hyperostosis	-Dental calculus	None
A1022	20-34	Male	163.25±3.91	-Porotic hyperostosis -Possible slight cribra orbitalia R orbit -Periostitis femur -Slight medio-lateral bowing of fibula -Possible osteomyelitis R calcaneus	-Enamel hypoplasia -Dental calculus -Abscess upper R M2	None
A1023	20-34	Possibly male	157.93±3.87	-Porotic hyperostosis -Schmorl's nodes on T8-10	-Enamel hypoplasia	-Possible peri-mortem fracture of L femur

				-Spondylolysis -Possible osteomyelitis L distal femur		
A1024	20-34	Possibly male	154.46±4.13	-Osteophytic lipping of proximal ulna	-Dental calculus -Carious lesions on upper L M2 and M3	None
A1025	Adult	Possible male	-	-Possible medio-lateral bowing femur in subtrochanteric region	-Enamel hypoplasia	None
A1026	Adult	Possible male	163.71±3.87	-Slight osteophytic lipping of talus	None	None
A1027	Adult	Possibly male	-	-Slight medio-lateral bowing of tibia	None	None
A1028	Adult	Male	156.82±3.48	-Slight periostitis femur and tibia	None	None
A1029	35-45	Possibly male	158.64±3.91	-Cribra orbitalia	-Enamel hypoplasia -Dental calculus -Large carious lesion on upper R M2 (possible abscess) -Carious lesion on lower L M2 -Antemortem tooth loss of lower R M3 -Possible periodontal disease	None
A1030	20-34	Male	163.14±3.91	-Porotic hyperostosis -Spina bifida occulta -Notochord defect T7-11	-Possible periodontal disease	-Possible peri-mortem wedge fracture of T12 -Possible peri-mortem fracture R tibia -Ante-mortem depressed fracture on L parietal
A1031	20-34	Possibly male	164.24±3.91	-Periostitis femur and tibia -Schmorl's nodes on L1-4 -Notochord defect of T10-12	-Enamel hypoplasia -Dental calculus	-Ante-mortem fracture of L foot affecting first metatarsal and associated tarsal

Table 4. 2: Frequency of pathological lesions observed in the 36 Chinese miners

N = number of individuals.
***= inspected if at least 80% of vertebral column was present.**
[1]= this was listed under the heading trauma although it may also be placed under Developmental Defects.
[2]= this was listed under the heading of Trauma although it may also be placed under Degenerative Changes.

Pathology	N affected	N inspected
DEVELOPMENTAL DEFECTS		
Sacralisation	3 (12%)	25
Spina bifida occulta	2 (10%)	20
Cleft neural arch*	2 (12.5%)	16
Notochord defects *	2 (12.5%)	16
METABOLIC DISEASES		
Osteomalacia / Residual rickets (tibia)	4 (11.8%)	34
Osteomalacia / Residual rickets (fibula)	6 (21.4%)	28
Osteomalacia / Residual rickets (femur)	2 (6%)	33
Porotic hyperostosis	18 (78.3%)	23
Cribra orbitalia	9 (34.6%)	26
DENTAL PATHOLOGY		
Enamel hypoplasia	27 (93.1%)	29
Caries	10 (34.5%)	29
AMTL	6 (22.2%)	27
Dental calculus	17 (58.6%)	29
Periodontal disease	9 (33.3%)	27
DEGENERATIVE CHANGES		
Vertebral osteophytosis*	3 (18.8%)	16
Peripheral osteoarthritis (elbow)	2 (9.1%)	22
Peripheral osteoarthritis (knee)	2 (7.1%)	28
Peripheral osteoarthritis (ankle)	3 (12%)	25
INFECTIONS		
Periostitis (tibia)	13 (38.2%)	34
Periostitis (femur)	9 (27.3%)	33
Osteomyelitis	2 (7.2%)	28
Caries sicca (syphilis)	1 (3.6%)	28
TRAUMA		
Ante-mortem fractures	3 (10%)	30
Spondylolysis[1]	2 (12.5%)	16
Schmorl's nodes[2]	3 (18.8%)	16
Peri-mortem fractures	9 (30%)	30
Multiple peri-mortem fractures	8 (26.6%)	30

Table 4. 3: Enamel hypoplasia intensity sorted by tooth type

N=number inspected
NA=number affected

Tooth type	N	NA	%
I1	43	16	37.2
I2	60	23	38.3
C	86	61	70.9
PM1	102	47	46.1
PM2	104	40	38.5
M1	113	26	23
M2	106	18	17
M3	62	0	0
Total	676	231	

Table 4. 4: Summary of dental caries frequency

[1]Total number of individuals affected/Total number of individuals inspected
[2]Total number of carious teeth/Total number of individuals affected
[3]Total number of carious teeth/Total number of teeth inspected
N=number inspected
NA=number affected
NTA=number of teeth affected
C/M=Average caries per mouth

Per individual[1]		
N	**NA**	**%**
29	10	34.5
Per mouth[2]		
N	**NTA**	**C/M**
10	28	2.8
Per tooth[3]		
N	**NTA**	**%**
676	28	4.1

Table 4. 5: Caries intensity sorted by tooth type

N=number inspected
NA=number affected

Tooth type	N	NA	%
I1	43	0	0
I2	60	0	0
C	86	0	0
PM1	102	2	2
PM2	104	2	1.9
M1	113	6	5.3
M2	106	13	12.3
M3	62	5	8.1
Total	676	28	

Table 4. 6: Summary of AMTL frequency

[1]Total number of individuals with AMTL/Total number of individuals inspected
[2]Total number of teeth lost ante-mortem/Total number of individuals inspected
[3]Total number of teeth lost ante-mortem/Total number of tooth sockets present
N=number inspected
NA=number affected
NTA=number of teeth affected
NTS=number of tooth sockets present

Individual AMTL frequency[1]		
N	**NA**	**%**
27	6	22.2
AMTL per mouth[2]		
N	**NTA**	**%**
27	9	33.3
AMTL intensity (tooth count)[3]		
NTS	**NTA**	**%**
826	9	1.1

Table 4. 7: Summary of pathologies associated with metabolic diseases as seen in cadaver and Chinese miner skeletal remains

Total number of cadaver skeletons examined = 100 Total number of Chinese miner skeletons examined = 36

N = total number of skeletons examined - = skeletal element was not present

N = number of skeletons with the specific skeletal element (usually associated with the form of pathology mentioned)

Pathological condition	**Cause of death as indicated in cadaver records**						
	Liver cirrhosis/alcoholism (N=38)	**Beriberi (N=1)**	**Pellagra (N=12)**	**Scurvy (N=1)**	**General malnutrition (N=55)**	**Anaemia (N=1)**	**Chinese miners (n=36)**
Porotic hyperostosis	26 (83.9%) *n=31*	1 (100%) *n=1*	8 (72.7%) *n=11*	1 (100%) *n=1*	29 (55.8%) *n=52*	1 (100%) *n=1*	18 (78.3%) *n=23*
Cribra orbitalia	17 (51.5%) *n=33*	0 (0%) *n=1*	9 (81.8%) *n=11*	1 (100%) *n=1*	30 (57.7%) *n=52*	1 (100%) *n=1*	9 (34.6%) *n=26*
Periostitis (tibia)	32 (91.4%) *n=35*	1 (100%) *n=1*	7 (70%) *n=10*	-	28 (53.8%) *n=52*	0 (0%) *n=1*	13 (38.2%) *n=34*
Periostitis (other)	20 (55.6%) *n=36*	1 (100%) *n=1*	7 (63.6%) *n=11*	-	23 (44.2%) *n=52*	0 (0%) *n=1*	9 (27.3%) n=33
Medio-lateral bowing (tibia and fibula)	4 (11.4%) *n=35*	0 (0%) *n=1*	2 (20%) *n=10*	-	2 (3.8%) *n=52*	0 (0%) *n=1*	7 (25%) *n=28*
Developmental defects (vertebra)	8 (22.2%) *n=36*	1 (100%) *n=1*	2 (18.2%) *n=11*	-	4 (7.7%) *n=52*	0 (0%) *n=1*	6 (37.5%) *n=16*
Enamel hypoplasia	6 (16.7%) *n=36*	0 (0%) *n=1*	5 (45.5%) *n=11*	1 (100%) *n=1*	10 (20.4%) *n=49*	0 (0%) *n=1*	27 (93.1%) *n=29*

Figure 4. 1: Sacralization with L5 also presenting with a cleft neural arch (indicated by the white arrow) (A1001)

Figure 4. 2: Spina bifida occulta (A1030)

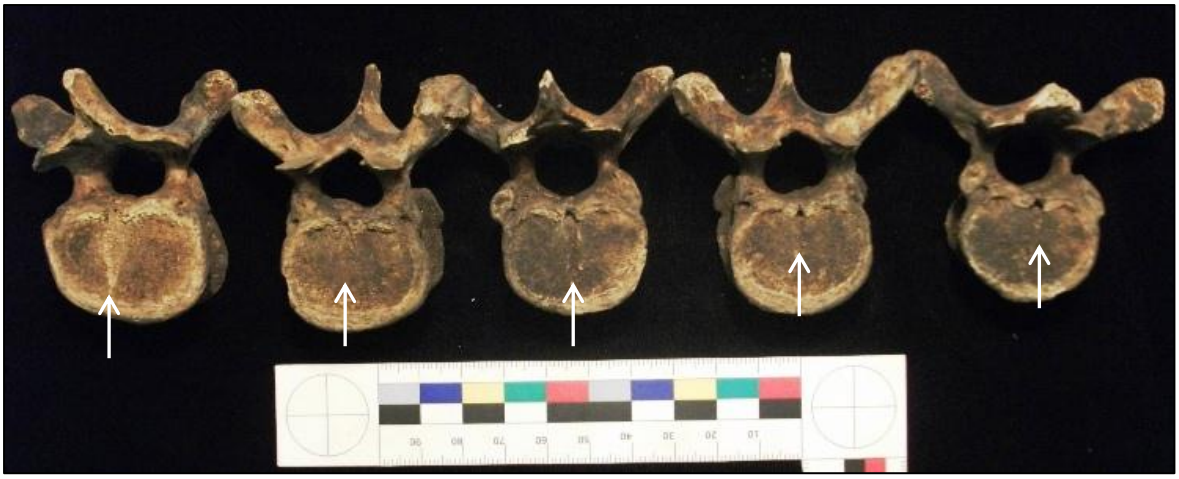


Figure 4. 3: Notochord defects (A1030)

Figure 4. 4: Cleft neural arch (A1002)

Figure 4. 5: Bowing of left tibia and fibula (A1011)

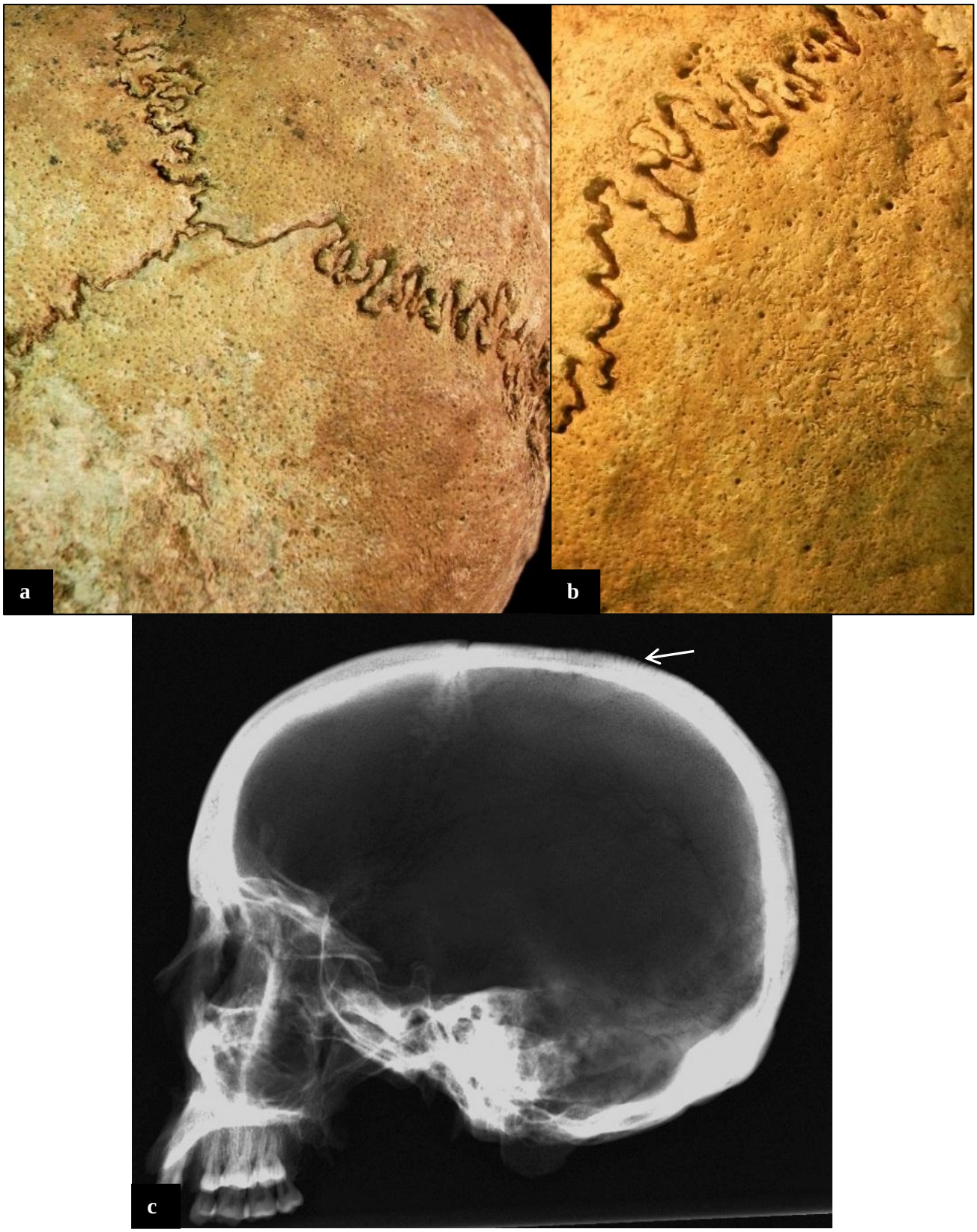


Figure 4. 6 a: Porotic hyperostosis observed on the occipital and parietal bones (A1011) b: Close up of porotic lesions observed on the occipital bone (A1009) c: Lateral aspect X-ray indicating hair-on-end appearance associated with porotic hyperostosis (A1030)

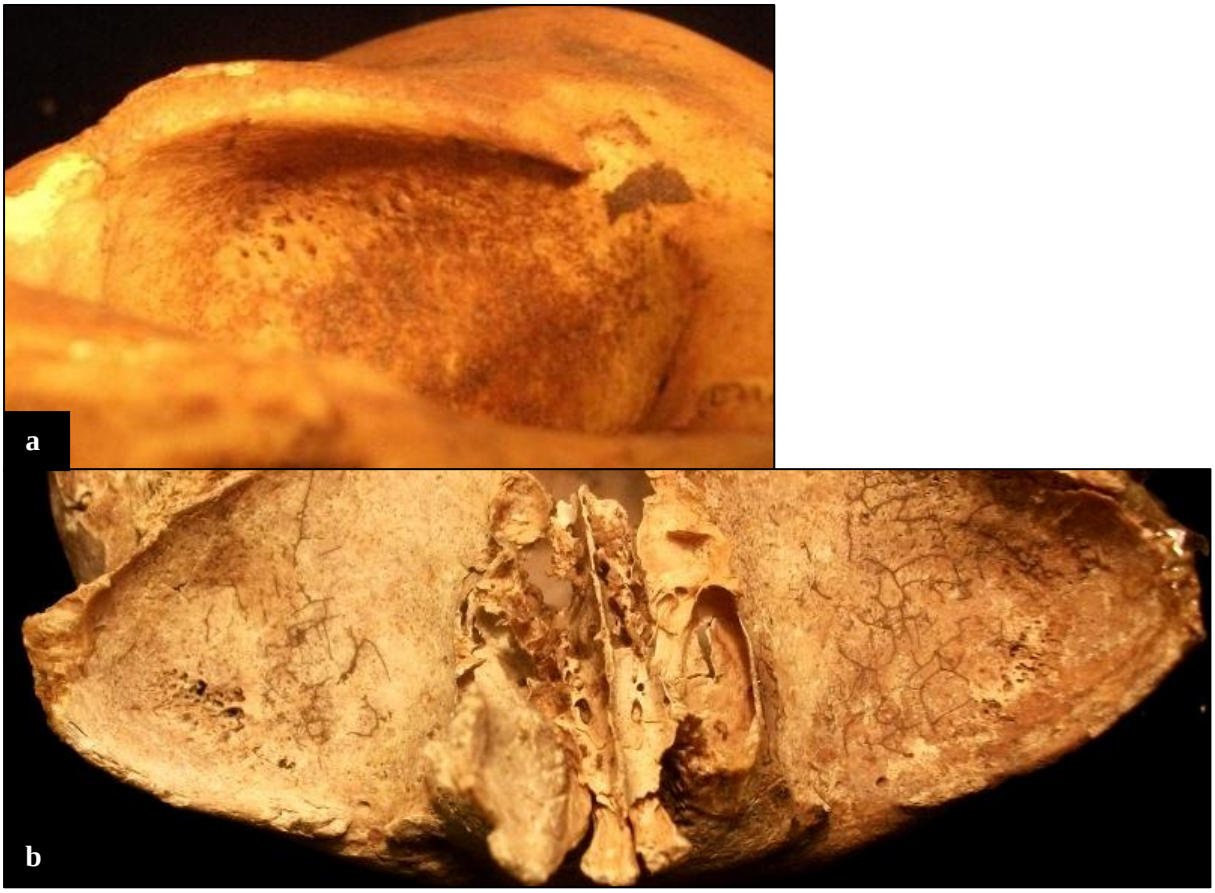


Figure 4. 7 a: Healed/healing cribra orbitalia (A1029) b: Possible active cribra orbitalia (A1009)

Figure 4. 8: Periostitis on the tibia (A998)

Figure 4. 9: Possible caries sicca (A1008)

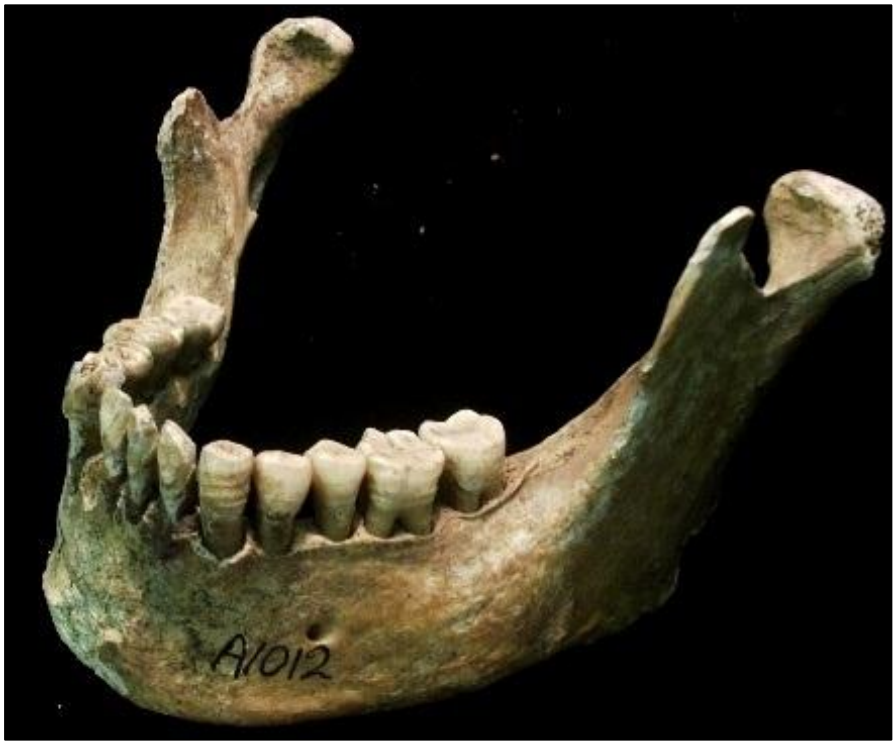


Figure 4. 10: Linear enamel hypoplasia (A1012)

Figure 4. 11: Ante-mortem fractures of a left fibula (A1003)

Figure 4. 12: Ante-mortem healed depressed fracture of left parietal bone (A1030)

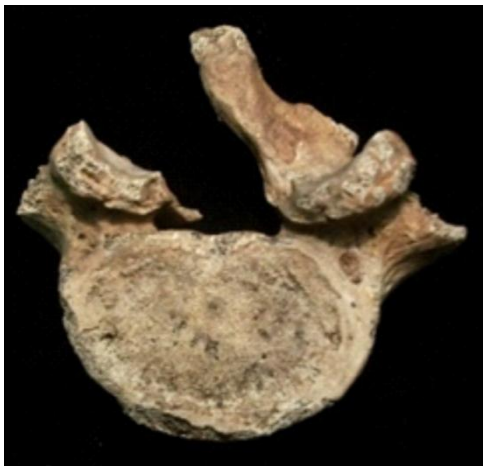


Figure 4. 13: Unilateral spondylolysis of L5 (A1023)

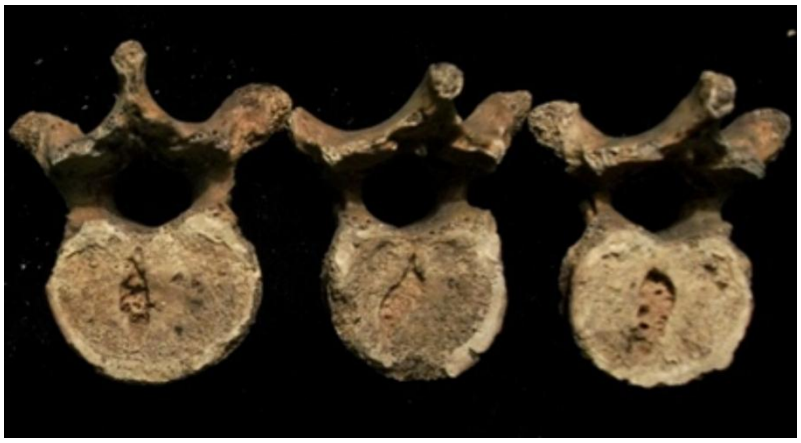


Figure 4. 14: Schmorl's nodes (A1023)

Figure 4. 15: Complete transverse fracture of the right humerus (A997)

Figure 4. 16: Burst fractures of the lumbar vertebrae (A1013)

Figure 4. 17: Spiral fracture of the right femur (A997)

Figure 4. 18: Butterfly fracture of the right femur (A1004)

CHAPTER 5: DISCUSSION

This study set out to assess any signs of metabolic bone disease in the skeletal remains of 36 Chinese miners who worked and died on the Witwatersrand mines during the period of Chinese indentured labour importation (1904-1910). In this study the identification and interpretation of nutritional and metabolic disease-related pathologies provided additional insight into the lifestyle and health of the Chinese miners prior to their arrival in South Africa as well as during their time on the Witwatersrand mines. Physical anthropological analyses of these remains provided an alternative means for cross referencing historical accounts concerning the circumstances surrounding the importation and employment of the indentured Chinese labourers. Specific to this were the identification and interpretation of pathology associated with metabolic and nutritionally related diseases. In order to better interpret the pathology seen in the Chinese miners' remains and cadaver skeletal remains, for whom the cause of death was known and related to metabolic and nutrition-related diseases were assessed. This aided in the interpretation of skeletal pathology and provided insight into the possible disease aetiologies associated with each. One of the shortcomings of this study was small sample sizes. The Chinese miners were only represented by 36 individuals currently housed in the Raymond A Dart Collection as part of their archaeological skeletal collection. These remains are the only known sample representative of this very short period in South African history. Small sample sizes were also noted for some of the cadaver groups. Of the 100 cadaver skeletons selected over half of this sample (55%) represented individuals who died as a result of general malnutrition. The generalized cause of death given for these cadaver remains meant that pathology observed could not be linked with any specific vitamin or mineral deficiency. The extremely small sample sizes for the more specific nutritional diseases like beriberi, anaemia and scurvy, only represented by one individual each, meant that the results obtained here were not statistically comparable. One of the limitations in this study was not having a complete background history for the cadaver remains. As the only information provided for cadaver remains is the final condition causing death, information pertaining to other underlying conditions (chronic and acute) which would not necessarily have caused the individual's death but which may have affected the person's skeletal health, is absent. This information would have been beneficial in more accurately interpreting skeletal pathology on the basis of disease aetiology. This study nevertheless provided some indication of the patterns and incidence rate of pathological lesions associated with specific

vitamin and mineral deficiencies and also aided in the overall interpretation of skeletal pathology observed in the Chinese miners.

5.1. Demographic characteristics of the Chinese miners

5.1.1. Age distribution

The age distribution of the 36 Chinese miners reflects the archival and historical records and in that it represents a demographic pattern consistent with migrant labourers. The majority (58%) of the Chinese individuals were estimated to be of young adult age (20-34 years). This is consistent with archival and historical records which suggests that the majority of individuals recruited for work on the Witwatersrand mines were between 20 and 35 years (Richardson, 1982). The general age distribution of Chinese employed on the Witwatersrand mines, however, ranged from 20-55 years (Richardson, 1982). This then accounts for the nine Chinese miners' skeletons estimated to be of middle adult age (35-45 years). The remaining two individuals were estimated to be younger than 20 years which does not seem to fit the general age distribution provided by archival and historical records. Employment contracts stipulated that employees needed to be a minimum age of 20 (Richardson, 1982). However, this only applied in cases where parental consent could not be obtained. Historical records also indicate that these rules were not readily enforced by the Chamber of Mines and that in the end very few men were rejected on the grounds of age (Richardson, 1982). It therefore seems likely that some of the Chinese employed on the mines could have been younger than 20 years and would therefore account for the two adolescent individuals identified in the sample.

The majority of the Chinese miners' skeletons were therefore estimated to be of young adult age which corresponds with averages obtained for the Gladstone (20–34 years) and Koffiefontein (25–33 years) populations. The relatively young age at death observed in all three mining populations is representative of the high mortality rate associated with mining labour. Even today, mining is still readily associated with occupational hazards (physical, chemical, biological, ergonomic and psychosocial) and unfortunately still contribute to a large proportion of the work-related fatalities each year (Donoghue, 2004).

5.1.2. Sex distribution

The distribution of sex is also consistent with a migrant labourer population as all individuals were male. Molecular sex identification done on 31 of the 36 Chinese miners' remains concluded that 13 of these individuals were male whereas the remaining 18 did not yield any results due to poor preservation and degradation of DNA (Gibbon *et al.*, 2010). According to the historical records no women were employed on the mines (Richardson, 1982; Harris, 1998). Migrant populations, however, often included a small number of women and children. Many companies employing migrant labourers often make provision for wives and children by providing 'married quarters' in the compound areas. This was also the case for the Gladstone and Koffiefontein populations, as both these presented with a small number of female and juvenile remains. Provisions, in terms of married quarters within the compounds, were made for the Chinese men who wanted to bring their wives (families) with, however, in the end only six women were reported to have come to South Africa during the period of importation (1904 to 1907) (Harris, 1998). The likelihood of finding female skeletal remains is therefore very slim.

5.1.3. Stature

Stature estimations indicated that the Chinese miners were slightly shorter (3cm) than averages reported for late 19th and 20th century Northern Chinese (Morgan, 2004). The reason for this may be twofold. Comparable averages were obtained from living individuals (Morgan, 2004). It may therefore be possible that the 3cm difference in stature is representative of the error margin between living stature calculations and stature calculations from skeletal remains. On the other hand, the slightly shorter stature may indicate growth stunting as a result of poor health and diet. The shorter stature can also be a factor of selection as most of the Chinese recruits came from the poorer peasant families which most probably already suffered growth stunts as a result of poor diet and health.

5.2. Pathology

5.2.1. Developmental defects

The Chinese labourers presented with developmental defects such as spina bifida occulta (10%), notochord defects (12.5%) and sacralisation of L5 (12%). Only spina bifida occulta (3.4%) was observed in the Gladstone population and was not considered to be

statistically significantly different to that observed in the Chinese (χ^2=1.567; P-value > 0.05; Fisher's exact test P=0.2337). Even though there is no statistical difference in the frequencies observed in the Chinese miners and the Gladstone populations the fact that more than one type of developmental defect was observed in the Chinese may suggest, to some degree, a congenital predisposition amongst the Chinese for these kinds of pathologies. Merbs (2004) also noted a relatively high frequency of sagittal clefting in historic Canadian Inuit populations (11.9%). When compared to the 12.5% observed in the Chinese miners no statistically significant difference could be obtained (χ^2=0.005; P-value > 0.05; P=0.9456). This study also indicated that a large proportion of individuals presenting with clefting also presented with other developmental defects such as spina bifida and notochord defects (Merbs, 2004). When considering the variety of developmental defects observed in the Chinese miners and the Inuit populations it may to some extent suggest that these defects are more pronounced in certain Asian populations, possibly due to a genetic predisposition. Some developmental defects such as spina bifida occulta and notochord defects have, however, also been suggested to be representative of a poor maternal health during pregnancy resulting from vitamin deficiencies (vitamin B12, iron, magnesium, folic acid, and zinc; Groenen *et al.*, 2004a; Groenen *et al*, 2004b). The relatively frequent occurrence of these developmental defects in the Chinese miners may thus also reflect the overall poor health in their country of origin, as these conditions are often related to maternal health and therefore represents the previous generation's health.

5.2.2. Metabolic bone diseases and non-specific signs of disease

- **Bowing of long bones**

Bowing deformities (Tibia=11.8%; Fibula=21.4%; Femur=6%) were observed in the long bones of nine of the Chinese miners. Deformation of the long bones is often associated with a vitamin D deficiency. Vitamin D plays a vital role in the mineralization of bone protein or osteoid ensuring bone strength and integrity (Aufderheide and Rodríguez-Martin, 1998; Ortner, 2003; Brickley and Ives, 2008). An indicator of vitamin D deficiency is the deformation of long bones with the medio-lateral or antero-posterior bending of weight bearing bones as seen in several of the Chinese individuals (Ortner, 2003; Brickley and Ives, 2008). These skeletal deformities cannot be ascribed to the adult form of vitamin D deficiency (osteomalacia) because other diagnostic lesions associated with osteomalacia are absent in these individuals. However, residual rickets, being a persistent childhood vitamin D

deficiency, leaves permanent deformities to the long bones even after vitamin D supplement in adulthood (Brickley *et al*., 2010). Unlike vitamin C, the human body can produce vitamin D itself. This ability depends on the exposure of the skin to the ultraviolet rays from sunlight or on the dietary intake of vitamin D from animal fats (Brickley and Ives, 2008; Pettifor, 2004). In addition to vitamin D, dietary calcium is also essential for the mineralization of the bone tissue precursor osteoid. This means that a deficiency in calcium on its own will also result in pathological manifestations as seen in rickets (Pettifor 2004).

Vitamin D deficiency has been described as an endemic condition in many provinces of north and south China (Lui *et al.*, 1937; Snapper; 1956; Chen and Xu, 1996; Popkin *et al.*, 2001; Du *et al.*, 2002; Fraser, 2004; Wat *et al*., 2007). This has been attributed to a lack of dietary vitamin D and calcium in the traditional Chinese diet (Lui *et al.*, 1937; Snapper; 1956; Chen and Xu, 1996; Du *et al.*, 2002; Fraser, 2004; Wat *et al*., 2007). "*In these regions* [north China], *the food is very poor in calcium, not high in phosphorus, and very low in vitamin D. In China, milk and butter are unknown as dietary staples, and eggs, liver, sea food, meat, fowl, and other animal products can be eaten only by the better-situated classes. The main source of calories for the poorer classes of north China consists of cereals, mostly millet, kaoliang (broom corn), and maize, eaten with varying amounts of vegetables*" (Snapper, 1956: 354). In some cases, where people were financially better situated, wheat, soybean and considerable amounts of vegetables would have been consumed (Snapper, 1956).

Vitamin D would therefore only have been available through sun exposure as the traditional Chinese diet contained almost no vitamin D. Even though China receives adequate amounts of ultraviolet B necessary for the synthesis of vitamin D, vitamin D is not the only vitamin necessary for calcium homeostasis and optimal bone formation. Dietary calcium has already been mentioned as an essential vitamin in the mineralization of bone osteoid, which if not formed properly will affect the structural integrity of bone which in turn may result in bowing deformities in the long bones (Ortner, 2003; Pettifor, 2004; Brickley and Ives, 2008; Waldron, 2009). Therefore, even though the Chinese may have had some vitamin D available through sun exposure, the severe lack of calcium in their diet still predisposed them in acquiring rickets or osteomalacia.

This condition was not observed in either the Gladstone or Koffiefontein populations. The reason for this may be that both comparable populations grew up in the southern regions of Africa and as a result had ample exposure to ultraviolet rays whereas China is somewhat further removed from the equator. Yet, one study conducted in South Africa did notice the effects of vitamin D deficiency or rickets amongst children readily exposed to sunlight

(Pettifor, 2004). In this case the lack of sun exposure was not the debilitating factor but rather the lack of calcium in these children's diet. This may also have been the case for the Chinese. Access to meat and animal products, especially dairy, were often limited to the more affluent families and with famines looming over China, as a result of internal and international struggles peasant families were becoming more malnourished (Richardson, 1982; Chen and Xu, 1996; Du *et al.*, 2002).

Whatever the underlying causes, the pathological lesions observed in these Chinese individuals are most probably the result of a deficiency in either vitamin D and/or calcium. The lack of any pathological lesions associated with the adult form of vitamin D deficiency seems to suggest that the bowing deformities observed are the result of a chronic childhood vitamin D/calcium deficiency or what is termed residual rickets (Brickley *et al.*, 2010). This therefore strongly suggests that the Chinese miners experienced malnutrition and poor health prior to their arrival in South Africa.

- **Porotic hyperostosis and cribra orbitalia**

Porotic hyperostosis (78.3%) and cribra orbitalia (34.6%) were observed in the Chinese miners. These two conditions are often described as indicators of nonspecific signs of disease and stress. Both these conditions have also been linked with metabolic bone diseases and nutritional deficiencies like scurvy, rickets/osteomalacia and more commonly iron deficiency anaemia (Stuart-Macadam, 1989; Ortner, 2003; Kozłowski and Witas, 2012). The specific cause of porotic hyperostosis and cribra orbitalia is, however, still debated in the literature. Both conditions are more often associated with deficiencies of iron and vitamin C (ascorbic acid) or with enteric parasitic diseases (Walker, 1985; Stuart-Macadam, 1989). The iron deficiency anaemia theory has recently been disclaimed by Walker *et al.* (2009) who argued that iron deficiency decreases the production of mature red blood cells and therefore cannot cause bone marrow hyperplasia. Oxenham and Cavill (2010), on the other hand, suggested that this is not necessarily the case and that even though iron deficiency decreases the production rate for functional erythrocytes the actual erythropoietic activity is increased. This suggests that bone marrow hyperplasia and the hypertrophic lesions associated with it can indeed occur as a result of iron deficiency anaemia. The appearance of hypertrophic lesions may therefore be caused by haemolytic anaemia (thalassemia, sickle cell anaemia), megaloblastic anaemia (chronic dietary deficiency, malabsorption, deficiencies of the

vitamins B12 and B9) and iron deficiency anaemia (Walker *et al.*, 2009; Oxenham and Cavill, 2010).

When considering the presence of porotic hyperostosis and cribra orbitalia in the Chinese miners the absence of associated skeletal pathologies, like ossified haematomas (Van der Merwe *et al.*, 2010b), scurvy was not considered here as a contributing factor in the appearance of these lesions. Porotic lesions have also been associated with rickets and osteomalacia, yet, this was also dismissed as being a possible cause for the appearance of porotic hyperostosis and cribra orbitalia. The reasoning behind this stems from the fact that the only other pathology associated with a vitamin D deficiency that could be observed in the Chinese was the bowing of the some of the long bones, especially the tibiae and fibulae. Pathology indicating active osteomalacia could not be observed. As has already been discussed these deformities were not attributed to active rickets or osteomalacia but rather residual rickets which represents the healed, but altered effects, of persistent childhood rickets. Even though these deformities indicate a vitamin D deficiency earlier on in life this condition was not active in adulthood and can therefore not be the cause of porotic lesions associated with the active form of the disease.

It is difficult to attribute the presence of porotic hyperostosis and cribra orbitalia to haemolytic, megaloblastic or iron deficiency anaemia. Medical records for the years 1905 to 1906, reflecting the causes of death for Chinese employees of Witwatersrand Deep Ltd., indicated that at least one individual died as a result of malaria (FLD 90, 13/7; FLD 91. 13/7; FLD 127, 18/7; FLD 166, 32/7). This disease could have been contracted in China; however, malaria is mostly limited to the tropical areas of southern China (Tian *et al.*, 2008). Since the majority of the Chinese employed on the mines came from the northern regions it is unlikely that many would have been infected with malaria or that they would have presented with thalassemia or sickle cell anaemia. Malaria could also have been contracted when the Chinese originally embarked in Natal before being transported by train to the Transvaal (Richardson, 1982; Yap and Man, 1996). This may therefore suggest that haemolytic anaemia could be a probable cause for the some of the hypertrophic lesions observed. However, it was not likely to have been a major cause as only one case of malaria was reported in two years. The fact that almost all the porotic lesions observed on the Chinese showed signs of healing further indicates that a chronic condition like malaria (thalassemia and sickle cell anaemia), which would have left active and intensive lesions affecting the whole skeleton (Kozłowski and Witas, 2012), was not the likely cause for the appearance of porotic hyperostosis and cribra orbitalia in the Chinese miners.

Beriberi (dietary deficiency of thiamine/vitamin B1 deficiency) was also a major cause of death amongst the Chinese (Yap and Man, 1996). The 1905 to 1906 medical records for Witwatersrand Deep Ltd mine indicated that 11 individuals died as a result of beriberi (FLD 90, 13/7; FLD 91. 13/7; FLD 127, 18/7; FLD 166, 32/7). Even though dietary beriberi is not directly associated with anaemia, a rare autosomal recessive disorder called thiamine-responsive megaloblastic anaemia syndrome, is invariably associated with anaemia (Diaz *et al.*, 1999; Mathews *et al.*, 2009). It may therefore be possible that a severe dietary beriberi or thiamine deficiency may also be associated with anaemia. Even if a dietary deficiency in thiamine is not extreme enough to cause anaemia, such as in the case of the hereditary form, deficiencies in several of the other B vitamins can lead to megaloblastic anaemia. This includes vitamins B3, B6, B9 and B12 (Ball, 1998; Truswell and Milne, 2002; Dakshinamurti and Dakshinamurti, 2007; Green and Miller, 2007; Suskind, 2009; Walker *et al.*, 2009). Vitamin B12 is almost exclusively found in animal products (Walker *et al.*, 2009) and as a result Chinese would most probably have been deficient in vitamin B12 as well. Although many non-animal derived foodstuffs, like green vegetables and yeast, which may have been consumed by the Chinese, and which contain folic acid it can easily be destroyed by heat during cooking (Bailey, 2007; Suskind, 2009). The consumption of a cereal-based diet, such as that consumed by the Chinese, can also predispose an individual in developing a folic acid deficiency as carbohydrate type foods only contain trace elements of folate and iron (Rose, 1982). Additionally, many of the B vitamins work in association with each other (coactivation), and are contained in similar food sources, which would make it more likely that the Chinese would have suffered from more than one vitamin B deficiency. This in turn may, therefore, partly be responsible for the occurrence of megaloblastic anaemia and the appearance of porotic lesions in the Chinese miners.

Medical records for 1905 to 1906 also indicated that conditions that may lead to iron deficiency anaemia were present. This includes enteric fever (cause of death for 9 individuals), haemorrhage (cause of death for 2 individuals), and chronic diarrhoea (cause of death for 4 individuals) (FLD 90, 13/7; FLD 91. 13/7; FLD 127, 18/7; FLD 166, 32/7). However, since the porotic lesions observed in the Chinese presented with a degree of healing it is unlikely that these conditions (which would have occurred just prior to death and therefore would have resulted in an active form of porotic hyperostosis and cribra orbitalia) would have been the cause for its appearance. In general the traditional Chinese diet did not contain a lot of animal products. This was especially the case in peasant societies where meat would only have been consumed on very special occasions or festivals (Chen and Xu, 1996;

Du *et al.*, 2002). Everyday diets mainly consisted of cereals (usually rice in the south and wheat, millet and maize in the north) with vegetables. These diets were typically low in fat, low-energy and very high in carbohydrates and dietary fibre (Du *et al.*, 2002). The lack of animal products may have contributed to a lack of dietary iron which in turn may have caused acquired iron deficiency anaemia. Plant derived proteins would, however, have been consumed which would have made iron available. Yet it is known that heme sources of iron (animal derived) are more readily absorbed than nonheme (plant derived) sources of iron (Larsen, 1997). Furthermore, phytates, found in many nuts, cereals and legumes, may actually inhibit iron absorption (Larsen, 1997; MacPhail, 2002). Both the southern and northern Chinese diets contained large amounts of cereals (phytates) which may have inhibited the little iron already consumed. It may therefore be likely that the Chinese miners, coming from peasant societies and therefore known to have had poor nutrition, could have suffered from iron deficiency which may explain the presence of porotic lesions. The presence of these lesions may therefore be a reflection of the general malnutrition and poor health experienced by the Chinese prior to their importation.

Only cribra orbitalia was noted in the Gladstone (11%) and Koffiefontein (8.3%) populations. These frequencies were statistically somewhat lower than the 34.6% observed in the Chinese (Gladstone: χ^2=7.943; P-value <0.05; Fisher's exact test P=0.0124; Koffiefontein: χ^2=6.681; P-value <0.05; Fisher's exact test P=0.0198). This may suggest to some degree that the living conditions back in China were somewhat worse than that experienced by the local Gladstone and Koffiefontein populations.

In general this is a condition not frequently seen in archaeological southern African populations. For example no cases of adult porotic hyperostosis or cribra orbitalia were reported for the Toutswe tradition sites (Mosothwane and Steyn, 2009) and the Venda (L'Abbé and Steyn, 2007). Some other archaeological populations, however, presented with cribra orbitalia and include the Stone Age adult remains from Oakhurst (20%; Patrick, 1989) and the Iron Age adult remains from Mapungubwe/K2 (33.3%; Steyn, 1997). Frequencies of cribra orbitalia in these groups seem to be similar to the frequency obtained in the Chinese miners (34.6%). In both the Oakhurst and Mapungubwe/K2 populations the appearance of cribra orbitalia was ascribed to an iron deficiency caused by parasitic infections (Patrick, 1989; Steyn, 1997). Even though the exact cause of the cribra orbitalia in the Chinese is not known their dietary practices seem to indicate that the presence of these porotic lesions can rather be ascribed to a dietary deficiency in iron and/or B vitamins. Comparing the frequency of cribra orbitalia observed in the Chinese miners with a prehistoric Chinese sample from the

Longshan period (51.8%; 5000-2200 BP; Pechenkina *et al.*, 2002) revealed no statistically significant difference (χ^2=1.602; P-value < 0.05; P=0.2056). Even though the Longshan Chinese predate the historic Chinese miners by a couple of thousand years, both these populations had similar diets. The Longshan period saw the shift towards a more restricted carbohydrate rich diet. Up to 70% of their diet consisted of millet (Pechenkina *et al.*, 2002). The availability of animal proteins, minerals and vitamins was also severely reduced. This would therefore have been somewhat similar to the traditional diet consumed by the historic Chinese miners. For this reason the frequency of cribra orbitalia observed in the Chinese miners may thus be reflective of the overall incidence rate expected for the Chinese population and may to some extent be associated with their dietary practices.

The occurrence of porotic hyperostosis in the Chinese miners (78.3%), however, remains significantly higher when compared to other southern African archaeological populations. When compared to frequencies of porotic hyperostosis in prehistoric Chinese skeletal samples (48% for the Longshan culture dating to 5000-4000 BP; Pechenkina *et al.*, 2002) there is still a slight statistically significant difference (χ^2=4.780; P-value < 0.05; Fisher's exacts test P=0.0417). This means that the frequency of porotic hyperostosis observed in the Chinese miners is somewhat higher than what would be expected for this population group, however this difference is very small and may just reflect slightly aggravated conditions brought on by the persisting floods and famines of late 19th century China (Richardson, 1982; Yap and Man, 1996).

In general the presence of healing/healed porotic lesions in the Chinese miners can therefore be interpreted as a reflection of the result of living and dietary conditions experienced in China, prior to their arrival in South Africa and not so much as an indication of their time on the Witwatersrand mines.

- **Periostitis**

Another form of pathology indicative of possible population stress and poor health is periostitis or periosteal new bone formation. Periostitis can occur anywhere in the body, but is more often found in the long bones, especially the tibia (Ortner, 2003; Weston, 2012). More than 20% of the total number of femora inspected and more than 30% of the total number of tibiae inspected in the Chinese miners presented with periostitis. Periostitis presented as multiple raised striations and seemed to be active in most cases. This condition may be the result of multiple afflictions including localised trauma (in most cases a unilateral

response), infections, metabolic diseases and overall malnutrition (Ortner 2003; Waldron 2009; Weston 2012). Generally, specific diagnosis is problematic. Recent studies by Weston (2008; 2009) investigated the specificity of periosteal reactions in pathology museum specimens by looking at the morphological and histological characteristics of lesions. However, no correlation to a specific disease could be found. Should periostitis occur in association with other, more diagnostic lesions such as treponemal diseases, tuberculosis and osteomyelitis, then the diagnosis can be more definitive (Ortner, 2003; Waldron, 2009). The periostitis observed on the Chinese miners' remains was not extensive and widespread enough to be associated with treponemal disease or tuberculosis in the case of hypertrophic pulmonary osteoarthropathy (Steyn *et al.,* 2013). Most of the periosteal new bone observed on the Chinese remains showed bilateral occurrence and may therefore be regarded, at least in part, as an indicator of non-specific infectious disease (Waldron, 2009), possibly set off by the over-crowded conditions associated with compound life. Bilateral involvement would also rule out most of the cases as localised trauma. Another probable cause of periostitis in the Chinese miners is metabolic insults as a result of environmental conditions and a nutritionally deficient diet. Periostitis is often associated with vitamin C and D deficiency (Ortner, 2003; Brickley and Ives, 2008), the latter of which has already been suggested as a possible cause for bowing deformities observed in the Chinese labourers' long bones.

Periostitis was also observed in the Gladstone (1.9% for the femora and 12.1% for the tibiae) and Koffiefontein (11.1% for the tibiae) individuals. The Chinese presented with much higher frequencies of periostitis in the femora (Gladstone: χ^2=22.482; P-value < 0.05; P=0.0001) and tibiae (Gladstone: χ^2=11.674; P-value < 0.05; Fisher's exact test P=0.0016; Koffiefontein: χ^2=8.867; P-value < 0.05; Fisher's exact test P=0.0040) which was found to be statistically significant. These differences may be attributed to the fact that the Chinese were probably suffering from more than one nutritional deficiency even before their arrival on the mines. Once they were employed on the mines they were further subjected to disease associated with the close confinement of mine compounds and additional hazards associated with mining which may have increased the appearance of periostitis.

- **Enamel hypoplasia**

Dental changes identified during the analysis suggested that these individuals encountered periodical hardships during childhood. Almost all of the individuals showed signs of linear enamel hypoplasia (93.1%). Enamel hypoplasia is an enamel defect that occurs

while tooth enamel is deposited during development (Hillson, 1998; Ortner, 2003). It may be caused by periods of malnutrition or illness (Larsen, 1997; Ortner, 2003). Enamel does not remodel, causing permanent visual indicators for stress (Roberts and Manchester, 1995; Larsen, 1997). These lesions were observed in all the tooth types and presented as multiple hypoplastic transverse defects, representative of reoccurring illnesses and/or periods of malnutrition.

The Gladstone (11%) and Koffiefontein (61.1%) populations both presented with enamel hypoplasia. Even though the frequency of enamel hypoplasia in the Koffiefontein individuals was quite high both of these populations presented with statistically smaller frequencies than that observed for the Chinese (Gladstone: χ^2=58.405; P-value < 0.05; P=0.0001; Koffiefontein: χ^2=8.859; P-value <0.05; Fisher's exact test=0.0035). An equally high prevalence of linear enamel hypoplasia has, however, been noted for prehistoric Chinese skeletons dating to the Longshan period (5000-2200 BP; Pechenkina *et al.*, 2002). An 84.6% prevalence rate of linear enamel hypoplasia was noted for the Longshan males. The high occurrence of enamel hypoplasia was attributed to a higher pathogen load as a result of the settlement overcrowding and iron deficiency anaemia brought on by intestinal parasites and a diet lacking in iron (Pechenkina *et al.*, 2002).

The high prevalence of linear enamel hypoplasia observed in the Chinese miners may therefore be a reflection of the overall conditions experienced by the less affluent families back in China. A traditional diet deficient in many essential vitamins and minerals and harsh living conditions brought on by the political and economic instability in China during the early 20th century may have contributed to the high percentage of stress indicators such as linear enamel hypoplasia.

5.2.3. Dental pathology

- **Dental caries**

Dental caries was observed in 34.5% of the Chinese miners. Caries is often observed in individuals consuming very carbohydrate rich diets. This is due to the fact that the dietary carbohydrates, especially sugars, in the right concentration of bacteria, ferment and produce organic acids which demineralize dental hard tissue and cause holes or carious lesions (Roberts and Manchester, 1995; Larsen, 1997; Lukacs, 2012). The Chinese miners would have consumed large amounts of carbohydrates in the form of wheat or wheat derived foodstuffs (back in China, especially northern China) and rice (in China, especially southern

China, as well as on the mines as part of the daily rations). Both these cereals are carbohydrate rich and would therefore have contributed to the appearance of dental caries. Food preparation methods can further increase the occurrence of caries. Rice in general is not very cariogenic, yet when it is prepared in the traditional Chinese way ("congee" by cooking it down with a lot of excess fluid) its consistency becomes much more like that of porridge which have been shown to actually enhance caries development (Tayles *et al.*, 2010). Foodstuffs with sticky paste-like consistencies are more likely to become lodged in between teeth (Tayles *et al.*, 2010). Here food particles are broken down into sugars, especially in the case of carbohydrates, which affect the mouth's natural pH balance. As the pH becomes more acidic demineralization of enamel takes place which enables the development of carious lesions (Roberts and Manchester, 1995; Larsen, 1997; Lukacs, 2012).

Dental caries has also been associated with vitamin D and calcium deficiencies (Brickley and Ives, 2008) which have been suggested as possible causes for other bone pathology observed in the Chinese miners. When considering the relatively low frequency of caries amongst the Chinese (4.1% of the teeth presented with carious lesions) it is however unlikely that the cause would be directly related to metabolic or nutritionally related disorders.

Both the Gladstone (47.8% of individuals) and Koffiefontein (86.1% of individuals) populations presented with dental caries. When compared to the frequency seen in the Chinese miners there does not seem to be any statistically significant difference in the occurrence of caries for the Gladstone population (χ^2=1.569; P-value > 0.05; P=0.2103). The Koffiefontein individuals did however present with statistically significantly higher frequencies of caries (χ^2=18.382; P-value < 0.05; P=0.0001). Dental caries frequencies observed in prehistoric Chinese from the Longshan period (5000-4000 BP) were also comparable to the frequency observed in the Chinese miners (χ^2=0.176; P-value > 0.05; P=0.6751; Pechenkina *et al.*, 2002). The Gladstone population as well as the Longshan population consumed agricultural diets consisting of carbohydrate based staple foods. Even though the actual carbohydrates differed (maize for the Gladstone populations and rice/wheat for the Longshan and Chinese miners) its bacterial fermentation would still have resulted in demineralization of the dental hard tissues. Dental caries is often associated with cereal based diets associated with the advent of agriculture (Larsen, 1997; Lukacs, 2012). When compared to the dental caries frequency observed in the much older prehistoric Chinese sample from the Yangshao period dating to 7000-5000 BP this becomes evident (Pechenkina *et al.*, 2002). The Yangshao Chinese diet was more varied and diverse, as the intensification of cereal-

based farming had not yet taken place. Subsistence strategies for the Yangshao period were therefore much like that practiced by hunter-gatherer populations (Pechenkina *et al.*, 2002). When the frequencies of caries observed in this early prehistoric Chinese population are compared to that of the much later Chinese miners a very statistically significant difference can be observed (χ^2=27.973; P-value < 0.05; P=0.0001). This therefore supports the notion that a cereal based (carbohydrate rich) diet associated with the introduction of agriculture would contribute to the incidence of caries. New research however cautions the use of caries as a sole indicator of dietary and subsistence change as its aetiology is multifactorial and complex (Lukacs, 2012). Some of the alternative mechanisms underlying cariogenesis are variations in the quality of enamel (which is invariably controlled by genes); variation in oral ecology; variation in dietary preferences; and variation in the pathogenic microorganisms of the oral cavity (Lukacs, 2012). This might help to explain the apparent difference in the frequency of dental caries observed in the Koffiefontein miners.

- **Ante-mortem tooth loss**

Ante-mortem tooth loss (AMTL) was observed in 22.2% of the Chinese miners. AMTL often occurs in the presence of dental disease such as caries, abscesses, dental calculus and periodontal disease (Roberts and Manchester, 1995; 2010). In the absence of other influencing factors, such as trauma and cultural practices of tooth extraction, AMTL frequencies should reflect the overall incidence of dental disease within a population (Roberts and Manchester, 1995; 2010). This relationship stems from the fact that without medical intervention dental caries is often treated by extraction. In cases where abscess formation or periodontal disease occurs, dental loss may be a natural process in the disease development (Hillson, 1998; Roberts and Manchester, 2010). The frequency of AMTL observed in the Chinese miners seems to reflect the same relative rate of dental caries which would be expected when factors such as trauma and extraction of teeth for cultural purposes are excluded. No trauma or associated facial trauma was observed in the cases where AMTL was present. Also no evidence could be found for cultural/ritual tooth extraction amongst the late 19th early 20th century Chinese.

When the AMTL frequency (per individual) for the Chinese miners was compared to frequencies obtained for the Gladstone (27.8%) and the prehistoric Longshan Chinese populations (18.9%) no statistically significant difference could be observed (Gladstone: χ^2=0.637; P-value > 0.05; P=0.4249; Longshan: χ^2=0.170; P-value > 0.05; P=0.6799). An

AMTL frequency (per individual) for the Koffiefontein population was not available for comparison here. This therefore corresponds with results obtained for the frequencies of caries observed in both the Gladstone and Longshan populations and again confirms the relationship between caries incidence and AMTL. When the AMTL frequency of the Chinese miners was compared to that of the much older prehistoric Chinese Yangshao population the latter population presented with statistically significantly lower AMTL frequencies (χ^2=9.533; P-value < 0.05; P=0.0020). This again would correspond with the relatively low frequencies observed in the Yangshao population which were attributed to differences in subsistence practices. It therefore seems that there is some relationship between dental caries intensity and AMTL.

Yet, when AMTL is compared to the frequency of dental calculus observed in the Chinese miners the dental calculus frequency (58.6%) is almost double that observed for AMTL. This is noteworthy when considering the fact that dental calculus (as a precursor for periodontal disease) is one of the major influencing factors in the appearance of AMTL. This difference may be explained as a factor of age. Higher frequencies of AMTL have been shown to occur in older individuals (Costa, 1980). Younger individuals generally present with higher frequencies of dental caries relative to AMTL as the diseased teeth still have to be shed. Older individuals, on the other hand, generally present with more AMTL and less caries as the diseased teeth are already extracted or lost due to the natural disease development processes (Roberts and Manchester, 2010). The majority of the Chinese miners were of young adult age at the time of their deaths which may explain why relatively little AMTL were noted when compared to the higher frequency of dental calculus present.

- **Dental calculus**

A relatively large proportion of individuals with dental calculus was observed in the Chinese miners (58.6%). This condition is caused by mineralized plaque that accumulates at the base of a living plaque deposit and attaches to the surface of the tooth (Hillson, 1998; Waldron, 2009; Roberts and Manchester, 2010). It is often sped up when there is a high protein and/or carbohydrate diet favouring an alkaline oral environment (Roberts and Manchester, 2010). For this reason it has been argued that an inverse relationship exists between dental caries, and calculus build-up. Whereas caries is the result of the net demineralization of tooth enamel as a result of an acidic oral environment, calculus formation is essentially a mineralization process (Duckworth and Huntington, 2005). In a study done by

Dahlén *et al.* (2010) on Thai adolescents this apparent inverse relationship could however not be confirmed as caries and calculus occurred concomitantly in the same individuals. It was found that a high calculus score only marginally reduced the individual likelihood of having caries. The co-occurrence of caries and calculus was ascribed to possible fluctuations in biochemical processes as no differences in salivary and plaque pH could be observed. The calculus groups however presented with significantly more bacteria in the saliva compared with the caries group. On this basis Dahlén *et al.* (2010) suggested that calculus is more closely related to poor oral hygiene.

The appearance of dental calculus amongst the Chinese may therefore be associated with their consumption of a carbohydrate rich diet (Roberts and Manchester, 2010). The sticky consistency created by the cooking techniques used to prepare especially rice would have increased the prevalence of plaque build-up (Tayles *et al.*, 2010). As has been shown by Dahlén *et al.* (2010) dental calculus is also closely related to poor oral hygiene, the presence of which may be further suggestive of socio-economic status. This may in turn be indicative of a lack of dental health care, as well as the consumption of cheaper cereal-based staple diets which would have increased the incidence of this condition amongst the Chinese (Larsen, 1997; Oliveira *et al.*, 2008). Tabacco smoking has also been shown to increase the rate of dental calculus formation even when oral hygiene is controlled for (Bergström, 2005). The Chinese miners were known to have smoked opium, however, the effects of opium smoking on oral hygiene and specifically the development of dental calculus has not yet been described in full and as such it was not dicussed here as a possible contributing factor in the presence of dental calculus.

When compared to the 32.2% observed for the Gladstone population, the Chinese miners presented with a frequency that is statistically significantly higher than the Gladstone population (χ^2=6.446; P-value < 0.05; P=0.0111). The Koffiefontein individuals did not present with any dental calculus or it was not scored for this population. This may suggest that the Chinese were consuming a more restricted diet consisting mainly of carbohydrates which is consistent with the traditional Chinese staple diet of white rice or wheat. Yet, when compared with frequencies of dental calculus obtained for prehistoric Chinese remains from the Longshan period (5000-4000 BP; also known to have consumed a cereal-based staple diet) the Chinese miners presented with a statistically significantly higher percentage of calculus (χ^2=59.236; P-value < 0.05; P=0.0001). This difference may be due to several factors. Sample bias may be evident in the Longshan remains as poor preservation hampered the scoring of dental calculus in some cases (Pechenkina *et al.*, 2002). If it is however

assumed that the frequency obtained for the Longshan population is a true representative of the actual incidence of calculus the differences seen may alternatively be attributed to differences in food texture as a result of food preparation techniques and/or a decline in oral health in the historic Chinese miners. Food with a high abrasive quality is more likely to remove plaque formation and can therefore to some extent decrease the degree of calculus formation (Turner, 1979; Roberts and Manchester, 2010). The prehistoric Longshan populations, even though dependent on cereal as a staple food, would have used processing methods that would transfer abrasive particles to food. Typically grains would have been ground on a stone mortar which would have incorporated tiny stone particles in the grounded grain (Roberts and Manchester, 2010). The grain and the foods produced from this would have been more abrasive as a result which in turn may have removed plaque before mineralization could occur. As has already been mentioned, oral hygiene plays an important role in the formation of dental calculus. A general high prevalence of dental calculus has been associated with many of the South East Asian populations, most of which have been attributed to poor oral hygiene (Chu *et al.*, 2008; Dahlén *et al.*, 2010). As political and economic conditions worsened in China by the late 19th century oral hygiene may very well have been neglected as famines and poverty would have been a more immediate concern.

- **Periodontal disease**

Periodontal disease was observed in 33.3% of the Chinese miners. This disease is closely associated with dental calculus as the build-up of calculus in the crevices between the tooth and soft tissue and bone of the jaw causes inflammation of the gums (gingivitis) (Roberts and Manchester, 1995; 2010; Hillson, 1998; Waldron, 2009). Continual inflammation can cause alveolar bone loss or the retraction of the alveolar bone from the tooth crown. Due to the high percentage dental calculus present in the Chinese miners the appearance of periodontal disease is to be expected. Periodontal disease may also be associated with scurvy as a result of haemorrhaging (Brickley and Ives, 2008). The lack of additional skeletal pathologies associated with scurvy however suggest that this condition can rather be ascribed to a diet rich in carbohydrates and poor oral hygiene which resulted in the build-up of dental calculus causing gingivitis and periodontitis.

Again, only the Gladstone population presented with periodontal disease. This condition was either absent or was not scored in the Koffiefontein individuals. The 41.1% observed on the Gladstone population was however not statistically significantly different to

the 33.3% observed in the Chinese miners (χ^2=0.527; P-value > 0.05; P=0.4680). This does not seem to correspond with the statistical difference seen in the frequencies of dental calculus. However, when considering that the Gladstone population suffered from scurvy, the higher frequencies of periodontal disease may be ascribed to haemorrhaging caused by the vitamin C deficiency rather than the degree of dental calculus (Van der Merwe *et al.*, 2010b-d). The Chinese, on the other hand, presented with a higher frequency of dental calculus, which would suggest that periodontal disease was caused by a build-up of calculus (possibly associated a carbohydrate-rich diet and poor oral hygiene) rather than haemorrhaging caused by scurvy.

5.2.4. Degenerative changes

Degenerative changes were observed in the form of so-called 'vertebral osteophytosis' (marginal osteophytes along the edges of vertebral endplates; 13.3% of the Chinese miners), seen especially in the regions of the lumbar vertebrae, and so-called 'peripheral osteoarthritis' (marginal osteophytic lipping along the edges of peripheral synovial joints) seen in the elbow (4.5%), knee (7.1%) and ankle (12%). The majority of the Chinese presenting with these conditions was estimated to be between 35-45 years at the time of their deaths. Many factors may influence the appearance of degenerative changes including age, sex, ancestry, obesity, trauma and even a genetic predisposition (Waldron, 2009; Waldron, 2012). Age is an important factor and both the incidence and prevalence increases with increasing age (Waldron, 2009). The occurrence of these degenerative changes in the older Chinese individuals may therefore be explained as a factor of older age. Movement, however, remains an important determining factor in the appearance of degenerative changes (Waldron, 2009; Waldron, 2012). Vertebral osteophytosis and peripheral osteoarthritis are commonly discussed as indicators of activity or mechanical function and lifestyle (Larsen, 1997; Jurmain *et al*., 2012). Some of the possible activity patterns associated with vertebral osteophytosis include physical activities that place strain on the spine, such as lifting or carrying heavy loads, whereas peripheral osteoarthritis of the calcaneo-talar joint may be the result of constant squatting or physical strain to this area (Larsen, 1997). Although not that often observed in the Chinese, the presence of osteophytic changes may therefore be associated with the strenuous physical working conditions presented by underground mining. Jurmain *et al*. (2012), however, cautions the use of

osteoarthritic changes in recreating activity and lifestyle patterns as there are no data to support the simplistic assumption that these changes directly derive from habitual activity.

Vertebral osteophytosis was observed in both the Gladstone (12.9%) and the Koffiefontein (22.2%) individuals. No statistically significant difference could however be seen between the frequency observed for the Chinese miners and the frequencies observed in the Gladstone and Koffiefontein populations (Gladstone: χ^2=0.404; P-value > 0.05; Fisher's exact test P=0.4577; Koffiefontein: χ^2=0.080; P-value > 0.05; Fisher's exact test P=1.000). All of these miners most probably suffered from back strain which is synonymous with physical labour activities associated with unskilled mining labour.

Peripheral osteophytosis was, however, more often observed in the Chinese miners than in any one of the other two mining populations. The Koffiefontein population did not present with any peripheral osteophytosis whereas the Gladstone population only presented with peripheral osteophytosis of the ankle. The 1% peripheral osteophytosis observed in the ankle joints of the Gladstone miners was statistically significantly lower than the 12% observed in the Chinese miners (χ^2=7.903; P-value<0.05; Fisher's exact test P=0.0245). This difference, even though statistically significant, is very small but may reflect different working conditions. Peripheral osteophytosis of the ankle more often occur when this area has suffered previous trauma (Waldron, 2012). The one case of peripheral osteophytosis seen in the Gladstone population and one of the three cases seen in the Chinese miners were observed in relatively young individuals (Gladstone: 20-25 years; Chinese: 20-34 years). The younger ages at onset may therefore suggest a cause related to reoccurring trauma rather than a factor of older age and repetitive movement. The Gladstone and Koffiefontein miners both worked in open groove mines and therefore mining was essentially done above ground. The Chinese miners, on the other hand, were specifically employed to do the deep level mining. Drilling and excavating underground, in confined tunnels, may have restricted their movement and vision which could have made them more susceptible to ankle injuries.

5.2.5. Specific infectious diseases

The presence of specific infectious diseases was observed in the form of possible osteomyelitis (7.2%) and possible treponemal disease (3.6%). Individuals A1022 (20–34 years) presented with destructive lesions to the right calcaneus whereas individual A1023 (20–34 years) presented with clear thickening of left distal femur. Osteomyelitis is a term used to describe any form of infection of bone and bone marrow which results in the

inflammatory destruction of bone (Waldron, 2009). The infection may result from either haematogenous spread, direct spread from and infection in the overlying or adjacent organs, or by direct implantation by penetrating injuries (Waldron, 2009). The cause of these infections in the Chinese miners is most probably due to a direct or haematogenous spread from soft tissue injuries as this condition is in both cases isolated and no signs of bone trauma could be observed.

The presence of osteomyelitis can to some extent be expected for a mining population, as such a working and living environment often gives rise to infections and trauma. Referring back to the medical records for Witwatersrand Deep Ltd., for the year 1905-1906, eleven Chinese were recorded to have died as a result of septicaemia. The large number of fatal injuries already attests to the hazardous working conditions and it can therefore be assumed that many non-fatal accidents must have occurred on a regular basis. Such injuries could have become infected, especially in conditions where the immune system was already impaired by other underlying conditions and vitamin deficiencies.

One individual (A1008; 35-45) presented with destructive lesions to the cranial vault, suggestive of treponemal disease (Fig. 4.10). This case could not definitively be diagnosed as treponemal disease, since the absence of any other associated pathology made this diagnosis problematic. However, these lesions look like possible caries sicca, caused by venereal syphilis (Waldron, 2009). Syphilis is often found in mining populations due to the limited access to women. Prostitution frequently occurred in and around mining compounds which increased the spread of treponemal diseases like syphilis. Historical and archival records also make mention of accounts of prostitution and 'unnatural vice' amongst Chinese miners and African and European women. Unnatural vice often referred to sexual relations between fellow Chinese, but also referred to the few cases of sexual relations with European prostitutes, which at that time was seen as an offence punishable by death (LTG 170, CON23B; Yap and Man, 1996; Harris, 1998). Complaints lodged by two Chinese inmates[1] also indicated that syphilis was often contracted in the jails, possibly as a result of sexual abuse (CD 157, AG664/08). Medical records for Witwatersrand Deep Ltd. (1905-1906)

[1]Chinese miners convicted of crimes such as desertion, theft and assault were placed in the local goals or jails for the period of the sentence (GOV 91, GEN 648/06; Yap and Man, 1996). With the repatriation of the Chinese from mid-1907 to 1910 those Chinese inmates who were still serving their sentences were released and sent back to China (Richardson, 1982; Yap and Man, 1996). A death penalty by hanging was given for more severe crimes like murder and sexual relations with European women (LTG 157, 140/35; LTG 157, 140/41; Yap and Man, 1996).

indicated that four Chinese died of syphilis and one of skull necrosis, possibly also related to this disease (FLD 90, 13/7; FLD 91. 13/7; FLD 127, 18/7; FLD 166, 32/7). It is therefore clear that this condition did occur amongst Chinese miners and could possibly therefore account for the presence of cranial vault lesions observed.

Only the Gladstone population presented with cases of osteomyelitis (0.9%) and treponemal disease (8.4%). No statistically significant difference could be observed between the Chinese miners' frequencies and those observed for the Gladstone individuals (Osteomyelitis: χ^2=0.758; P-value > 0.05; Fisher's exact test P=0.6871; Treponemal disease: χ^2=3.937; P-value > 0.05; Fisher's exact test P=0.1090). This may be expected since both population groups were subjected to similar conditions which would result in the appearance of such pathologies.

No pathology indicative of tuberculosis could be observed on any of the Chinese miners which is somewhat surprising when considering the high infection rate of this disease, especially in South African mines (Roberts and Ives, 2003; Steyn *et al.*, 2013). Archival records do indicate the presence of tuberculosis amongst some of the Chinese (health records indicating cause of death; FLD 240, 76/7); however, none of these cases were reported for the Witwatersrand Deep mine where this study's individuals came from. The poor preservation of some of the more fragile bones like the ribs may have hampered the identification of less severe pathology associated with tuberculosis.

5.2.6. Trauma

- **Ante-mortem trauma**

Ante-mortem healed fractures were observed in three individuals: one presenting with a partially healed fracture of the right fibula, involving the shaft and distal epiphysis; one presenting with a possible healed crush fracture of the foot, involving the first metatarsal and associated tarsal; and a third with a partially healed depressed fracture of the left parietal bone.

Differences in the frequency of ante-mortem trauma could be observed for the Chinese miners and the Gladstone population. Ante-mortem trauma was observed in 34% of the individuals from Gladstone which was significantly higher than the 10% observed in the Chinese miners (χ^2=6.391, P-value < 0.05; P=0.0115). This may be due to the fact that deep level mining presented a much more hazardous working environment and that injuries sustained in such working conditions were more often fatal. This may to some extent explain

why very few ante-mortem fractures were observed in the Chinese miners as opposed to the Gladstone individuals. Another possible reason for this discrepancy could be that the Chinese miners were only employed for a relatively short time on the Witwatersrand mines (contract of three to five years between 1904 and 1910) whereas African labourers were generally employed for much longer time periods and therefore subject to more working hours and possible more work-related accidents. The fact that the Chinese were hired on a contract basis also meant that once they got injured to such an extent that they could no longer fulfil their contract agreement they would have been sent home. Historical records indicate that many Chinese were repatriated because of sustained injuries which made them unable to continue work. They were paid a once-off compensation salary and then put on the first ship going back to China (FLD 240, 76/3; Richardson, 1982; Yap and Man, 1996). Due to the significant degree of healing in most of the ante-mortem cases it may even be considered that these injuries were already sustained before their arrival and employment on the Witwatersrand mines.

Most of the ante-mortem trauma seen in the Gladstone individuals was restricted to the cranium. Even though one case of cranial trauma was observed in the Chinese miners, this was still significantly lower when compared to the 18% observed in the Gladstone miners (Van der Merwe, 2007). Cranial trauma is often associated with a blow to the head. Such an injury could easily have been sustained during a rock fall or cave-in, especially in the case of the Chinese miners who worked underground. Cranial trauma is also sometimes interpreted as evidence of interpersonal violence (Walker, 2001). In the Gladstone individuals, the large proportion of cranial ante-mortem trauma was also interpreted as signs of interpersonal violence (Van der Merwe *et al.*, 2010a). Compound settings often gave rise to interpersonal violence as tension rose in the confined spaces that these compounds created (Richardson, 1982; Kynoch, 2005; Van der Merwe *et al*., 2010). Historical reports also make mention of the violent circumstances Chinese labourers were subjected to, suggesting that fights often broke out as a result of abuse and assault from supervisors or disagreements between fellow Chinese labourers (Kynoch, 2005). Yet, the Gladstone miners were subjected to a lot more interpersonal violence.

A possible explanation for this is that the Gladstone mine dates to a period before the importation of Chinese labour. During negotiations between China and the Chamber of Mines it was decided that mines should be upgraded before the Chinese labourers arrived. This especially included renovations to the living quarters in the mine compounds. More space had to be made available in the sleeping quarters as well as upgrades to the ablution

facilities as both these were regarded as being inadequate (Richardson, 1982; Yap and Man, 1996). The Gladstone population would therefore still have been subjected to the old compounds which presented with very confined living quarters and poor sanitary facilities. These aggravating conditions may therefore account for the high percentage of cranial ante-mortem trauma observed in the Gladstone population.

Ante-mortem trauma observed in modern populations like the Venda (L'Abbè and Steyn, 2007) and modern South African black and white populations (Steyn *et al.*, 2010) indicate a significant degree of variability in the incidence of ante-mortem trauma. For instance only 7% of the modern Venda presented with ante-mortem trauma whereas the modern South African black and white populations presented with significantly higher frequencies of ante-mortem trauma (black population: 73%; white population: 51%). The very high frequency of ante-mortem trauma observed in the South African black population was mainly attributed to interpersonal violence as a direct effect of their socio-economic status, whereas the relatively high frequency observed in the South African white population could be explained as a factor of older age (osteoporosis) and interpersonal violence, again as a result of their socio-economic backgrounds (Steyn *et al.*, 2010). It therefore becomes clear that extrinsic factors play an important role in the incidence of something like ante-mortem trauma. When considering the Chinese miners, who also presented with a very low percentage of ante-mortem trauma (10%), the effects of such social and economic factors should be taken into account. Even though they came from poverty-stricken areas of China, social factors (family bonds) may have prevented them from being affected by frequent acts of interpersonal violence. Once employed on the mines this may have changed as indicated by the single case of ante-mortem cranial trauma observed in one of the Chinese miners (A1030). Archival and historical accounts indicate that tension rose in the confined spaces created by compounds and was worsened by abuse from mining officials and the Chinese police boys employed to exercise control over the Chinese miners (FLD 51, 6/7; FLD 224, 62/7; GOV 79, GEN 56A/05; Yap and Man, 1996; Kynoch, 2005). Gambling was also a major contributing factor in creating tension amongst fellow Chinese, often leading to violence and even homicide (LTG 157, 140/35; LD 1323, AG 4040/06; LTG 157, 140/41; Yap and Man, 1996).

None of the Koffiefontein individuals presented with ante-mortem or peri-mortem fractures associated with traumatic events. Archival research done by L'Abbè et al. (2003) indicated that many of the deaths occurring at Koffiefontein were actually attributed to a typhoid fever/enteric fever outbreak. The presence of skeletal pathology associated with

chronic conditions of malnutrition (cribra orbitalia, periostitis and enamel hypoplasia) also meant that these individuals were more susceptible to infectious diseases in general (L'Abbè et al., 2003). The cause of death in the Koffiefontein individuals was therefore most probably attributable to poor nutrition and infectious disease and therefore we would not expect to see any peri-mortem trauma. The lack of ante-mortem trauma, on the other hand, may suggest that the working and living conditions were better than those experienced by the Chinese and Gladstone miners.

- **Spondylolysis and Schmorl's nodes**

Spondylolysis (12.5%; 2 individuals) and Schmorl's nodes (18.8%; 3 individuals) were observed in the thoracic and lumbar vertebrae. The aetiology of spondylolysis is twofold and it is argued that this condition can either be caused by a congenital anomaly (Aufderheide and Rodríguez-Martin, 1998; Haun and Kettner, 2005; Ward and Latimer, 2005; Ward *et al.*, 2007) or as a result of stress (fatigue) fractures cause by hyperflexion of the lumbar vertebrae (Merbs, 1996; Aufderheide and Rodríguez-Martin, 1998; Ortner, 2003; Waldron, 2009). The former has recently been argued by Ward and Latimer (2005) and Ward *et al.* (2007) suggesting that spondylolytic fractures, especially of L5, is the result of an inadequate increase in interfacet distances between the adjacent vertebrae. It has further been argued that this insufficient increase may have a genetic component, as interfacet distances are not altered throughout life (Ward and Latimer, 2005; Ward *et al.*, 2007).

Any one of these aetiologies may be considered when looking at the presence of spondylolysis in the Chinese miners. The first suggests that this type of pathology is associated with stress or fatigue fractures of the spine as a result of hyperflexion of the lumbar vertebrae (Merbs, 1996; Aufderheide and Rodríguez-Martin, 1998; Ortner, 2003; Waldron, 2009). This may be a very plausible cause for the spondylolysis seen in the Chinese individuals as it is known that these people engaged in very strenuous work during their time on the mines. In order for the mines to make a profit after the incurred expenses they moved the Chinese to areas where they would be most strategically productive (Yap and Man, 1996). This meant that they were mainly employed for underground work whereas the African labourers were employed above ground. This ensured that the necessary work underground could be done without increasing the wages for African labourers. Underground work was much more labour-intensive and was made worse when mechanised means of mining were substituted for hand drilling, extraction and sorting (Yap and Man, 1996). Factors like these

would have placed additional strain on their bodies which may in turn have resulted in typical fatigue fractures like spondylolysis.

Additionally, it may also be considered that the spondylolysis observed in the Chinese is the result of an underlying genetic predisposition (Ward and Latimer, 2005; Ward *et al.*, 2007). Spondylolysis is often associated with developmental defects such as spina bifida and notochord defects which in turn may be related to prenatal deficiencies of folic acid, zinc and B vitamins (Aufderheide and Rodríguez-Martin, 1998; Sairyo *et al.*, 2006). It has been shown that Asian population groups present with somewhat higher frequencies of spondylolysis in general (Merbs, 1989; Merbs, 2002). It may therefore be possible that the presence of spondylolysis in the Chinese also had a genetic and even metabolic aetiology. When considering their socio-economic status this may be a reflection of the overall poor diet and health amongst the peasant societies in China at that time. These people would have come from families of similar socio-economic backgrounds which indicate that aspects such as malnutrition and disease could have been transferred from one generation to the next.

Schmorl's nodes may also occur as a result of alternative causes, other than stress-related trauma. It has been suggested that metabolic diseases, genetic developmental defects and/or a genetic predisposition towards developing the condition may also be considered as possible aetiologies for this condition (Aufderheide and Rodríguez-Martin, 1998; Barnes, 1994; Ortner, 2003; Haun and Kettner, 2005; Ward and Latimer, 2005; Ward *et al*., 2007; Williams *et al*., 2007; Brickley and Ives, 2008; Waldron, 2009).

Schmorl's nodes form when the vertebral endplate decompresses, the adjacent nucleus resulting in a herniation (Williams *et al*., 2007; Waldron, 2009). The herniated tissue then forms a dent on the upper and lower involved vertebrae (Williams *et al*., 2007). These lesions predominate in the region of T10-L1 suggesting that this area is more susceptible to stress (Williams, *et al*., 2007). This then supports the view that continuous trauma weakens cartilaginous endplates and eventually results in herniation and the formation of Schmorl's nodes. Yet, cartilaginous weakening through the mechanism of repeated trauma is not a prerequisite for the formation of the condition. It is argued that alterations to subchondral bone of the vertebrae, either due to developmental defects or a systematic process such as osteopenia, underlie the formation of Schmorl's nodes (Williams *et al*., 2007). In the same sense Schmorl's nodes are often associated with Scheurmann's disease (Aufderheide and Rodríguez-Martin, 1998; Ortner, 2003; Williams *et al*., 2007; Waldron, 2009) which has a strong genetic aetiology. This condition often occurs in combination with juvenile osteoporosis, and/or activity-related traumatic influences (Jurmain *et al*., 2012).

Schmorl's nodes have also been associated with degenerative disc disease such as ageing-related osteoporosis (Aufderheide and Rodríguez-Martin, 1998; Williams *et al*., 2007; Brickley and Ives, 2008; Waldron, 2009). However, new research has shown that disc degeneration can be separated into two categories namely 'endplate-driven' and 'annulus-driven' disc degeneration and that the latter, which is associated with older age, does not commonly present with Schmorl's nodes (Adams and Dolan, 2012). Endplate-driven disc degeneration involves endplate defects and the inward collapse of the annulus which mostly affects the upper lumbar and thoracic spine. This phenotype has been shown to have a high heritability and often starts before the age of 30 years (Williams *et al.*, 2007; Adams and Dolan, 2012). Annulus-driven disc degeneration arises from radial fissures and/or a disc prolapse and mostly affects L4-5 and L5-S1. This category does not have a high heritability and develops progressively after the age of 30 years as a result of repetitive bending and lifting (Adams and Dolan, 2012). Schmorl's nodes are relatively rare in the lower lumbar spine and it is suggested that where it is present it is only weakly associated with disc degeneration (Adams and Dolan, 2012).

When considering this, the presence of Schmorl's nodes observed in the Chinese miners can be attributed to both traumatic and genetic factors. The presence of Schmorl's nodes in the lower thoracic and upper lumbar vertebrae of the Chinese, as well as their relative young ages at death (all three individuals were estimated to be between 20-34 years), suggests that they suffered from endplate-driven disc degeneration. As has been shown this condition can be attributed to a genetic predisposition for developing the conditions, which may invariably be worsened in cases where there is an insufficient intake of calcium (for example juvenile osteoporosis, osteopenia). It may therefore be that the Chinese were genetically inclined to developing the condition. As has already been mentioned, their traditional diet also contained very little animal-derived products which meant that calcium intake would have been limited (Chen and Xu, 1996; Popkin *et al.*, 2001; Du *et al.*, 2002; Fraser, 2004). This in turn may have slightly increased the incidence of this condition. This condition is also caused by stress-related trauma, possibly due to the strenuous work associated with menial mining labour. Schmorl's nodes are sometimes also associated with compressive injuries caused by a fall on the buttocks (Adams and Dolan, 2012), an injury which might have occurred often in deep level mining.

The Gladstone population was the only one of the two comparable populations that presented individuals with spondylolysis (8.2%) and its incidence was not statistically significantly different to the 12.5% observed in the Chinese miners (χ^2=0.3934; P-value

>0.05; Fisher's exact test P=0.729). The Gladstone and Koffiefontein remains both presented with Schmorl's nodes (31% and 13.9% respectively) (L'Abbè *et al.,* 2003; Van der Merwe, 2007). No statistically significant difference could be observed between these incidences and the 18.8% observed for the Chinese miners (Gladstone: χ^2=0.362; P-value > 0.05; Fisher's exact test P=0.7577; Koffiefontein: χ^2=0.201; P-value > 0.05; Fisher's exact test P=0.6894). No differences in the frequencies of spondylolysis or Schmorl's nodes may to some extent be expected as all three populations are known to have come from low income societies, suffered from poor nutrition, and had to engage in physical labour from a young age.

A study by Steyn *et al.* (2010) indicated the frequency of spondylolysis in modern cadaveric skeletal remains for Greek and South African black and white populations. This study showed an extremely small incidence rate with only 0.43% of the Greek and 0.2% of the South African white population presenting with spondylolysis. The South African black population did not present with any spondylolysis. This condition therefore does not seem to be a frequent occurrence in normal populations and may therefore be reflective of the very specific work related stresses associated with mining.

- **Peri-mortem trauma**

Peri-mortem trauma was present in nearly a third of individuals and was mainly observed in the extremities, especially in the lower limbs. However, poor preservation and incomplete skeletal elements of more fragile skeletal remains such as the ribs, hand and foot bones, and to some extent of vertebrae and cranial remains meant that a complete assessment of injuries sustained due to traumatic events could not be done. The trauma observed here may therefore only be representative of the minimum amount of the total trauma sustained.

Peri-mortem fractures observed in the thoracic and lumbar vertebrae and pelvis were mainly due to compression/crush forces as defined by Lovell (1997) and Ortner (2003). Compression fractures are usually the result of a sudden excessive (axial) impaction and result in a variety of fracture patterns.

The most common injuries associated with thoracic and lumbar fractures are caused by excessive flexion. Flexion usually occurs when a person falls from a height into a sitting or hunched position, when a weight falls onto the hunched back of a person, or when a person is struck from behind by a moving object (Galloway, 1999; Waldron, 2009). Pelvic fractures are also often associated with falls from heights, especially in cases where the person lands on the buttocks or on his/her side (Galloway, 1999). These types of fractures are generally associated with a high mortality rate especially in cases where the internal soft tissue

structures and organs have been perforated causing haemorrhage and excessive blood loss (Galloway, 1999).

Most of the peri-mortem trauma could however be observed in the extremities, especially in the femur and tibia shafts. The majority of these fractures were classified as butterfly fractures followed by a couple of spiral fractures. Butterfly fractures are comminuted fractures that form as a result of a combination of compression and tensions forces (Lovell, 1997). This type of fracture can be characterised by the separation of a triangular fragment of bone. Spiral fractures, on the other hand, are linear fractures occurring as a result of rotational and downward loading stress on the longitudinal axis (Lovell, 1997). Femoral and tibial shaft fractures are at a higher level of incidence amongst younger adults, particularly males between the ages of 20–30 years (Galloway, 1999). This occurrence may be attributed to the participation of activities in which high-energy injuries are often found (Galloway, 1999). Lower limb injuries are also often associated with falls from heights, especially in the cases where landing is primarily focussed on the lower limbs (Galloway, 1999). Shaft fractures constitute major trauma and may result in death, often as a result of excessive blood loss (Galloway, 1999; Waldron, 2009).

Historical records indicate that the majority of the work-related deaths were caused by falls from heights (down mine shafts and falls from skips transporting miners up and down the shafts) as well as cave-ins and rock falls (mainly due to accidental detonation of explosives). All of these accidents could therefore have contributed to the types of fractures seen here. Injuries sustained through such accidents would have caused multiple injuries resulting in major trauma. This is evident in the large proportion of Chinese miners presenting with multiple fractures. Of the 30% of Chinese presenting with peri-mortem trauma, 26.6% presented with multiple fractures. None of the fractures mentioned here showed any macroscopic signs of healing and it can therefore be assumed that they were sustained around the time of death and may therefore have contributed to the death of these individuals.

Indeed, the historical records clearly indicate that these indentured labourers were often unfamiliar with the workings of underground mines and as a result sustained many injuries either due to falls or cave-ins. This was especially the case with blasting and drilling activities as many of the Chinese labourers were not properly trained in the use of explosives (Richardson, 1982, Yap and Man, 1996; Harris, 1998). Furthermore, due to the fact that underground mining was a relatively new method of mining many of the safety measures associated with modern-day underground mining were not yet in place. Historical records

indicate that many of the work related deaths were caused by inhalation of toxic gasses, electric shocks, and falls from skips whilst transporting workers up and down the mine shafts, as well as falls down the mine shafts itself (FLD 90, 13/7; FLD 91. 13/7; FLD 127, 18/7; FLD 166, 32/7). It also seems that many of the Chinese that were employed to do the underground mining were not fully aware of the circumstances surrounding their employment. This can be corroborated by the fact that so many complaints were lodged by Chinese miners once they were employed on the Witwatersrand, stating that they were not made aware of the fact that they had to work underground (especially deep underground) as mining in China was mainly done above ground or at very shallow depths (FLD 240, 76/7). This is possibly due to the fact that none of the Transvaal emigration agents could speak the northern dialects and as a result applicants were not fully briefed in what was expected of them before they were employed in South Africa (Yap and Man, 1996).

In the Gladstone population (n=107), only 4.9% of individuals presented with peri-mortem fractures. This is statistically significantly lower than the 30% observed in the Chinese individuals (χ^2=11.481, P-value <0.001, P=0.0007; Fisher's exact test: P=0.0021). This difference may be attributed to the fact that the Chinese miners were specifically employed for underground mining whereas the Gladstone and Koffiefontein miners would have mined above ground (open groove mines associated with diamond mining in Kimberley). Deep level mining has always presented with additional hazards such as cave-ins, rock falls and falls down mine shafts (Richardson, 1982). When considering the fracture patterns observed in the Chinese and the Gladstone miners respectively, these differences become apparent. The Chinese mostly presented with multiple fractures located in the lower extremities and the back. The Gladstone population rarely presented with multiple fractures. The one case where multiple fractures were observed in one of the Gladstone individuals there were no signs of healing which suggest that this individual died as a result of his injuries (Van der Merwe, 2007). Fractures were also largely limited to the cranium in the Gladstone population (18%), most of these showing signs of healing indicating that this was not the cause of death (Van der Merwe *et al.*, 2010a). Even with femoral fractures, which was the second most frequent fracture observed in the Gladstone miners (2.8%) and other long bone fractures, the individuals still lived long enough for them to have receive surgical intervention in the form of amputations (5.6%; Van der Merwe, 2007; Van der Merwe *et al.*, 2010a). It therefore seems that the high occurrence of multiple fractures in the Chinese miners as well as the lack of any surgical intervention and healing of fractures attest to the higher accident and mortality rate associated with deep level mines.

The general fracture pattern observed in the Chinese miners seems to suggest unique working conditions and work-related injuries associated with deep level mining. This becomes evident when the frequency and occurrence of fractures are compared to Asian populations that did not engage in mining. Fracture patterns observed in prehistoric northeast and southeast Thailand and two prehistoric Pacific Island populations (Papua New Guinea and the Solomon Islands) presented with very low percentages of long bone fractures, especially of the femur and tibia (northeast Thailand: 2% femur; 0% tibia; southeast Thailand: 0% femur; 0% tibia; Papua New Guinea: 0.5% femur and tibia; Solomon Islands: 0.1% femur and tibia; Domett and Tayles, 2006; Scott and Buckley, 2010). Long bone fractures observed were healed and were attributed to daily activities associated with agriculture (Domett and Tayles, 2006; Scott and Buckley, 2010).

Both the Thailand and the Pacific Island populations presented with cranial trauma. The latter of the population groups presented with a very high percentage of cranial trauma as well as sharp force trauma (Papua New Guinea: 21.4%; Solomon Islands: 4.5%; Scott and Buckley, 2010). Both the cranial trauma and the sharp force trauma were attributed to interpersonal violence as a shift in agricultural practices and land shortages lead to disputes amongst clans. It therefore seems that the majority of trauma observed in these two Asian populations, not engaging in mining, was attributed to interpersonal violence rather than work or lifestyle related injuries. This stands in direct contrast to the relatively large percentage of peri-mortem (work related) trauma (30%), especially of the long bones, observed in the Chinese miners.

5.3. Signs of metabolic and non-specific bone disease as observed in cadaver skeletal remains

In order to better interpret pathological signs of metabolic bone disease and malnutrition observed in the Chinese miners, a sample of 100 individuals from the Raymond A Dart Collection (Dayal *et al.*, 2009) as well as the Pretoria Bone Collection (L'Abbe *et al.*, 2005), were assessed for macroscopic signs of pathology. These cadaveric skeletal remains were selected on the basis of their cause of death. Individuals for whom the cause of death was given as malnutrition, specific nutritional deficiencies or alcoholism were selected. Each skeleton was assessed for macroscopic signs of pathology specifically associated with

metabolic disease and malnutrition and then compared to the results obtained for the Chinese miners (Refer to Table 4.7 for a summary of these results).

5.3.1. Porotic hyperostosis

The highest frequency of porotic hyperostosis (83.9%) was observed in individuals for whom cause of death was given as liver cirrhosis or alcoholism. This was followed by 72.7% observed in the individuals who died of pellagra and then 55.8% who died of general malnutrition. When these frequencies are compared to the 78.3% observed in the Chinese miners no statistical difference could be observed between the frequency of porotic hyperostosis observed in the Chinese and either of these three groups (Liver cirrhosis/alcoholism: χ^2=0.275; P-value > 0.05; Fisher's exact test P=0.5997; Pellagra: χ^2=0.127; P-value > 0.05; Fisher's exact test P=1.000; General malnutrition: χ^2=3.448; P-value > 0.05; P=0.0633).

Frequencies of porotic hyperostosis observed in the individuals who died from liver cirrhosis/alcoholism and pellagra are the closest to that observed for the Chinese miners. This apparent association may be as a result of similar underlying vitamin and mineral deficiencies. Patients suffering from liver cirrhosis/alcoholism have been shown to suffer from several vitamin and mineral deficiencies including B-vitamins (B1 (thiamine), B2 (riboflavin), B3 (niacin), B6 (pyridoxine), and B12 (cobalamin); folates (specifically folic acid); iron; zinc; calcium; and vitamin D (Ishii and Nishihara, 1981; Mobarhan *et al*., 1984; Rodriguez-Moreno *et al.*, 1997; Ball, 1998; Truswell, 2002; Groenen *et al.*, 2004a; Groenen *et al.*, 2004b; Suskind, 2009). One of the conditions also readily associated with chronic alcoholism is vitamin B1 deficiency or beriberi (Carpenter, 2000; Truswell, 2002).

Pellagra is also closely associated with vitamin B deficiencies and is specifically attributed to a vitamin B3 or niacin deficiency (Ball, 1998; Truswell and Milne, 2002; Brenton and Paine, 2007; Suskind, 2009). It may also indirectly be associated with iron deficiency. In order for niacin to be absorbed effectively it has to be consumed with tryptophan which makes it biologically available for absorption (Ball, 1998; Truswell and Milne, 2002; Brenton and Paine, 2007). Tryptophan is contained in most proteins and the lower the levels of niacin, the higher the demand for more tryptophan becomes which in turn means that the body will have to have access to more iron (Brenton and Paine, 2007). This may therefore lead to an iron deficiency. This may be exacerbated by the fact that deficiencies in niacin are most commonly observed in poorer communities which subsist

mainly on maize and do not have access to more expensive protein-based foods (Brenton and Paine, 2007; Kirkland, 2007).

It is therefore clear that individuals suffering from both alcoholism and pellagra would have been deficient in B vitamins and iron. As has already been mentioned these deficiencies often lead to the appearance of hypertrophic bone lesions (Brenton and Paine, 2007; Walker *et al.*, 2009; Oxenham and Cavill, 2010) and would therefore account for the high frequencies of porotic hyperostosis observed in these individuals.

Historical accounts suggested that the Chinese too were deficient in B vitamins, specifically vitamin B1, resulting in beriberi, as well as iron as a result of traditional diets containing very little protein based foodstuffs (Richardson, 1982; Yap and Man, 1996; Carpenter, 2000). It therefore seems likely that the Chinese miners would have presented with similar skeletal pathology and it would explain why the frequency of porotic hyperostosis observed in the Chinese miners were the closest to the frequencies observed for individuals who died as a result of liver cirrhosis/alcoholism and pellagra. The one modern case of beriberi and the one modern case of anaemia also presented with porotic hyperostosis, which may indicate that this condition can be associated with beriberi and anaemia. However, it should be kept in mind that these two conditions may occur simultaneously, which makes it difficult to pinpoint the actual vitamin deficiency responsible for the pathology.

Even though the frequency of porotic hyperostosis observed in the individuals who died of general malnutrition and the frequency observed in the Chinese miners showed no statistical difference, there still seems to be less of an association between the two (P=0.0633). This may be due to the fact that individuals for whom the cause of death was given as general malnutrition obviously presented with more than one vitamin deficiency. However, the overlapping responses of bone to metabolic insults, like vitamin deficiencies, make it difficult to distinguish between different aetiologies. This is substantiated by the fact that other metabolic bone diseases, like scurvy and vitamin D deficiency, can also cause the appearance of porotic lesions (Brickley and Ives, 2008). This may be substantiated by the one case of scurvy which also presented with porotic hyperostosis.

In summary, the frequency of porotic hyperostosis observed in the Chinese miners showed the closest association with the cadaver individuals for whom cause of death was given as liver cirrhosis/alcoholism and pellagra. This association is most probably due to the same underlying deficiency in specifically B vitamins and iron. Less of an association was found for the Chinese miners and the general malnutrition group which suggests that without more specific information on the cause of death and the presence of underlying conditions

prior to death a specific aetiology for the occurrence of porotic hyperostosis cannot be confirmed.

5.3.2. Cribra orbitalia

Among the cadaver samples with specific causes of death cribra orbitalia was most often observed in individuals who died as a result of pellagra (72.7%) followed by 57.7% observed in general malnutrition cases and 51.5% in liver cirrhosis/alcoholism. Cribra orbitalia was also noted in the one case of scurvy and the one case of anaemia. Comparisons between results obtained for the Chinese individuals (34.6%) and the cadaveric remains suggested that there is no statistically significant difference between the frequencies obtained for the Chinese and those individuals who died as a result of liver cirrhosis/alcoholism (χ^2=1.685; P-value > 0.05; P=0.1943) and general malnutrition (χ^2=3.692; P-value > 0.05; P=0.0547). Yet, when compared with the individuals who succumbed to pellagra there is a statistically significant difference (χ^2=6.894; P value < 0.05; P=0.0086).

The closest association in frequencies of cribra orbitalia could be seen for the liver cirrhosis/alcoholism individuals and the Chinese miners. This association may be ascribed to the fact that alcoholism is so closely associated with beriberi or vitamin B1 deficiency. As has been mentioned before the Chinese miners were known to have suffered from beriberi as a result of their thiamine deficient diets. Similar osteological responses would therefore be expected. The one case of beriberi observed in the cadaver remains did however not present with cribra orbitalia. This may indicate that cribra orbitalia is rather a result of anaemia than vitamin B1 deficiency; however, it is difficult to base such an assumption on the assessment of only one individual.

The frequency of cribra orbitalia observed in the individuals who died of general malnutrition was found to be not statistically different to that observed for the Chinese miners. Even though the frequency of cribra orbitalia observed in the Chinese miners is comparable to the frequency obtained for the general malnutrition cases, there is very little association between the two as indicated by the relatively small P-value obtained (P=0.0546). This may again be attributed to the generalised cause of death given for cadaveric remains in this group, as well as the problem with overlapping responses of bone to different metabolic insults, making it impossible to distinguish between different aetiologies.

The pellagra individuals presented with significantly higher frequencies of cribra orbitalia than the Chinese group. This may suggest that this condition is more often

observable in a vitamin B3 deficiency. In a study of the impact of pellagra on human skeletal remains Brenton and Paine (2000; 2006; 2007) identified cribra orbitalia as one of the skeletal pathologies often observed in pellagrins. No difference in the incidence of cribra orbitalia was, however, found between pellagrins and other cases of mineral and vitamin deficiencies. This was attributed to the fact that pellagra, often seen in maize dependent populations, would generally co-exist with other deficiencies, especially vitamin C and iron, both of which have been associated with cribra orbitalia. Vitamin C and iron deficiency should, therefore, be considered as alternative causes for the higher frequencies of cribra orbitalia seen in the in this study.

5.3.3. Periostitis

The highest frequency of periostitis was observed in the individuals for whom cause of death was given as liver cirrhosis/alcoholism (tibia: 91.4%; other: 55.6%). This was followed by those who died as a result of pellagra (tibia: 70%; other: 63.6%) and general malnutrition (tibia: 53.8%; other: 44.2%). When compared to the frequencies observed for the Chinese miners (tibia: 38.2%; other: 27.3%) the individuals who died from liver cirrhosis/alcoholism presented with a statistically higher frequency of periostitis (tibia: χ^2=21.512; P-value < 0.05; P=0.0001; other: χ^2=5.653; P-value < 0.05; P=0.0174). No statistical difference could, however, be seen between the frequencies of periostitis observed in the pellagra (tibia: χ^2=3.145; P-value > 0.05; Fisher's exact test P=0.1466; other: χ^2=4.714; P-value > 0.05; Fisher's exact test P=0.0668) and general malnutrition individuals (tibia: χ^2=2.008; P-value > 0.05; P=0.1564; other: χ^2=2.473; P-value > 0.05; P=0.1158) and the Chinese miners.

The higher frequency of periostitis observed in the liver cirrhosis/alcoholism individuals may be ascribed to factors associated with lifestyle. It has been noted that periostitis of especially the tibiae and fibulae are commonly associated with liver cirrhosis and chronic alcoholism (Epstein *et al.*, 1979; 1981; Simpson and Finlayson, 1995). A condition known as hepatic hypertrophic osteoarthropathy has been identified in cases of chronic liver disease (Epstein *et al.*, 1979; 1981; Pitt *et al.*, 1994). This condition is characterised by bilateral periostitis in the shafts of long bones (tibiae, fibulae, radii and ulnae) and in severe cases clubbing of the fingers and toes (only affects soft tissue and would therefore not be observable in skeletal remains) (Pitt *et al.*, 1994; Ortner, 2003). In most of the cases of periostitis observable in the liver cirrhosis/alcoholism group the involvement was

bilateral and not just limited to the tibiae. This may suggest that that the higher frequencies of periostitis observed in the liver cirrhosis/alcoholism group may to some extent be associated with hepatic hypertrophic osteoarthropathy. The one case of beriberi also presented with periostitis. This may either be representative of the smaller frequency of periostitis observed in dietary beriberi or, should this case represent alcoholic derived beriberi, this may fit into the general association of periostitis with liver cirrhosis/alcoholism.

Periostitis is regarded as a non-specific indicator of disease and stress as diagnosis of this condition is often impossible, especially in non-clinical cases (Ortner, 2003). It may be associated with specific infectious diseases, such as tuberculosis and treponemal disease, localised trauma and infection (osteomyelitis), and nutritionally related diseases like scurvy and rickets (Ortner, 2003; Brickley and Ives, 2008; Waldron, 2009). Yet, it is also classified as a disease on its own, specifically in cases where other more diagnostic pathologies are not found in association, making a specific diagnosis problematic (Ortner, 2003). The high percentage of periostitis in the liver cirrhosis/alcoholism group again reflects this apparent non-specific nature of periostitis.

5.3.4. Medio-lateral bowing

Medio-lateral bowing was more frequently observed in the individuals who died from pellagra (20%) as opposed to those with liver cirrhosis/alcoholism (11.4%) and/or general malnutrition (3.8%). When compared to the 25% observed in the Chinese miners no statistical difference was obtained for the individuals who died of liver cirrhosis/alcoholism (χ^2=1.988; P-value > 0.05; Fisher's exact test P=0.1935) or pellagra (χ^2=0.102; Fisher's exact test P-value > 0.05; P=1.000). The frequencies obtained for bowing deformities in liver cirrhosis/alcoholism and pellagra individuals and the frequency obtained for the Chinese miners are therefore similar. In all three cases bowing deformities may be associated with residual rickets (prevailing effects of childhood rickets) caused by a vitamin D and/or calcium deficiency. It has already been mentioned that the Chinese miners consumed traditional grain base diets which contained very little vitamin D and/or calcium, which would therefore explain the presence of this condition amongst them. The one case of beriberi also presented with minor bowing deformities of the tibia and fibula which again may indicate a possible relationship.

Similarly, individuals suffering from chronic alcoholism are known to have diets that are nutritionally severely deficient, mostly because it supresses appetite (Truswell, 2002).

Calcium and vitamin D deficiencies, both of which may be associated with the deformation of long bones, may therefore be present in chronic alcoholics. Furthermore, kidney and liver function becomes impaired in cases of liver cirrhosis/alcoholism which will further inhibit vitamin D and calcium metabolism (Brickley and Ives, 2008; Arteh *et al.*, 2010). For this reason a symptom of excessive ethanol consumption has been associated with the manifestation of osteoporosis and osteomalacia (Chakkalakal, 2005).

When considering the presence of bowing deformities in the pellagra individuals, additional factors of diet and metabolism may contribute to the presence of bowing deformities rather than the niacin deficiency itself. Pellagra is more often seen in populations consuming an exclusively maize dependent diet (Brenton and Paine, 2007; Kirkland, 2007). These diets are usually devoid of especially animal proteins and therefore contain insufficient amounts of calcium and vitamin D. The former has also been described by Pettifor (2004) as a major cause of rickets in South African populations. Traditional practices such as the swaddling of African babies may further increase the incidence of bowing deformities (Holick, 2007; Brickley and Ives, 2008). These conditions may therefore explain why this condition was observed in the cadaveric remains.

When compared to the general malnutrition individuals the Chinese miners presented with a significantly higher frequency of bowing deformities (χ^2=8.157; P-value < 0.05; Fisher's exact test P=0.0075). This may again be ascribed to the fact that more specific medical information pertaining to the cause of death is not available. It may however be assumed that significantly less individuals, for whom cause of death was given as general malnutrition, suffered from vitamin D and/or calcium deficiencies and that they therefore must have suffered from other types of vitamin and/or mineral deficiencies.

5.3.5. Developmental defects

Even though many of the developmental defects occurring in the skeleton stem from a congenital predisposition (Ortner, 2003) it has been argued that some developmental defects such as spina bifida occulta and notochord defects may be representative of a poor maternal health during pregnancy. The presence of developmental defects may therefore provide insight into the previous generation's health. If present it may be representative of a long-standing nutritional problem which would have been transferred to the next generation, possibly placing them at a disadvantage.

Vertebral developmental defects were noted in individuals who died of liver cirrhosis/alcoholism (22.2%), pellagra (18.2%), as well as general malnutrition (7.7%). When compared to the 37.5% observed in the Chinese miners the liver cirrhosis/alcoholism and pellagra individuals showed no statistically significant difference in the frequencies of developmental defects observed (Liver cirrhosis/alcoholism: χ^2=1.314; P-value > 0.05; Fisher's exact test P=0.3158; Pellagra: χ^2=1.167; P-value > 0.05; Fisher's exact test P=0.4048). The one case of beriberi also presented with neural tube defects. The Chinese miners did, however, present with statistically higher frequencies of developmental defects than the general malnutrition individuals (χ^2=8.667; P-value < 0.05; Fisher's exact test P=0.0084).

The frequency of developmental defects observed in the Chinese miners were therefore similar to that observed for the liver cirrhosis/alcoholism and pellagra individuals. This may be attributed to a prenatal zinc, vitamin B12 and folic acid deficiency in all three groups. A zinc, vitamin B12 and folic acid deficiency has been readily associated with abnormal development and growth and is known lead to especially neural tube defects (Ball, 1998; Groenen *et al.*, 2004a-b; Bailey, 2007; Suskind, 2009). A deficiency in either of these is usually attributed to dietary practices and in some cases (for example vitamin B12) as a result of other underlying conditions like infections, gastro-intestinal parasites and general malabsorption (Truswell and Milne 2002; Green and Miller, 2007; Walker *et al.*, 2009). In the case of dietary practices these are often associated with the individual's socio-economic status (poorer communities consuming cheaper cereal based diets) and cultural practices (vegetarian and vegan diets). These aspects may be present from one generation to a next. As such, aspects of maternal health should also be considered and may here be used as an explanation for the possible cause for the presence of developmental defects in the Chinese miners and cadaver remains.

The traditional Chinese diet contains very little zinc, vitamin B12 and folic acid. This is mostly due to the fact that their diet contains very little animal products (meat, seafood, and milk containing zinc, vitamin B12 and folic acid) (Suskind, 2009). Furthermore, the consumption of a cereal based staple diet would decrease the amount of zinc, vitamin B12 and folic acid intake and in some cases may actually inhibit the absorption of these vitamins and minerals (for example zinc) as a result of the high phytate content of cereals (Rose, 1982; Carpenter, 2000; Samman, 2002; Suskind, 2009).

This is also true for individuals suffering from pellagra. As pellagra is often caused by a maize dependent diet the intake of zinc, vitamin B12 and folic acid would also be affected

along with the inhibiting effects of a concentrated phytate diet. It has been shown that pellagra in itself may also cause low plasma zinc levels (Brenton and Paine, 2007).

In the case of the liver cirrhosis/alcoholism individuals it has been shown that alcohol increases the level of zinc and iron excretion (Rodriguez-Moreno *et al.*, 1997). It also accelerates megaloblastic changes which in turn will increase the need for iron, folic acid and vitamin B12 (Rose, 1982). It therefore seems likely that individuals dependent on alcohol would be deficient in zinc, vitamin B12 and folic acid.

The individuals who died as a result of general malnutrition however presented with significantly lower frequencies of developmental defects than observed in the Chinese miners. This may indicate that these individuals were not so readily affected by prenatal deficiencies in zinc, vitamin B12 and folic acid and that this condition may be more prone in cases where metabolic diseases directly affect the intake of these vitamins and minerals.

The apparent association between the frequencies of developmental defects obtained from the Chinese miners, the pellagra and the general malnutrition groups may therefore be reflective of their mother's health and a may represent a lasting nutritional problem. This then not only provides evidence of maternal health, representative of the previous generation health, but also indicates that the children born from them are already placed at a disadvantage and that these conditions may have been exacerbated during life.

5.3.6. Enamel hypoplasia

In the modern skeletal remains (cadavers), the largest incidence of enamel hypoplasia was observed for those individuals who died as a result of pellagra (45.5%). This was followed by 20.4% observed in the general malnutrition cases and 16.7% in the liver cirrhosis/alcoholism cases. Enamel hypoplasia was also noted in the single cases of beriberi, scurvy and anaemia alternatively. These frequencies were statistically significantly lower than the 93.1% observed in the Chinese miners (Pellagra: χ^2=11.317; P-value < 0.05; Fisher's exact test P=0.0026; General malnutrition: χ^2=37.545; P-value < 0.05; P=0.0001; Liver cirrhosis/alcoholism: χ^2=38.611; P-value < 0.05; P=0.0001). This seems to suggest that the Chinese individuals were subjected to very poor conditions during childhood as these frequencies are high when compared to the cadaveric remains. This supports the notion that the Chinese individuals experienced very harsh living conditions back in China and came from very poor peasant families. Many factors play a role in the appearance of enamel hypoplasia but they can broadly be categorized as either due to nutritional deficiencies, or

childhood illnesses (Roberts and Manchester, 1995). Historical records continuously refer to large scale famines across China as well as the apparent effects of malnutrition amongst Chinese as indicated by the high incidence rate of beriberi. These factors would, therefore, easily have contributed to the occurrence of enamel hypoplasia.

5.3.7. Summary of findings

An assessment of skeletal pathology in the 36 Chinese miners revealed a high prevalence of nutrition-related changes. These changes revealed a long-standing nutritional problem amongst the Chinese. Chinese miners were already born into nutritionally compromised families as suggested by the presence of developmental defects. These conditions continued throughout childhood and into adulthood as suggested by the presence of linear enamel hypoplasia and residual rickets. At some point prior to their arrival in South Africa or during their time on the Witwatersrand mines their nutritional intake must have improved as suggested by the healing of porotic lesions.

When referring back to the archival and historical information it is known that Chinese labourers imported from China were already subjected to less than ideal living conditions. Most of those recruited came from low income peasant societies across China, which were already affected by famine (Richardson, 1982; Yap and Man, 1996; Harris, 1998). Polished white rice or wheat based cereals were the staple diet in China at the time, mostly consumed on its own with little or no dairy or animal proteins (Carpenter, 2000). This diet lacked most of the essential vitamins and minerals. A nutritionally imbalanced diet most probably contributed to the appearance of metabolic diseases in most of the Chinese labourers. Even though the Chinese labourers were allowed to consume their traditional diets once employed on the Witwatersrand mines, the Chamber of Mines soon insisted that rations be provided that complied with nutritional standards and minimum daily requirements (Richardson, 1982). It therefore seems that these individuals may have experienced a period of healing possibly associated with a more nutritionally balanced diet once employed on the mines.

However, the living and working conditions on the mines subjected the Chinese miners to additional hazards. The occurrence of infectious disease like syphilis and the high frequency of peri-mortem trauma, which contributed to these people's deaths, attest to the harsh living and working conditions associated with mining. Historical records indicated that one in twenty deaths could be attributed to work related accidents (Yap and Man, 1996).

Many of these accidents were associated with explosions, rock falls and cave-ins, and falls from heights (down mining shafts) which may explain the high frequency of peri-mortem trauma observed in the Chinese miners. Therefore, even though the Chinese may have experienced a period of improved nutrition on the mines, the hazardous working conditions created by deep-level mining lead to the eventual death of many.

Cross-comparison of skeletal pathology observed in the Chinese miners with that of cadaver skeletal remains, for whom the cause of death was known, to some extent enabled better identification and interpretation of skeletal pathology related to metabolic and nutritionally-related diseases. The closest association in the skeletal pathology observed in the cadaver skeletal remains and that observed in the Chinese miners were for those individuals for whom the cause of death was given as liver cirrhosis/alcoholism and pellagra. All three groups (Chinese miners, liver cirrhosis/alcoholism and pellagra) presented with similar frequencies of porotic hyperostosis, bowing deformities and developmental defects. This apparent association may be as a result of similar underlying vitamin and mineral deficiencies.

Archival and historical records indicate a high incidence rate of dietary beriberi amongst Chinese. One condition readily associated with chronic alcoholism is beriberi, which may explain the presence of similar skeletal pathologies in the liver cirrhosis/alcoholism individuals and the Chinese miners. It has also been shown that liver cirrhosis/alcoholism can cause several other vitamin deficiencies as a result of diminished liver function, poor diet, and alcohol's inhibiting effects on the absorption of certain vitamin and minerals (other B vitamins, folic acid, zinc, calcium, vitamin D, and iron).

The traditional Chinese diet (a cereal-based staple diet of white polished rice or wheat with very little animal derived foods) also lacked many of these essential vitamins and minerals. Most of the B vitamins, zinc, calcium, vitamin D and iron are contained in animal derived foods and would therefore not have been readily available to the Chinese. A cereal based staple diet may further have contributed to a deficiency in these vitamins and minerals as such diets contain large quantities of phytates which are known to decrease the bioavailability of for example zinc and iron.

The manifestation of pellagra is also most often associated with the consumption of a cereal based staple diet, specifically of maize. This condition is caused by a deficiency in vitamin B3, but may also contribute to a deficiency in other B vitamins, zinc and iron. Pellagra is often associated with a vitamin B2 and B6 deficiency as many B vitamins have interlinking functions which means that a deficiency in the one may lead to a deficiency in

another. B vitamins also play a role in the effective metabolism of iron which if lacking may cause iron deficiency. In cases where pellagra is acquired as a result of a cereal-based staple diet the high amount of phytate intake associated with it may inhibit zinc and iron absorption. A cereal-based staple diet, generally nutritionally restricted, will decrease the overall intake of B vitamins, folic acid, zinc, calcium, vitamin D, iron and vitamin C (as possibly indicated by the higher frequencies of cribra orbitalia observed in the pellagra individuals) leading to the appearance of more than one deficiency and therefore more than one type of skeletal pathology.

The appearance of similar frequencies of porotic hyperostosis in all three these groups may therefore be associated, in part, with a deficiency in B vitamins, folic acid and iron. Bowing deformities, on the other hand, may be related to a coinciding deficiency in calcium and vitamin D, whereas developmental defects may reflect a prenatal deficiency in zinc and folic acid.

Fewer similarities in skeletal pathologies observed in the general malnutrition individuals and the Chinese miners were found. The reason for this may be that the cause of death, even though it was attributed to some form of nutritional deficiency, cannot be specified in terms of the actual vitamins/minerals absent. One individual would most probably have suffered from more than one vitamin/mineral deficiency and as a result could have presented with multiple skeletal pathologies. The overlapping responses of bone to metabolic insults further complicate the specific diagnosis of skeletal pathology. It therefore seems that without more specific information on the cause of death and the presence of underlying conditions prior to death a definitive diagnosis cannot be based on skeletal pathology alone.

CHAPTER 6: CONCLUSIONS

- The biological profile of the Chinese miners is consistent with that of an indentured mining community. All 36 individuals were determined to be males and the majority of them were of young adult age (20-34). Some were slightly older (35-45 years) and two were of adolescent age (younger than 20).
- The demographic features of the 36 Chinese miner skeletons correspond with the archival and historical records which suggested that only males were employed on the mines and that the general age distribution of Chinese men ranged between 20 and 55 years (the majority of which were between 20 and 35 years). Records do, however, indicate that some of the Chinese employed were younger than 20 years, which would account for the two adolescent individuals.
- Pathology that could be observed included a high prevalence of nutrition-related changes and linear enamel hypoplasia which suggests that the Chinese miners had been subjected to long periods of malnutrition and illness throughout childhood continuing into adulthood. The occurrence of remodelling (especially of porotic lesions and bowing deformities) suggests a period of healing prior to their arrival on the Witwatersrand mines or prior to death during their time on the mines.
- Archival and historical records indicated that the communities from which Chinese men were recruited were of poor socio-economic status. These people already suffered from malnutrition as is evident by the high incidence rate of beriberi. Conditions were made worse by episodes of famine and the effects of political and economic instability seen throughout China.
- A high frequency of traumatic lesions, specifically peri-mortem fractures, was observed and may have contributed to the death of many of the Chinese miners. This is reflective of the occupational hazards associated with deep level mining especially considering that deep level mining was in its infancy on the Witwatersrand mines when the Chinese were imported and employed as contract labourers.
- Archival and historical records confirmed the high death rate associated with work-related accidents. Many of these accidents were caused by drilling into explosives, rock falls and cave-ins, and falls down mining shafts. These types of fatal accidents

may therefore explain the high percentage of peri-mortem fractures observed amongst the Chinese miners.

- In order to aid in the interpretation of skeletal pathology associated with metabolic and nutritional diseases, non-specific signs of disease observed in a cadaver skeletal sample with known causes of death (related to specific metabolic or nutritional diseases) were compared to pathology observed in the Chinese miners. This provided pathological patterns which enabled a better interpretation of the pathology observed in the Chinese skeletal remains.
- The closest association in the skeletal pathology observed in the cadaver skeletal remains and that observed in the Chinese miners were for those individuals for whom the cause of death was given as liver cirrhosis/alcoholism and pellagra. All three groups presented with similar frequencies of porotic hyperostosis, bowing deformities and developmental defects. This apparent association may be as a result of similar underlying vitamin and mineral deficiencies. Porotic hyperostosis may therefore be associated, in part, with a deficiency in B vitamins (beriberi and pellagra), folic acid and iron. Bowing deformities may be related to a coinciding deficiency in calcium and vitamin D, whereas developmental defects may reflect a prenatal deficiency in zinc and folic acid.
- Fewer similarities in skeletal pathologies observed in the general malnutrition individuals and the Chinese miners were found. This may be due to the generalized cause of death given for these individuals and the lack of more specific information pertaining to the actual vitamins and minerals absent. These individuals would have presented with more than one vitamin/mineral deficiency and as a result could have presented with multiple skeletal pathologies. The overlapping responses of bone to metabolic insults further complicate the differentiation of skeletal pathology on the basis of its aetiology.
- Specific diagnoses are not always possible when dealing with skeletal pathology associated with nutritional deficiencies and metabolic bone diseases. This stems from the fact that vitamins and minerals often work hand in hand and as a result may lead to co-occurring deficiencies. Bone's limited ability to respond to metabolic insults means that different deficiencies may present with similar skeletal pathologies. However, this study has shown that by combining the occurrence of skeletal pathology as well as their known aetiologies with more specific information

pertaining to the cause of death (in the case of cadaveric skeletal remains) as well as possible underlying conditions prior to death (in the case of the Chinese miners' social and nutritional history) it may actually aid in the differentiation and diagnosis of specific nutritional deficiencies and metabolic diseases.

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APPENDIX 1: INDIVIDUAL SKELETAL REPORTS

This appendix shows the skeletal reports for each of the 36 Chinese individuals. The preservation and skeletal inventory are provided in Table 1 at the end of the reports. Each report discusses the estimation of age at death as well as the individual's sex, followed by their stature and dentition. Sex estimation was also compared to the results obtained by Gibbon *et al.* (2010). Finally the presence of any trauma and/or pathology was discussed for each individual. Since it is already known from the historical accounts that these individuals were of Chinese ancestry, ancestry was not discussed here. All cranial, postcranial and dental measurements taken are provided in Table 2-4.

1. A996

1.1. Age

The upper and lower third molars were in complete occlusion and showed almost no signs of occlusal wear which is consistent with someone of young adult age. An adult age was confirmed by the complete fusion of all visible epiphyses. It is uncertain whether the medial ends of the clavicles were completely fused as this area was badly deteriorated. Cranial suture closure suggested a young adult age between 15-40 years, whereas the few usable sternal ends of the ribs fell within phase 3 suggesting an age range between 24 and 28 years. No signs of degenerative changes on the joints and vertebrae could be observed, further suggesting a younger adult age. A final age estimate of 25-35 years was therefore given. This individual would therefore fall within the 20-34 year age category according to Falys and Lewis (2011).

1.2. Sex

The morphological features of the skull and pelvis were consistent with that of males. The skull presented with a sloped forehead, a well-developed glabella, and rounded orbital margins. The nuchal crest was well developed and the mastoid processes large. The mandible presented with a square-shaped chin and prominent mental eminence. Only the left pubic symphysis was present with morphological features confirming a male sex. Discriminant function analyses of the humerus (0.852437) and femur (Function 1: 0.053766; Function 2: 0.899381) fell well within the limits for Chinese males. This diagnosis of sex agrees with the findings of Gibbon *et al.* (2010).

1.3. Stature

Stature was determined from the maximum lengths of the femur and tibia. A stature of 163.91 ± 3.91 cm was obtained.

1.4. Dentition

Almost all the teeth were present except for the upper central and lateral incisors, the upper right canine and the lower central incisors, the lower right lateral incisor and the lower right canine. These teeth were all lost post-mortem. Dental pathology included linear enamel

hypoplasia on the lower right lateral incisor, the lower left canine, as well as the lower left and right first and second premolars. Linear enamel hypoplasia could not be observed on any of the maxillary teeth due to the erosion of the buccal surfaces. Occlusal wear was observed on the surfaces of the upper and lower first and second molars. These presented with small patches of dentine exposure. The upper and lower first and second premolars also showed some occlusal wear, but these teeth did not present with any dentine exposure.

1.5. Trauma and pathology

No trauma could be observed. Other pathologies that could be observed included porotic hyperostosis (degree score 1 to 2; healed) on the parietal and occipital bones especially in the region of the lambdoid suture, as well as periostitis on the medial and lateral surfaces of the left and right femur and tibia.

1.6. Summary

Adult male, 20-34 years, 163.91 ± 3.91 cm tall. Linear enamel hypoplasia, porotic hyperostosis and periostitis were present.

2. A997

2.1. Age

All visible teeth were completely erupted and in occlusion. The upper and lower third molars could not be observed in the alveolar bone, suggesting a possible young age. All the long bone epiphyses were fused suggesting an age older than 20 years. The medial ends of the clavicles and S1 and S2 of the sacrum were not completely fused, suggesting an age younger than 30 years. Cranial suture closure also suggested a young adult age at a range of between 15-40 years. The auricular surfaces presented as phase 1 which is consistent with an age range between 20-24 years. The final age estimate for this individual was between 20 and 25 years at the time of death. This individual would therefore fall within the 20-34 years age category as suggested by Falys and Lewis (2011).

2.2. Sex

The morphological features of the skull and pelvis were consistent with that of a male individual. The skull presented with a well-developed glabella with rounded orbital margins. The nuchal crest was pronounced and the mastoid processes large. The pelvis presented with a narrow greater sciatic notch and no pre-auricular sulcus. The pubic bone showed no concavity and a relatively broad medial surface suggestive of a male individual. Metric assessment of sex also indicated a male. Discriminant function analyses of the humerus (0.341718) and femur (Function 1: 0.354792; Function 2: 0.430918) fell well within the limits for male individuals. Gibbon *et al.* (2010) also determined a male sex for this individual.

2.3. Stature

The stature was determined by using the maximum lengths of the femur and tibia. A stature of 166 ± 3.91 cm was obtained.

2.4. Dentition

The upper and lower third molars had not yet erupted whereas the lower right first molar was lost ante-mortem, presenting with almost complete alveolar resorption. The upper central and lateral incisors, along with the lower central incisors, left lateral incisor and the left first premolar were lost post-mortem. The lower left first molar presented with a small carious lesion on the occlusal surface. Linear enamel hypoplasia was also noted on the buccal surfaces of the upper left and right canines and first and second premolars. All of the mandibular teeth showed enamel defects associated with linear enamel hypoplasia. Occlusal wear was observed on the surfaces of the upper and lower, left and right, first and second molars and second premolars.

2.5. Trauma and pathology

Trauma in the form of peri-mortem fractures was observed in the right humerus (complete transverse fracture) and right femur (spiral fracture) with a possible wedge fracture of T12. These showed no signs of healing. This individual also presented with porotic hyperostosis (degree score 1 to 2; healed) on the parietal and occipital bones, concentrated around the lambdoid suture. Periostitis was evident on the left femur (medial surfaces) and a Schmorl node could be seen on T11.

2.6. Summary

Adult male, 20 – 34 years, 166 ± 3.91 cm tall. Linear enamel hypoplasia, caries, peri-mortem fractures, porotic hyperostosis, periostitis, and a Schmorl node were present.

3. A998

3.1. Age

The lower third molars were in complete occlusion with the upper right third molar absent possibly due to agenesis. The upper left third molar was only partially erupted. All of the visible epiphyses were completely fused although poor preservation hampered this technique to some extent. It was noted, however, that the speno-occipital synchondrosis still presented with a fusion scar, suggesting an age younger than 25 years. Cranial suture closure was also consistent with someone of young adult age (15-40 years). An age estimate of 18-25 years was therefore given here. This individual would therefore most probably fall within the 20-34 year age category suggested by Falys and Lewis (2010).

3.2. Sex

The only morphological features that could be observed were those from the skull. The pelvis was in a very poor state of preservation and could not be used for the determination of sex. The skull presented with a pronounced nuchal crest, glabella and supraorbital margins. The mastoid processes were very large and the mandible square shaped with a well-developed mental eminence. These features are synonymous with male individuals. The only metric data that could be used was the midshaft circumference of the femur (Function 4: 0.678132) which fell within the limits for male individuals. Gibbon *et al*. (2010) also obtained a male sex for this individual.

3.3. Stature

Stature was determined using the maximum lengths of the femur and tibia. A stature of 170.84 ± 3.91 cm was obtained.

3.4. Dentition

Almost all the teeth were present except for the upper right third molar which seemed to be genetically absent along with the upper right central incisor which was lost post-mortem. Slight occlusal wear could be observed on all the molars and premolars. Moderate dental calculus was also observed on the lingual surfaces of especially the lower central and lateral incisors as well as the lower canines. One large carious lesion was noted on the upper left first molar. This lesion presented as a coronal caries and was situated medially. Almost all the teeth presented with multiple linear enamel hyploplastic lesions. These were mainly concentrated on the upper and lower canines and upper and lower central and lateral incisors. Some hypoplastic lines could also be observed on the upper and lower premolars and molars.

3.5. Trauma and pathology

No trauma was observed. Pathological lesions included porotic hyperostosis (degree score 1 to 2; healed) on the occipital and parietal bones and very slight cribra orbitalia (degree score 1; healed) in the left orbital roof. Periostitis was observed on both tibiae (medial surfaces). The left and right femur presented with slight lateral bowing in the subtrochanteric region.

3.6. Summary

Adult male, 20-34 years, 170.84 ± 3.91 cm tall. Linear enamel hypoplasia, dental calculus, caries, porotic hyperostosis, cribra orbitalia, periostitis, and the bowing of the femur were present.

4. A999

4.1. Age

All teeth were erupted and in full occlusion suggesting an adult age. Very slight occlusal wear could be observed on the molars. All the visible epiphyses were completely fused along with the spheno-occipital synchondrosis (no scar line present) which suggested and age older than 25 years at the time of death. Cranial suture closure suggested a young adult age with and age range between 15-40 years. None of the sternal rib ends, pubic bones or auricular surfaces survived and therefore these techniques could not be used here. A final age estimation of 25-35 years was therefore provided here. According to Falys and Lewis's (2010) categories this individual would therefore fall in the 20-34 years age category.

4.2. Sex

Morphological features of the skull could be observed and were consistent with male features. The skull presented with a pronounced glabella, supraorbital margins and a nuchal crest. The mastoid processes were intermediate in size and the mandible presented with a

square shaped chin and a well-developed mental eminence. The pelvis was badly preserved and the only diagnostic area that could be used for the determination of sex was the right greater sciatic notch. The greater sciatic notch was intermediate in size and could therefore not be used. The skeleton in general was quite robust with the long bones presenting with marked muscle attachments. Metric analysis of the femur also suggested a male sex (Function 1: 1.451402; Function 3: 1.522221). Gibbon *et al.* (2010) could not obtain any result for the sex of A999.

4.3. Stature

For the determination of stature the maximum lengths of the femur and tibia were used and a stature of 165.56 ± 3.91 cm was obtained.

4.4. Dentition

Almost all the teeth were present except for the upper right third molar, left central incisor and the left canine which were lost post-mortem. The crowns of the upper right second molar and the lower right canine were also damaged post-mortem. Dental pathology that could be observed included moderate dental calculus on the lingual and buccal surfaces of the maxillary and mandibular teeth. Slight alveolar regression was observed which may suggest the presence of periodontal disease. Slight occlusal wear could be observed on the upper and lower occlusal surfaces of the molars with multiple linear enamel hypoplastic lesions on the upper and lower first and second premolars and canines as well as on the upper central and lateral incisors.

4.5. Trauma and pathology

The only trauma that was observed was post-mortem cut marks to the skull consistent with autopsy procedures. Slight porotic hyperostosis was observed on the occipital bone, however, poor preservation of the cortical bone and distortion resulting from the removal of the cranium during autopsy hindered the positive identification of this lesion. Slight cribra orbitalia (degree score 1 to 2; healed) was noted in both orbital roofs. The left and right scapula also presented with the calcification of the superior transverse scapular ligament.

4.6. Summary

Adult male, 20-34 years, 165.56 ± 3.91 cm tall. Linear enamel hypoplasia, dental calculus, periodontal disease, and cribra orbitalia were present.

5. A1000

5.1. Age

The upper and lower third molars were in complete occlusion and presented with slight occlusal wear. All the visible epiphyses were fused including the medial ends of the clavicles and the speno-occipital synchondrosis. This suggested an age older than 30 years. The degree of cranial suture closure suggested an age between 35 and 45 whereas the sternal ribs ends

represented as a phase 6 consistent with an age range of between 43 and 55 years. The left auricular surface presented as a phase 5, suggestive of an age of 40 to 44 years at the time of death. Osteophytes were also observed on the lumbar vertebrae, especially in L5, along with osteophytic lipping of the calcaneus and talus. This is also suggestive of someone older than 35 years. A final age estimation of 35-45 years was therefore provided here. For the age categories suggested by Falys and Lewis (2010) this individual would also fall within the 35-45 age range.

5.2. Sex

The skull presented with morphological features consistent with that observable in males. The skull presented with a pronounced glabella and supraorbital margins with relatively large mastoid processes and a nuchal crest presenting with marked muscle attachments. The mandible was square shape and presented with some gonial flaring and a well-defined mental eminence. The pelvis was not very viable for the morphological assessments of sexual traits and the only trait that could be observed was the left greater sciatic notch which was quite narrow and therefore more representative of a male. Metric analysis of the femur (Function 1: 2.610338; Function 2: 1.562666) and the humerus (Function 2: 0.998357) were consistent with values found for males. This individual was therefore classified as male and corresponded with the findings of Gibbon *et al*. (2010).

5.3. Stature

Stature was calculated by using the maximum lengths of the femur and tibia. A stature of 162.62 ± 3.25 cm was obtained.

5.4. Dentition

Most of the teeth were present except for the upper right second premolar, the upper central and lateral incisors, the lower right third molar, the lower right second premolar, the lower central incisors and the lower left lateral incisor. All of these teeth were lost post-mortem as suggested by the lack of alveolar resorption. Ante-mortem tooth loss of the upper left second molar, lower right first molar and the lower right first and third molars were observed. These teeth were lost as a result of abscesses formation. Various other carious lesions were observed on the maxillary teeth, presenting in the right first and second molars as well as the first premolar, left first molar and second premolar. As for the mandibular teeth dental caries was also noted in the right first molar and first premolar as well as the left second molar and premolar. The maxillary caries presented as coronal caries and affected the medial-lateral surfaces in adjacent teeth. The mandibular caries presented as both coronal and root caries with carious lesions occurring at the cement enamel border and extending into the crown and root. Some dental calculus was observed on the buccal surfaces of the upper and lower molars. Severe alveolar regression was also noted and can be associated with periodontal disease. Multiple linear enamel hypoplastic lesions were observed on the upper canines and the lower right first premolar and lower left canine.

5.5. Trauma and pathology

No trauma was observed. Porotic hyperostosis (degree score 1 to 2; healed) was observed on the parietal and occipital bones specifically in the region of the lambdoid suture. Periostitis was observed on the left and right tibia (medially). Osteophytic lipping of L5, the calcaneus and talus was also observed.

5.6. Summary

Adult male, 35-45 years, 162.62 ± 3.25 cm tall. Linear enamel hypoplasia, dental calculus, caries, abscess, periodontal disease, ante-mortem tooth loss, porotic hyperostosis, periostitis and osteophytic lipping were present.

6. A1001

6.1. Age

The upper and lower third molars were in complete occlusion but presented with no occlusal wear suggestive of a young adult. All the long bone epiphyses were fused. S1 and S2 were still open and the spheno-occipital synchondrosis presented with a clear fusion line. These suggest a very young adult age, probably younger than 25 years. Cranial suture closure provided an age range between 15 and 50 years whereas the auricular surfaces presented as phase 2, suggestive of an age of between 25-29 years. A final age estimation of between 20-30 years was therefore given here. For the Falys and Lewis (2010) age categories this individual would fall within the 20-34 age range.

6.2. Sex

Overall the skeleton was quite robust with marked muscle attachments. The morphological features of the skull and pelvis were consistent with that observable in males. The skull presented with a very robust glabella and supraorbital margin. The nuchal crest was also very well-defined and the mastoid processes large. The mental eminence was well-developed. The pelvis presented with narrow greater sciatic notches and the absence of a pre-auricular sulcus. Metric assessment of sex was done using the femur (Function 1: 0.872062; Function 2: 1.043947) and was consistent with the averages for male individuals. A male sex for this individual was also found by Gibbon *et al.* (2010).

6.3. Stature

Stature was calculated by using the maximum lengths of the femur and tibia. A stature of 168.20 ± 3.91 cm was obtained.

6.4. Dentition

Most of the teeth were present except for the maxillary right second premolar, the crowns of the central incisors, the left lateral incisors, the mandibular right first premolar and central and lateral incisors. Slight occlusal wear could be observed on the upper and lower first molars. Slight dental calculus was observed on the buccal surfaces of the maxillary molars.

Small carious lesions were observed on the occlusal surfaces of the upper right third molar as well as the upper left second and third molars. A large coronal caries was also observed on the lower right second molar. Some alveolar regression was present which could suggest periodontal disease. Multiple linear enamel hypoplastic lesions were also noted on the upper and lower canines.

6.5. Trauma and pathology

No trauma was observed. Porotic hyperostosis (degree score 1 to 2; healed) could be observed in the region of the lambdoid suture and extending towards the parietal and occipital bones. Slight cribra orbitalia (degree score 1; healed) was noted in both orbital roofs. Both tibiae presented with periostitis (medially). The tibiae and fibulae showed some medial-lateral bowing. Sacralization of L5 and the sacrum was observed. L5 also presented with a cleft neural arch.

6.6. Summary

Adult male, 20-34 years, 168.20 ± 3.91 cm tall. Linear enamel hypoplasia, dental calculus, caries, periodontal disease, porotic hyperostosis, cribra orbitalia, bowing of tibiae and fibulae, sacralisation and cleft neural arch in L5 were present.

7. A1002

7.1. Age

The upper third molars were in full occlusion and presented with almost no occlusal wear, suggestive of a young adult age. The speno-occipital synchondrosis was completely fused, suggesting an age older than 25 whereas the medial ends of the clavicles were also fused suggestive of an age of older than 30 years. No degenerative changes to the vertebrae or major joints could be observed which would be more consistent with a younger adult. A final age estimation of between 25-35 years was given here. This age would fall within the 20-34 years age category as suggested by Falys and Lewis (2010).

7.2. Sex

The morphological features observable on the skull were consistent with male features. The skull presented with a well-developed glabella with prominent supraorbital margins, a somewhat sloped forehead, a well-developed nuchal crest and relatively large mastoid processes. The mandible was square shaped with a pronounced mental eminence. The only observable feature on the pelvis was the greater sciatic notches which were very narrow and therefore also indicative of a male individual. Discriminant function analysis of the femoral measurements (Function 2: 1.999664) also confirmed a male sex for this individual. The study by Gibbon *et al.* (2010) could not obtain any result for this individual.

7.3. Stature

Stature was calculated using the maximum lengths of the femur and tibia. A stature of 166 ± 3.91 cm was obtained.

7.4. Dentition

Almost all the teeth were present except for the upper right central and lateral incisors, the lower left and right central incisors and the lower left canine. These teeth were lost post-mortem. The lower left and right third molars were also missing but seemed to have been genetically absent. Dental caries were observed on the upper left and right third molars on the buccal surfaces in the region of the cemento-enamel junction. The lower left and right second molars also presented with small carious lesions, but these were positioned on the occlusal surfaces. Multiple linear enamel hypoplastic lesions were also noted on all the teeth except for the upper third molars.

7.5. Trauma and pathology

No trauma could be observed. Possible slight cribra orbitalia (degree score 1; healed) was observed in both orbital roofs. L5 presented with a cleft neural arch whereas the sacrum presented with possible spina bifida occulta.

7.6. Summary

Adult male, 20-34 years, 166 ± 3.91 cm tall. Linear enamel hypoplasia, dental caries, possible cribra orbitalia, possible spina bifida and a cleft neural arch in L5 were present.

8. A1003

8.1. Age

The lower third molars were in complete occlusion and presented with some occlusal wear, suggesting an adult age for this individual. All visible epiphyses were fused including the medial ends of the clavicle, S1 and S2 and the spheno-occipital synchondrosis which suggested an age older than 30 years. Cranial suture closure placed this individual between the ages of 27 and 56 years whereas the auricular surfaces suggested an age range between 30-39 years at the time of death. A final age estimation of 30-45 years was given here. This places this individual in the 35-45 year age category as suggested by Falys and Lewis (2010).

8.2. Sex

Overall this individual was quite robust and most of the long bones presented with marked muscle attachments which is suggestive of someone of a more masculine build. The morphological features of the skull were consistent with that observable in males. The skull was somewhat sloped and presented with a very well-developed glabella, prominent supraorbital margins, a well-developed nuchal crest and relatively large mastoid processes. The mandible was square shaped with some gonial flaring and a pronounced mental eminence. The pelvis also presented with characteristics associated with male individuals.

The pubic bone presented with no subpubic concavity and a broad ischiopubic ramus. The greater sciatic notches were very narrow and no pre-auricular sulcus could be observed. Metric assessment of the femur (Function 2: 0.686949) also suggested a male sex. Results obtained for this individual in the study by Gibbon *et al.* (2010) correlates with this finding.

8.3. Stature

The maximum lengths taken from the femur and tibia were used to determine the stature. A stature of 152.87 ± 3.25 cm was obtained for this individual.

8.4. Dentition

Almost half of the teeth were lost post-mortem. The lower right first molar was however lost ante-mortem and presented with alveolar resorption. The maxillary teeth that were present included the left first and second molars, second premolar and the right canine and first premolar. As for the mandibular teeth the right third and second molars and first and second premolars along with the left first premolar and first, second and third molars were present. Slight occlusal wear could be observed on all of the teeth.

8.5. Trauma and pathology

Peri-mortem trauma of the spinious process of T2 (compression fracture) as well as in the right os coxa (compression fracture) was observed. No healing could be observed. Ante-mortem trauma was also observed with two healed fractures in the shaft and the distal epiphysis of the right fibula. Pathological lesions that could be observed included porotic hyperostosis (degree score 1 to 2; healed) mainly on the occipital bone with some involvement of the parietal bones (concentrated around the lambdoid suture). Possible slight cribra orbitalia (degree score 1; healed) was also noted in both orbital roofs. The right tibia also presented with slight periostitis (laterally) which could be related to the ante-mortem fracture observed on the right fibula. The left scapula presented with the calcification of the superior transverse scapular ligament.

8.6. Summary

Adult male, 35-45 years, 152.87 ± 3.25 cm tall. Peri-mortem and ante-mortem fractures, porotic hyperostosis, possible cribra orbitalia and periostitis were observed.

9. A1004

9.1. Age

The upper and lower molars showed no occlusion suggesting a young age. Epiphyseal fusion seemed to confirm this. The medial ends of the clavicles as well as S1 and S2 were still completely unfused, suggesting an age younger than 25 years at the time of death. The spheno-occipital synchondrosis also seemed to be unfused, suggesting an age younger than 18 years. The proximal epiphyses of the left and right humerus were only recently fused and a clear fusion line was still visible. This indicated an age of between 16-20 years. The iliac

crest also presented with a recent fusion line which is suggestive of an age range of between 17 and 20 years. A final age estimation of 17-20 years was therefore provided. This individual would therefore fall within the >20 years age category as suggested by Falys and Lewis (2010).

9.2. Sex

The individual presented with an overall robust skeleton. The morphological features of the skull were consistent with those observable in male individuals. The skull presented with a well-developed glabella, pronounced supraorbital margins and a square chin shape with a well-developed mental eminence. The only observable morphological features of the pelvis were the greater sciatic notch which was very narrow and the absence of a pre-auricular sulcus. Metric analysis also confirmed a male sex for this individual. Discriminant function analysis of the femoral measurements provided results that fell well within the limits set for male individuals (Function 1: 0.215114; Function 2: 0.081709). Molecular analysis done by Gibbon *et al.* (2011) also suggested a male sex for this individual.

9.3. Stature

Due to the young age of this individual stature could not be determined here.

9.4. Dentition

Almost all the teeth were present except for the upper and lower third molars which have not erupted yet. The upper right lateral incisor and lower left lateral incisor were lost post-mortem. Dental caries was observed on three teeth. The upper left second molar presented with a large coronal carious lesion situated on the occlusal surface and extending to the lingual surface. The lower right second molar presented with a very large carious lesion which affected almost the whole crown. No root involvement was noticed. The lower right first molar also presented with a small carious lesion on the occlusal surface. This individual also presented with severe linear enamel hypoplasia. These defects could be seen on all the teeth and presented as multiple lines.

9.5. Trauma and pathology

Peri-mortem fractures could be observed on the left radius, the right femur, as well as the right tibia and fibula. All of these fractures presented as complete butterfly fractures of the shafts. No healing was observed. Slight periostitis was observed on the left and right femur and tibia (medially).

9.6. Summary

Adolescent male, >20 years. Linear enamely hypoplasia, dental caries, peri-mortem fractures and periostitis were present.

10. A1005

10.1. Age

The upper and lower third molars were in complete occlusion and presented with almost no occlusal wear, suggestive of a young adult age. All the long bone epiphyses were fused. The spheno-occipital synchondrosis showed a clear fusion line and S1 and S2 were still unfused suggesting an age younger than 25 years. The medial ends of the clavicles only started to fuse, suggesting an age between 23 and 29 years. The observable cranial sutures also showed minimal fusion. A final age estimation of between 18-25 years was therefore given here. This age estimate would fall within the 20-34 year age group as given by Falys and Lewis (2010).

10.2. Sex

Overall the skeletal remains were quite robust with marked muscle attachments. The skull presented with male features in the form of a well-developed nuchal crest and glabella. The mastoid processes were intermediate and could therefore not be used to predict sex. The mental eminence was again very pronounced and the mandible square shaped, suggestive of a male. The pelvic dimensions also presented with some male characteristics with a narrow greater sciatic notch and the absence of a pre-auricular sulcus. The dimensions of the pubic bone were however intermediate between male and female characteristics and could therefore not be used. Metric analysis of femoral measurements Function 1 (-0.0591037) and Function 3 (-0.828254) suggested a female sex for this individual whereas Function 2 (0.779536) suggested a male sex. Molecular sex determination was also inconclusive for this individual, possibly due to poor preservation (Gibbon *et al.*, 2010). Overall this individual did however present with morphological and some metric features suggestive of someone of a more masculine build. This individual was therefore assigned to be possibly male.

10.3. Stature

The stature of this individual was obtained from the maximum lengths of the femur and tibia. A stature of 163.91 ± 3.91 cm was obtained.

10.4. Dentition

Most of the teeth were present except for the upper left central incisor, the upper right central incisor crown, the upper third molar, the lower right first premolar, canine and lateral incisor. All of these teeth were lost post-mortem. Slight occlusal wear was observed on the upper and lower first and second molars. Linear enamel hypoplasia was noted on the upper and lower left lateral incisors, canines and first and second premolars.

10.5. Trauma and pathology

No trauma was observed. Pathology that could be observed included porotic hyperostosis (degree score 1 to 2; healed) on the occipital and parietal bones, concentrated around the lambdoid suture, and possible cribra orbitalia (degree score 1; healed) in both orbital roofs. Some slight medial-lateral bowing of the tibia and fibula were also noticed.

10.6. Summary

Adult male, 20-34 years, 163.91 ± 3.91 cm tall. Linear enamel hypoplasia, porotic hyperostosis, possible cribra orbitalia and slight bowing of tibia and fibula were present.

11. A1006

11.1. Age

The upper and lower third molars were in complete occlusion and showed some occlusal wear. Occlusal weathering was more severe on the second and especially the first molars, suggesting a possible middle adult age. This was corroborated with the degree of epiphyseal fusion. All the visible epiphyses were completely fused including the spheno-occipital synchondrosis. This suggested that this individual was older than 30 years. Cranial suture closure was also consistent with a middle adult age. A final age estimation of 35-45 years was therefore provided here. This corresponds with the age category provided by Falys and Lewis (2010).

11.2. Sex

The morphological features of the skull and pelvis were consistent with those associated with males. The skull presented with a well-developed nuchal crest, a sloped forehead, a large glabella and pronounced supraorbital margins and a well-defined mental eminence. The pelvis, however, was badly preserved except for the greater sciatic notches which were narrow and deep and presented without a pre-auricular sulcus. Discriminant function analysis of the femoral dimensions suggested a male sex for Function 1 (0.532294595) as well as Function 2 (0.681608188). Gibbon *et al.* (2010) could not obtain any results.

11.3. Stature

The stature was determined by using the maximum lengths of the femur and tibia. A stature of 170.55 ± 3.25 cm was obtained for this individual.

11.4. Dentition

Most of the teeth were lost post-mortem. Teeth that were present included the upper right first molar, the upper left first and second premolars, the upper left first molar, and the lower left and right first, second and third molars. Some occlusal wear was noted on the molars.

11.5. Trauma and pathology

No trauma was observed. Pathology that could be observed included porotic hyperostosis (degree score 1; healed) in the region of the lambdoid suture extending towards the occipital and parietal bones. Slight medial-lateral bowing of the fibulae was observed.

11.6. Summary

Adult male, 35-45 years, 170.55 ± 3.25 cm tall. Porotic hyperostosis and bowing of fibulae were observed.

12. A1007

12.1. Age

The upper and lower third molars were in complete occlusion with slight wear on the occlusal surfaces, suggesting an adult age. Epiphyseal fusion was complete in all the observable areas including the medial ends of the clavicles and the spheno-occipital synchondrosis, suggesting an age older than 30 years. The coccyx was also fused to the sacrum indicating a middle adult age. The degree of cranial suture closure suggested an age range of between 30-40 years. Some degenerative changes could be observed on the elbow joints and lumbar vertebrae in the form of osteophytic lipping and outgrowths, suggesting that the individual was most probably older than 35 years. A final age estimate of 35-45 years was therefore given here. This corresponds with the age category suggested by Falys and Lewis (2010).

12.2. Sex

The morphological features of the skull and pelvis were consistent with that of males. The skull presented with a robust and well-developed nuchal crest, large mastoid processes, robust supraorbital margins and glabella, and a prominent mental eminence. The pelvis presented with a narrow and deep greater sciatic notch, a heart-shaped pelvic inlet and no pre-auricular sulcus. Metric analysis of the femoral dimensions confirmed a male sex. Function 1 (0.716936), Function 2 (0.707142) and Function 3 (0.225185) were all consistent with averages obtained for males. This individual was therefore classified as male and corresponds with the molecular sex results obtained by Gibbon *et al.* (2010).

12.3. Stature

The stature was calculated by using the maximum lengths of the femur and tibia. A stature of 169.25 ± 3.25 cm was obtained.

12.4. Dentition

Almost all the teeth were present except for the upper right second and third molars, the upper left central and lateral incisors, and the upper left third molar. Occlusal wear was noted on the upper and lower molars and premolars. Dental pathology occurred in the form of severe linear enamel hypoplasia. These defects could be seen on all the teeth and presented as multiple lines.

12.5. Trauma and pathology

No trauma could be observed. Pathology that was noted included spondylolysis of L5 (bilateral involvement) and some osteophytic lipping of the ulna and the lumbar vertebrae.

12.6. Summary

Adult male, 35-45 years, 169.25 ± 3.25 cm tall. Linear enamel hypoplasia, spondylolysis of L5 and osteophytic lipping were present.

13. A1008

13.1. Age

The lower third molars were in complete occlusion with minimal wear suggesting an adult age. The upper third molars could not be observed in the alveolar bone possible due to agenesis as no alveolar scarring, suggestive of ante-mortem tooth loss could be observed. Epiphyseal fusion further suggested an adult age with the complete fusion of spheno-occipital synchondrosis, S1 and S2 of the sacrum, and the medial ends of the clavicles, suggesting an age older than 30 years. A relative degree of cranial suture closure was observed along with the presence degenerative changes in the lumbar vertebrae, the left clavicle and tibia as well as the left and right calcaneus indicating an older adult age. A final age estimate of between 35 and 45 years was given here. This corresponds with the age category suggested by Falys and Lewis (2010).

13.2. Sex

The morphological features observable on the skull and pelvis were consistent with someone of male sex. The skull was quite robust presenting with a sloped forehead, relatively pronounced supraorbital margins, rounded orbital margins, large mastoid processes and a well-developed nuchal crest. The mandible was square shaped and presented with a prominent mental eminence. The pelvic bones were somewhat damaged although observable features such as the greater sciatic notch, which was narrow, and the absence of a pre-auricular sulcus further suggested a male sex. Metric analysis of the femoral dimensions, Function 1 (1.791394) and Function 2 (1.835088), were consistent with averages obtained for males. This individual was therefore classified as male and corresponds with the molecular sex results obtained by Gibbon *et al.* (2010).

13.3. Stature

The stature was determined by using the maximum lengths of the femur and tibia. A stature of 158.72 ± 3.25 cm was obtained.

13.4. Dentition

Almost all the teeth were present except for the upper left and right third molars that seemed to have been absent due to agenesis, as well as the upper right central incisor and the upper left lateral incisor which were lost post-mortem. Occlusal weathering of all of the teeth except for the lower left and right third molars was observed. Slight dentine exposure could be observed on the lower canines and lateral and central incisors as a result. Thin bands of dental calculus deposits were observed on the upper canines and lateral incisors on the lingual surfaces as well as on the lower first premolars and canines on the buccal surfaces. Slight alveolar regression was observed suggesting the presence of periodontal disease. Linear

enamel hypoplasia was observed on the upper and lower first and second premolars and canines. These defects presented as multiple lines.

13.5. Trauma and pathology

No trauma could be observed. Pathology that could be observed included destructive lesions on the cranium possibly representative of caries sicca associated with treponemal disease. The lack of any associated postcranial pathology may also suggest that these lesions could be associated with osteomyelitis. Furthermore, degenerative changes in the form of osteophytic lipping of the lumbar vertebrae and an osteophytic outgrowth of the left clavicle, the left proximal tibia and the left and right calcaneus were observed. Partial sacralisation of L5 was also noted.

13.6. Summary

Adult male, 35-45 years, 158.72 ± 3.25 cm tall. Linear enamel hypoplasia, dental calculus, periodontal disease, possible caries sicca and osteophytic lipping were present.

14. A1009

14.1. Age

The upper and lower third molars could not be observed in the alveolar bone whereas the second and first molars showed minimal occlusal weathering, suggesting a possible young age. Epiphyseal closure also suggested a young age with the incomplete fusion of the medial ends of the clavicles as well as S1 and S2. This suggested an age younger than 25 years at the time of death. All the other long bones were, however, completely fused suggesting an age older than 20 at the time of death. A final age estimation of 20-25 at the time of death was therefore given here. This age estimate would however fall within the 20-34 year age category as suggested by Falys and Lewis (2010).

14.2. Sex

Most of the morphological features observable on the skull and pelvis suggested a male sex. The skull presented with a sloped forehead, prominent glabella, rounded orbital margins, and large mastoid processes and a well-developed mental eminence. The only diagnostic feature observable on the pelvis was the greater sciatic notch which was relatively narrow and the absence a pre-auricular sulcus. Overall the skeletal remains were quite robust and presented with marked muscle attachments suggestive of a more masculine build. Metric analysis of the femoral dimensions however suggested a female sex with Function 1 (-1.407877) and Function 3 (-1.377807) falling within the limits for female individuals. These calculations may however have been influenced by the poor preservation of the periosteal surfaces of the long bones. Gibbon *et al.* (2010) could not obtain any molecular sex results for this individual possibly due to poor preservation. The majority of the morphological characteristics that could be observed however suggested a male sex and therefore this individual was classified as a possible male.

14.3. Stature

The maximum lengths of the humerus and radius were used to determine the stature. The stature was calculated to be 159.01 ± 4.31 cm.

14.4. Dentition

Most of the teeth were present except for the upper and lower third molars which have not yet erupted. Furthermore, the upper right central incisor, the upper left lateral incisor and canine, the lower left and right central and lateral incisors and the lower left second premolar were lost post-mortem. The only dental pathology that could be observed was severe linear enamel hypoplasia on the upper and lower premolars and canines. These defects presented as multiple lines.

14.5. Trauma and pathology

Peri-mortem trauma was observed in the form of a spiral fractures of the left femur, the left tibia and the left fibula. No healing could be observed. Pathology included porotic hyperostosis (degree score 1 to 2; active and healed) of especially the left and right parietal and occipital bones. Cribra orbitalia (degree score 1 to 2; active and healed) was also noticed in both the left and right orbital roofs.

14.6. Summary

Adult, possibly male, 20-34 years, 159.01 ± 4.31 cm tall. Linear enamel hypoplasia, peri-mortem fractures, porotic hyperostosis and cribra orbitalia were present.

15. A1010

15.1. Age

The upper and lower third molars were in full occlusion and showed some occlusal weathering, suggestive of an adult age at death. Epiphyseal closure also suggested an adult age with the complete fusion of all long bone epiphyses as well as the medial ends of the clavicles and S1 and S2. This suggested an age older than 30 years. Some cranial suture closure was observed although most of the sutures were still open, suggesting a young adult age. Due to poor preservation no other techniques could be used here. A final age estimation of 30-40 years at the time of death was given for this individual. This would fall within the 20-34 year age category as provided by Falys and Lewis (2010).

15.2. Sex

The morphological features of the skull and pelvis were consistent with features associated with males. The skull presented with a sloped forehead and relatively pronounced supraorbital margins, rounded orbital margins, a very well-developed nuchal crest and a prominent mental eminence. The pelvis presented with a narrow greater sciatic notch and the absence of a pre-auricular sulcus which is also consistent with male individuals. Metric analysis, Function 1 (0.404827) and Function 2 (13.013908), both fell well within the limits

for male individuals. This individual was therefore classified as male. Molecular sex results could however not be obtained for this individual (Gibbon *et al.*, 2010).

15.3. Stature

The stature was calculated using the maximum lengths of the femur and tibia. A stature of 166.99 ± 3.91 cm was obtained.

15.4. Dentition

Almost all the teeth were present except for the upper right central incisor which was lost post-mortem. Occlusal weathering of the upper and lower molars and premolars were observed. Dental pathology included thin bands of dental calculus on the lingual surfaces of the lower central and lateral incisors and severe linear enamel hypoplasia on the upper and lower central and lateral incisors, canines and premolars. The latter defects presented as multiple lines.

15.5. Trauma and pathology

No trauma was observed. Pathology that could be observed included slight medial lateral bowing of the left and right fibula as well as slight periostitis on the left and right tibias (laterally).

15.6. Summary

Adult male, 20.34 years, 166.99 ± 3.91 cm. Dental calculus, linear enamel hypoplasia, bowing of fibulae and periostitis were observed.

16. A1011

16.1. Age

The upper left and the lower left and right third molars were in complete occlusion and showed no occlusal weathering. The upper right third molar was absent possibly as a result of agenesis. The complete eruption of the third molars suggested an adult age. All the long bone epiphyses were completely fused, suggesting an age older than 20 years. The medial ends of the clavicles were not yet completely fused, suggesting an age younger than 30 years. The spheno-occipital synchondrosis still presented with a fusion line suggesting an age younger than 25 years at the time of death. A final age estimate of 20-25 years at the time of death was given here. This would fall within the 20-34 year age category as suggested by Falys and Lewis (2010).

16.2. Sex

The morphological characteristics observable on the skull and pelvis were consistent with those features associated with males. The skull presented with a sloped forehead, a prominent glabella, rounded orbital margins and large mastoid processes. The supraorbital margin and

the nuchal crest were less distinct. The pelvis presented with a narrow greater sciatic notch and the absence of any pre-auricular sulcus. Overall the skeletal remains were fairly robust and presented with marked muscle attachments suggestive of someone of more masculine build. Metric assessment using measurements obtained from the femur also suggested a male sex with Function 1 (0.389081) falling within the limits for male individuals. This individual was therefore classified as male. No molecular sex results could be obtained for this individual (Gibbon *et al.*, 2010).

16.3. Stature

The stature was determined by using the maximum length of the tibia. A stature of 158.16 ± 3.87 cm was obtained.

16.4. Dentition

Almost all the teeth were present except for the upper right third molar which was absent due to agenesis. The lower left lateral incisor was lost post-mortem. Dental pathology included thin bands of dental calculus on the lingual surfaces of the upper and lower central and lateral incisors. Multiple linear enamel hypoplasia defects were also noted on the lower right premolars and canine. These defects presented as multiple lines.

16.5. Trauma and pathology

Trauma that could be observed included a peri-mortem butterfly fracture of the left femur as well as a possible peri-mortem compression fracture of T4. These fractures showed no signs of healing. Pathology observed included porotic hyperostosis (degree score 1; healed) on the left and right parietal as well as the occipital bones concentrated in the region of the lambdoid suture. Slight medial lateral bowing of the left and right ulna, tibia and fibula was observed with slight periostitis on the tibia (medially). Partial sacralisation of L5 was also observed.

16.6. Summary

Adult male, 20-34 years, 158.16 ± 3.87 cm tall. Peri-mortem fractures, dental calculus, linear enamel hypoplasia, porotic hyperostosis, bowing of ulna, tibia and fibula, periostitis, and partial sacralisation of L5 were present.

17. A1012

17.1. Age

The upper right third molar was in full occlusion, suggesting an adult age at death. The upper and lower first and second molars showed some occlusal weathering with small dentine patches suggestive of a possible older adult age. All the visible epiphyses were completely fused including the spheno-occipital synchondrosis which suggest an age older than 30 years. Degenerative changes were observed on the proximal left and right tibias as well as the talus, again suggesting an older adult age possibly older than 35 years. A final age estimate of 35-

50 was therefore given here. This would fall within the 35-45 year age category as suggested by Falys and Lewis (2010).

17.2. Sex

Due to the poor preservation of the remains only morphological characteristics could be used to determine sex. The only morphological features that could be observed were the mastoid processes which were large, with a well-developed nuchal crest and mental eminence, and a narrow greater sciatic notch. These characteristics suggested a male sex. No metric analysis could be done. Due to the poor preservation of the remains no results could be obtained by the molecular sex study done by Gibbon *et al.* (2010). This individual was therefore tentatively assigned as being male.

17.3. Stature

The remains of this individual were too badly preserved to calculate stature.

17.4. Dentition

Most of the maxillary teeth were missing as a result of post-mortem tooth loss except for the left third molar which was either lost ante-mortem or possibly as a result of agenesis (poor preservation made it difficult to observe the alveolar bone for scarring). Maxillary teeth that were present included the right second molar and first premolar as well as the left second premolar and first and second molars. The mandibular teeth were more complete and the only teeth that were missing was the left lateral incisor which was lost post-mortem as well as the left and right third molars which seems to have been genetically absent as there was no indication of alveolar scarring suggesting ante-mortem loss. Occlusal wear was observed on all the molars and premolars with small dentine patches. Alveolar regression observed may indicate the presence of periodontal disease. Linear enamel hypoplasia was noted on the lower left and right molars, premolars and canines. These defects presented as multiple lines.

17.5. Trauma and pathology

No trauma could be observed. The only pathology that could be observed was osteophytic lipping of the proximal left and right tibia and talus.

17.6. Summary

Adult, possibly male, 35-45 years. Linear enamel hypoplasia, possible periodontal disease and osteophytic lipping were observed.

18. A1013

18.1. Age

The upper and lower third molars were in complete occlusion with minimal occlusal wear, suggesting a young adult age. All the visible epiphyses were completely fused including the

spheno-occipital synchondrosis which further indicates an adult age older than 30 years. No degenerative changes could be observed, suggesting an age younger than 35 years. No other techniques could be employed here due to poor preservation. A final age estimate of 30-35 years was given for this individual which would correspond with the 20-34 age at death category as provided by Falys and Lewis (2010).

18.2. Sex

The morphological features of the skull and pelvis were consistent with that of a male. The skull presented with a sloped forehead, a prominent glabella and rounded orbital margins, whereas the pelvis presented with narrow greater sciatic notches. Metric analysis confirms this with femur dimensions measurements, Function 1 (0.102118) and Function 2 (1.172528), falling within the limits for males. No molecular sex results could be obtained for this individual (Gibbon *et al.*, 2010). This individual was therefore classified as male.

18.3. Stature

Stature was determined by using the maximum lengths for the femur and tibia. A stature of 166.33 ± 3.91 cm was obtained.

18.4. Dentition

Most of the teeth were present except for the upper right first molar, first and second premolars, canine and central and lateral incisors as well as the upper left central and lateral incisors. As for the mandibular teeth the left and right central incisors, the left lateral incisor and left canine were missing. All missing teeth were lost post-mortem. Slight occlusal wear of the upper and lower first and second molars were observed and presented with small dentine patches on the first molars. Thin bands of dental calculus were noted on the buccal surfaces of the maxillary molars along with alveolar regression throughout possibly suggestive of periodontal disease. Linear enamel hypoplasia was observed on the upper and lower canines and premolars and presented as multiple lines.

18.5. Trauma and pathology

Possible peri-mortem compression fractures of L3-L5 were observed. No healing could be observed. Slight periostitis was also observed on the left and right femur and tibia (medially and laterally).

18.6. Summary

Adult male, 20.34 years, 166.33 ± 3.91 cm tall. Linear enamel hypoplasia, dental calculus, periodontal disease, peri-mortem fractures and periostitis were present.

19. A1014

19.1. Age

The upper and lower third molars were in full occlusion and presented with no occlusal wear, suggesting a possible young adult age. All visible long bone epiphyses were completely fused along with the medial ends of the clavicles, suggesting an age older than 30 years. Some cranial suture closure could be observed however pathological lesions hampered the use of this technique to some extent. No degenerative changes could be observed, suggesting a possible younger adult age at death. Due to the poor preservation of the remains no other techniques could be employed. A final age estimation of 30-35 was given here which would correspond with the 20-34 year age category of Falys and Lewis (2010).

19.2. Sex

Morphological features observable on the skull included a prominent glabella, well-developed supraorbital margins, rounded orbital margins, large mastoid processes and a well-developed nuchal crest which suggested a male sex for this individual. The pelvic dimensions also presented with male features with a narrow greater sciatic notch and the absence of a pre-auricular sulcus. Metric assessment of the femoral dimensions seemed to confirm this with Function 1 (0.087412) falling within the limits set for male individuals. No molecular sex results could be obtained for this individual (Gibbon *et al.*, 2010).

19.3. Stature

The maximum lengths of the femur and fibula were used to determine stature. A stature of 158.58 ± 3.72 cm was obtained.

19.4. Dentition

None of the mandibular teeth were present due to the absence of a mandible. As for the maxillary teeth the left and right central incisors, the left canine and left first and second premolars were lost post-mortem. The upper right canine was also absent but was lost ante-mortem as suggested by the alveolar scarring. Slight occlusal wear was observed on the left and right first and second molars. Dental pathology included linear enamel hypoplasia on the left and right lateral incisors. These defects presented as multiple lines. Possible periodontal disease was also identified as indicated by the regression of the alveolar bone.

19.5. Trauma and pathology

No trauma could be observed. Porotic hyperostosis (degrees score 1; healed) especially of the parietal and occipital bones were noted.

19.6. Summary

Adult male, 20-34 years, 158.58 ± 3.72 cm tall. Linear enamel hypoplasia, possible periodontal disease and porotic hyperostosis were present.

20. A1015

20.1. Age

The upper and lower third molars were in complete occlusion with no occlusal wear, suggesting a young adult age. Most of the epiphyses were fused except for the medial ends of the clavicles, suggesting an age younger than 30 years as well as the iliac crest which seems to have only recently fused, suggesting an age younger than between 20 and 23 years. A final age estimate of 20-23 was therefore given here. This estimate would fall within the 20-34 year age category as provided by Falys and Lewis (2010).

20.2. Sex

The morphological characteristics of the skull and pelvis were consistent with male features. The skull presented with a prominent glabella and rounded orbital margins whereas the pelvis presented with no subpubic concavity and no ishiopubic ramus ridge. The greater sciatic notch was very narrow and there was no indication of a pre-auricular sulcus. Metric assessment confirmed a male sex for this individual. Discriminant function analysis of femoral dimensions, Function 1 (0.878548) as well as the femoral circumference (86 cm), fell well within the limits for male individuals. Molecular sex results also suggested a male sex for this individual (Gibbon *et al.*, 2010).

20.3. Stature

The stature was determined by using the maximum length of the humerus and radius. A stature of 166.07 ± 4.31 cm was obtained.

20.4. Dentition

Almost all the teeth were present except for the upper left and right central incisors, the upper left lateral incisor, the left third molar and the lower left and right central and lateral incisors. All teeth were lost post-mortem. Dental pathology included dental calculus on the lingual surfaces of the lower left molars, premolars and canine as well as the lower right canine and first premolar. Linear enamel hypoplasia was also noted on the lower left and right canines presenting as multiple lines.

20.5. Trauma and pathology

Trauma that could be observed included a peri-mortem spiral fracture of the right humerus, and peri-mortem butterfly fractures of the left femur and tibia. None of these fractures presented with healing. Pathology included possible porotic hyperostosis (degree score 1; healed) especially of the parietal and occipital bones.

20.6. Summary

Adult male, 20-34 years, 166.07 ± 4.31 cm tall. Linear enamel hypoplasia, dental calculus, peri-mortem fractures, and possible porotic hyperostosis were observed.

21. A1016

21.1. Age

All the visible long bone epiphyses were completely fused, suggesting and adult age for this individual. The upper and lower first molars showed a relative degree of occlusal weathering with dentine exposure, suggesting a possible older adult age. The second and third molars were lost post-mortem and could not be used here. A relative degree of cranial suture closure could be observed, suggestive of a middle adult age, although pathology and poor preservation hampered the optimal use of this technique. Slight degenerative changes could be observed on the ulna and tibia further, suggesting an older adult age. The age estimation given here was between the ages of 30 and 50 at the time of death. This would fall within the 35-45 year age category given by Falys and Lewis (2010).

21.2. Sex

The observable features of the skull and pelvis were consistent with that of a male. The glabella and nuchal crest were quite prominent whereas the greater sciatic notch was very narrow. The pre-auricular sulcus was absent on both os coxae. Metric analysis of the femoral dimensions confirmed a male sex with Function 1 (0.281235) as well as the femoral midshaft circumference (87 cm) falling within the limits for males. This corresponds with the molecular sex results obtained for this individual (Gibbon *et al.*, 2010).

21.3. Stature

The stature was calculated by using the maximum lengths of the femur and tibia. A stature of 168.60 ± 3.25 cm was obtained.

21.4. Dentition

Most of the teeth were present except for the upper and lower left and right second and third molars, the upper right central and lateral incisors, and the lower right central incisor which were all lost post-mortem. Some occlusal weathering with small dentine patches could be observed on the first molars. The upper right first molar also presented with a small carious lesion on the occlusal surface towards the medio-lingual quadrant. Thin bands of dental calculus were noticed on the lingual surfaces of the lower left and right central and lateral incisors and canines. Linear enamel hypoplasia, presenting as multiple lines, were observed on the lower left and right canines.

21.5. Trauma and pathology

No trauma could be observed. Pathology included porotic hyperostosis (degree score 2; active and healed) especially situated on the parietal and occipital bones as well as slight periostitis on both tibiae (medially and laterally).

21.6. Summary

Adult male, 35-45 years, 168.60 ± 3.25 cm. Linear enamel hypoplasia, dental calculus, caries, porotic hyperostosis and periostitis were present.

22. A1017

22.1. Age

All the visible long bone epiphyses were completely fused, suggesting an adult age for this individual, possibly older than 20. The lower right third molar was however not yet in full occlusion, suggesting a younger age. The spheno-occipital synchondrosis presented with a fusion scar, suggesting an age younger than 25 years. Due to the poor preservation of the remains no other techniques could be used here. A final age estimate of 20-25 years was given here and would fall within the 20-34 year age category provided by Falys and Lewis (2010).

22.2. Sex

The few morphological features that could be observed seem to have suggested a male sex for this individual. Overall the skeletal remains were quite robust and presented with marked muscle attachments. The skull presented with a somewhat sloped forehead, rounded orbital margins and a square shaped chin whereas the pelvis presented with a very narrow greater sciatic notch. No metric analysis could be done due to the poor preservation of the remains. Poor preservation also meant that no molecular sex results could be obtained for this individual. This individual was therefore only tentatively assigned as male based on the observable morphological features.

22.3. Stature

The stature was determined by using the maximum length of the tibia. A stature of 161.49 ± 3.87 cm was obtained for this individual.

22.4. Dentition

Most of the teeth were present except for the upper left and right third molars, the upper right central and lateral incisor, the upper left lateral incisor, the lower left third molar, the lower left central and lateral incisors, and the lower right canine which were lost post-mortem. Dental pathology included thin bands of dental calculus on the buccal surfaces of the upper and lower molars and linear enamel hypoplasia on the lower left and right premolars. The latter presented as multiple linear lines.

22.5. Trauma and pathology

No trauma or pathology could be observed.

22.6. Summary

Adult, possibly male, 20-34 years, 161.49 ± 3.87 cm tall. Linear enamel hypoplasia and dental calculus were present.

23. A1018

23.1. Age

The lower right third molar was in full occlusion with no occlusal wear, suggesting a possible young adult age. Most of the cranial sutures were still completely unfused, further suggestive of a young age at death. All the visible epiphyses were completely fused and no degenerative bone changes could be observed. As a result a wide age range estimate of 20-35 years was given which placed this individual in Falys and Lewis (2010) 20-34 year age category.

23.2. Sex

The morphological characteristics of the skull and pelvis, where it was possible to observe these, were consistent with male features. The skull presented with a very pronounced nuchal crest, whereas the mandible was square shaped with a prominent mental eminence. The pelvis presented with very narrow greater sciatic notches and the absence of a pre-auricular sulcus. The only metric analysis that could be done was to compare the femoral midshaft circumference to known standards for males and females. The femoral midshaft circumference (83 cm) fell within the limits for male individuals. Due to the poor preservation of the remains no molecular sex results could be obtained for this individual (Gibbon *et al.*, 2010).

23.3. Stature

The stature was calculated by using the maximum length of the femur. A stature of 164.64 ± 3.48 cm was obtained.

23.4. Dentition

Most of the teeth were lost post-mortem with the only the upper left first and second molars, the lower right first second and third molars, the lower right second premolar, the lower right canine, the lower left second premolar and the lower left second and first molars being present. Slight occlusal wear was observed on the upper and lower molars with two small dentine patches on the lower right first molar.

23.5. Trauma and pathology

No trauma could be observed. The only pathology that could be observed was porotic hyperostosis (degree score 1; healed) on the parietal and especially the occipital bones.

23.6. Summary

Adult male, 20-34 years, 164.64 ± 3.48 cm tall. Porotic hyperostosis was present.

24. A1019

24.1. Age

The lower third molars were not yet in full occlusion, suggesting a young age at death. Most of the long bone epiphyses were fused except for the proximal humerus which still presented with a fusion line placing this individual around 20 years at the time of death. The medial ends of the clavicles and S1 and S2 of the sacrum were also still unfused confirming an age younger than 30 years. A final age estimate of 20 to 25 years was given here correlating with the 20-34 year age category from Falys and Lewis (2010).

24.2. Sex

Overall the skeletal remains were quite robust and presented with marked muscle attachments. The skull presented with a well-developed nuchal crest and a prominent mental eminence. The right pubic bone did not present with any ventral arch or subpubic concavity whereas the right and left os coxa presented with a very narrow greater sciatic notch and the absence of a pre-auricular sulcus suggesting a male sex for this individual. Metric analysis confirmed a male sex with discriminant function analysis of the femoral measurements specifically Function 1 (1.3628604) and Function 2 (1.097009) falling within the limits for male individuals. No molecular sex results could be obtained for this individual (Gibbon *et al.* 2010).

24.3. Stature

The stature was determined by using the maximum lengths of the femur and tibia. The stature was calculated to be 165.23 ± 3.91 cm.

24.4. Dentition

Almost all the teeth were present except for the upper right third molar, the upper right second premolar, the upper left and right central incisors, the lower left and right third molars, and the lower left canine. All of these teeth were lost post-mortem except for the lower left and right third molars which were not yet in occlusion. Slight occlusal wear of the upper and lower first molars was noted. Dental pathology that could be observed included dental calculus on the buccal surfaces of the upper molars and the lower premolars, canines and incisors. The lower left and right premolars and the lower right canine presented with linear enamel hypoplasia which presented as multiple lines.

24.5. Trauma and pathology

No trauma could be observed. The only pathology that could be observed was periostitis of the left and right femur and tibia (medially) and proximally in the right tibia.

24.6. Summary

Adult male, 20-34 years, 165.23 ± 3.91 cm tall. Linear enamel hypoplasia, dental calculus and periostitis were present.

25. A1020

25.1. Age

The lower right third molar was not completely erupted, suggesting a young age for this individual. Epiphyseal union confirmed this with the incomplete fusion of the proximal head of the humerus, the proximal epiphyses of the tibia and the distal epiphyses of the femur. The fusion times for these areas range between 16-21 years. A final age estimate of 16-20 was given for this individual. This would correspond with the >20 years age category provided by Falys and Lewis (2010).

25.2. Sex

Overall the remains were quite robust with marked muscle attachments, suggesting someone of more masculine build. The observable morphological features of the skull and pelvis were consistent with features associated with males. The skull presented with a sloped forehead and a prominent mental eminence whereas the pelvis presented with narrow greater sciatic notches and no pre-auricular sulcus. Due to the poor preservation of these remains no metric analysis could be done. No molecular sex results could be obtained (Gibbon *et al.*, 2010). This individual was therefore tentatively assigned as male based on the observable morphological features.

25.3. Stature

Due to the poor preservation of the remains stature could not be determined here.

25.4. Dentition

None of the maxillary teeth were present. Most of the mandibular teeth were present except for the left and right central incisors, the left lateral incisor, canine and third molar. Absent teeth were all lost post-mortem. The only dental pathology that could be observed was multiple linear enamel hypoplasias on the left premolars.

25.5. Trauma and pathology

No trauma could be observed. The left femur presented with periostitis to the lateral surface towards the midshaft.

25.6 Summary

Adolescent, possible male. Linear enamel hypoplasia and periostitis were observed.

26. A1021

26.1. Age

The upper and lower left third molars were in full occlusion and presented with slight occlusal wear, suggesting an adult age for this individual. The upper and lower second and first molars showed more extensive occlusal wear with large dentine patches, possibly suggestive of an older adult. All epiphyses were completely fused including the medial ends of the clavicles confirming an age older than 30 years. A tentative final age estimation of between 35 and 45 was given for this individual in correspondence with Falys and Lewis' (2010) age category.

26.2. Sex

Overall the remains were robust with well-developed muscle attachments, suggestive of a possible male sex. The observable morphological features of the skull and pelvis seem to confirm this. The skull presented with a very prominent nuchal crest, a somewhat sloped forehead and a well-defined mental eminence whereas the pelvis presented with narrow greater sciatic notches and the absence of any pre-auricular sulcus. No metric analysis could be done due to the poor preservation of the remains. This individual was therefore tentatively assigned as male. Due to poor preservation this individual was not sampled for molecular sex analysis (Gibbon *et al.*, 2010).

26.3. Stature

Due to the poor preservation of the remains stature could not be determined here.

26.4. Dentition

A lot of teeth were lost post-mortem including the upper left and right central and lateral incisors and canines as well as the upper left first premolar and the right third molar, the lower left and right central and lateral incisors, the left and right canines and the left first premolar. The lower right third molar was also absent but seems to have been lost ante-mortem as indicated by a slight alveolar scar. Occlusal wear was observed on the upper and lower molars with large dentine patches visible on the upper and lower first molars. Thin bands of dental calculus could be observed on the buccal surfaces of the lower left and right molars.

26.5. Trauma and pathology

No trauma could be observed. Possible porotic hyperostosis (degree score 1; healed) could be observed on the occipital bone.

26.6. Summary

Adult, possible male, 35-45 years. Dental calculus and possible porotic hyperostosis were present.

27. A1022

27.1. Age

The upper and lower third molars were in complete occlusion and presented with no occlusal wear, suggesting an age older than 18 years at the time of death. All the visible epiphyses were fused except for the medial ends of the clavicles, suggesting an age younger than 30 years. The degree of cranial suture closure also confirmed a young adult age for this individual. A final age estimate of 20-25 years was given here which would fall within the 20-34 year age category suggested by Falys and Lewis (2010).

27.2. Sex

The morphological features of the skull and pelvis were consistent with that observable in males. The skull presented with a relatively prominent supraorbital margin, a rounded orbital margin, a sloped forehead, a large mastoid process, a prominent nuchal crest and a well-defined mental eminence. The pelvis presented with a narrow greater sciatic notch and the absence of any pre-auricular sulcus. Metric analysis also suggested a male sex for this individual. Discriminant function analysis of the femoral dimensions, specifically Function 1 (0.278387) and Function 2 (1.228981), fell well within the limits for male individuals. No molecular sex results could be obtained for this individual for comparison (Gibbon *et al.*, 2010).

27.3. Stature

The stature was calculated by using the maximum lengths of the femur and tibia. A stature of 163.25 ± 3.91 cm was obtained.

27.4. Dentition

Almost all the teeth were present except for the upper left third molar which was lost ante-mortem. Severe coronal caries was observed in the upper right second molar affecting the whole crown and extending into the alveolar bone as an abscess. Small bands of dental calculus were observed on the lower left and right central and lateral incisors on the buccal surfaces. Multiple linear enamel hypoplasia lesions were observed on the upper left and right canines and the lower left and right canines and first premolars.

27.5. Trauma and pathology

No trauma could be observed. Observable pathology included possible porotic hyperostosis (degree score 1; healed) situated on the parietal and occipital bones and possible slight cribra orbitalia (degree score 1; healed) in the right orbital roof. Periostitis was also observed on the left and right femur situated on the anterior surfaces of the midshaft and possible osteomyelitis in the right calcaneus. The left and right fibula presented with slight medial lateral bowing.

27.6. Summary

Adult male, 20-34 years, 163.25 ± 3.91 cm tall. Linear enamel hypoplasia, dental calculus, caries, possible porotic hyperostosis and possible cribra orbitalia were present.

28. A1023

28.1. Age

The lower right third molar was in full occlusion with very slight occlusal wear, suggesting an adult age at death. The upper and lower first and second molars presented with moderate occlusal wear with dentine exposure of especially the first molars. This may be suggestive of someone of middle adult age. All the visible epiphyses also showed complete fusion, including the medial ends of the clavicles, the spheno-occipital synchondrosis and S1 and S2 of the sacrum, suggesting an age older than 30 years. Degenerative changes could be observed in the area of the calcaneus, further suggesting a possible middle adult age for this individual. The degree of cranial suture closure suggested an age range of between 30 and 60 years of age at the time of death. A final age estimate of between 30 to 40 years was given placing this individual in either the 20-34 or 35-45 year age categories as provided by Falys and Lewis (2010). Here the 20-34 age category was used since it is known from historical records that most of the Chinese were young adults between 20-30 years.

28.2. Sex

The morphological features of the skull and pelvis were consistent with someone of male sex. The skull presented with a prominent glabella, a sloped forehead, a prominent supraorbital margin, rounded orbital margins, medium sized mastoid processes, a well-developed nuchal crest and a prominent mental eminence. The pelvis presented with very narrow greater sciatic notches and the absence of any pre-auricular sulcus. Metric analysis of the femoral dimensions however suggested a female sex with Function 1 (-0.247856) falling within the limits for female individuals. No molecular sex results could be obtained for this individual (Gibbon *et al.*, 2010). The morphological features were however quite distinctly male and therefore this individual were tentatively classified as male.

28.3. Stature

Stature was determined by using the maximum length of the tibia. A stature of 157.93 ± 3.87 cm was obtained.

28.4. Dentition

Almost all the teeth were present except for the upper left and right third molars, the upper left and right central incisors, the upper left lateral incisor, the lower left central incisor and the lower left third molar. Slight occlusal wear could be observed on the upper and lower first and second molars with small dentine patches on the lower first molars. Linear enamel hypoplasia was also noted on the lower left and right canines and presented as multiple lines.

28.5. Trauma and pathology

The only trauma that could be observed was a possible peri-mortem spiral fracture of the left femoral shaft. This fracture showed no signs of healing. Pathology that could be observed included porotic hyperostosis (degree score 1 to 2; healed), specifically concentrated on the occipital bone and extending towards the parietal bones. Schmorl's nodes were noted on T8-T10 with unilateral spondylolysis of L5. Possible osteomyelitis was observed in the left distal femur.

28.6. Summary

Adult, possible male, 20-34 years, 157.93 ± 3.87 cm tall. Linear enamel hypoplasia, peri-mortem fracture, porotic hyperostosis, Schmorl's nodes, spondylolysis and possible osteomyelitis were present.

29. A1024

29.1. Age

The remains of this individual were very poorly preserved and the only diagnostic features that could be used to determine age at death were the presence of the third molars and the fusion of the medial end of the clavicle. The third molars presented with slight occlusal wear suggesting complete eruption of the third molars before death, suggesting an age older than 18. The medial end of the clavicle was completely fused, suggesting an age older than 30 years. Osteophytic lipping was observed on the left proximal ulna, suggesting a possible middle adult age. A final age estimate of 30-40 years was provided here. This may either fall within the 20-34 or the 35-45 year age category as suggested by Falys and Lewis (2010). Here the 20-34 age category was used since it is known from historical records that most of the Chinese were young adults between 20-30 years.

29.2. Sex

The morphological features of the skull and pelvis could not be assessed due to its poor preservation or absence. The only features that could be assessed were the upper left arm which presented with a robust build with marked muscle attachments. The distal humerus presented with characteristically male features with a triangular shaped olecranon fossa and a right angle for the medial epicondyle. Due to the very poor preservation of this individual's remains no other techniques could be used. This individual was also excluded from the molecular sex (Gibbon *et al.*, 2010) study due to its poor preservation. This individual was therefore only tentatively assigned as male based on the observable morphological features of the upper left arm.

29.3. Stature

Stature could only be determined from the maximum length of the humerus. A stature of 154.46 ± 4.13 cm was obtained for this individual.

29.4. Dentition

Almost all the teeth were lost post-mortem. The only teeth present were the upper left second premolar, the upper left second and third molar, and the lower left central and lateral incisors. The lower incisors presented with very slight dental calculus situated on the lingual surfaces whereas the upper left second and third molars presented with coronal caries.

29.5. Trauma and pathology

No trauma could be observed. Degenerative changes were observed in the distal ulna presenting as osteophytic lipping of the trochlear notch.

29.6. Summary

Adult, possibly male, 20-34 years, 154.46 ± 4.13 cm tall. Dental calculus, caries and osteophytic lipping were observed.

30. A1025

30.1. Age

The remains of this individual were very badly preserved and the only age estimate that could be given here was an adult age. This was based on the fact that all the observable epiphyses were fused and that the one third molar presented with complete apical development.

30.2. Sex

Again the poor preservation of the remains hampered the determination of sex. Overall the skeletal remains were quite robust and presented with marked muscle attachments consistent with someone of a more masculine build. The only observable morphological features were the greater sciatic notch which was very narrow and the pre-auricular sulcus which was absent. These characteristics are consistent with someone of male sex. Metric analysis could not be done here due to the poor preservation. This individual was also excluded in the study by Gibbon *et al.* (2010) and therefore no molecular sex results could be used for comparison here. This individual was therefore only tentatively assigned as male.

30.3. Stature

Due to the poor preservation of the remains stature could not be determined here.

30.4. Dentition

The fragmented remains of only 12 teeth could be observed and included a possible upper left first molar, an upper second and first premolar, canine and lateral incisor, a possible lower central and lateral incisor, a first and second premolar and a third molar. The only dental pathology that could be observed was linear enamel hypoplasia on the upper and lower canine. These defects presented as multiple lines.

30.5. Trauma and pathology

No trauma was observed. Possible medio-lateral bowing of subtrochanteric region of especially the right femur was observed.

30.6. Summary

Adult, possibly male. Linear enamel hypoplasia and possible bowing of femur were present.

31. A1026

31.1. Age

Due to the poor preservation of the remains the only age estimate that could be provided for this individual was an adult age based on the complete fusion of all the visible epiphyses and the slight osteophytic lipping of the right talus.

31.2. Sex

Overall the remains were very robust and presented with marked muscle attachments. The morphological features of the pelvis were consistent with that associated with males. The pelvis presented with narrow greater sciatic notches and an absence of a pre-auricular sulcus. The sacrum was long and narrow with an evenly distributed curvature suggestive of a male individual. Due to the poor preservation of the remains metric analysis could not be done. This individual was also excluded from the study by Gibbon *et al.* (2010) due its poor preservation. This individual was therefore only tentatively assigned as male.

31.3. Stature

The stature was determined by using the maximum length of the tibia. A stature of 163.71 ± 3.87 cm was obtained.

31.4. Dentition

No teeth were present.

31.5. Trauma and pathology

No trauma could be observed. Degenerative pathology in the form of osteophytic lipping of the right talus was observed.

31.6. Summary

Adult, possibly male, 163.71 ± 13.87 cm tall. Osteophytic lipping was present.

32. A1027

32.1. Age

The only age estimate that could be provided for this individual was an adult age. This was based on the complete fusion of all visible epiphyses.

32.2. Sex

The poor preservation of the remains hampered the determination of sex. The only diagnostic features that could be used were the overall skeletal build and the morphological features of the pelvis. Overall the skeletal remains were very robust, representative of a tall and masculine individual. The pelvis presented with narrow greater sciatic notches and an absence of any pre-auricular sulcus. These features are consistent with male individuals. No metric analysis could be done and no molecular sex results could be obtained for this individual (Gibbon *et al.*, 2010). This individual was therefore only tentatively assigned a male.

32.3. Stature

Due to the poor preservation of the remains stature could not be determined here.

32.4. Dentition

No teeth were present.

32.5. Trauma and pathology

No trauma was observed. Slight medio-lateral bowing of the left and right tibia was observed.

32.6. Summary

Adult, possibly male. Bowing of left tibia was observed.

33. A1028

33.1. Age

Due to the poor preservation the only age estimate that could be provided for this individual was an adult age at death. This was based on the fact that all the visible long bone epiphyses were completely fused. The first molar also presented with substantial occlusal wear also suggestive of an adult age.

33.2. Sex

Overall the remains were very robust with marked muscle attachments. The pelvis presented with narrow greater sciatic notches and an absence of a pre-auricular sulcus consistent with

male features. The only metric analysis that could be used was the femur midshaft circumference (81 mm) which fell well within the limits for male individuals. Molecular sex results also suggested a male sex for this individual (Gibbon *et al.* 2010).

33.3. Stature

Stature was determined by using the maximum length of the femur. A stature of 156.82 ± 3.48 cm was obtained.

33.4. Dentition

Only three teeth could be identified and included an upper canine, a lower second premolar and a lower first molar (all unsided).

33.5. Trauma and pathology

No trauma was observed. The only pathology that could be observed was slight periostitis on the left and right femur and tibia (medially).

33.6. Summary

Adult male. Periostitis was observed.

34. A1029

34.1. Age

The upper and lower molars and premolars showed extensive occlusal wear with large dentine patches, suggestive of an adult age, possibly middle adult age. All the epiphyses were fused including the medial ends of the clavicle and the spheno-occipital synchondrosis which suggested an age older than 30 years at the time of death. The degree of cranial suture fusion suggested an age range of between 27 and 45 years at the time of death. A final age estimate of 30-45 was given for this individual which would fall within the 35-45 year age category provided by Falys and Lewis (2010).

34.2. Sex

The morphological features of the skull and pelvis were consistent with that of a male. The skull presented with a prominent supraorbital margin, rounded orbital margins, a prominent nuchal crest, medium sized mastoid processes and a well-defined mental eminence. The pelvis presented with narrow greater sciatic notches and the absence of a pre-auricular sulcus. Metric analysis of the femoral dimensions, however, suggested a female sex for Function 2 (-0.338221). The poor preservation of the periosteum may however have influenced the measurements obtained here. This individual's remains were excluded from the molecular sex study due to its poor preservation. Based on the definite male morphological characteristics observed in the skull and pelvis this individual was tentatively assigned to be male.

34.3. Stature

The stature was obtained from using the maximum length of the humerus. A stature of 158.64 ± 3.91 cm was obtained.

34.4. Dentition

Almost all the teeth were present except for the upper and lower left and right third molars, the upper left second molar, the upper left and right central incisors, the lower right canine, and the lower right central incisor. All these teeth were lost post-mortem except for the lower right third molar, which seems to have been lost ante-mortem as suggested by the alveolar regression, and the lower left third molar which was either lost very early on in life with no alveolar involvement being visible any more or it was absent due to agenesis. Substantial occlusal wear was observed on the upper and lower molars and premolars with large patches of dentine exposure. Dental pathology included dental calculus on the lingual surfaces of the upper canines and the buccal surfaces of the upper and lower molars and premolars. Alveolar regression in the maxilla was possibly suggestive of periodontal disease. Coronal caries were observed on the lower left second molar as well as the upper right second molar which extended into the alveolar bone as an abscess. Multiple linear enamel hypoplasia defects were observed on all of the mandibular teeth.

34.5. Trauma and pathology

No trauma could be observed. Possible cribra orbitalia (degree score 2; healed) was observed in the left and right orbital roofs.

34.6. Summary

Adult possible male, 35-45 years, 158.64 ± 3.91 cm tall. Linear enamel hypoplasia, dental calculus, caries, possible periodontal disease, ante-mortem tooth loss and cribra orbitalia were present.

35. A1030

35.1. Age

The upper and lower third molars were in complete occlusion and presented with no occlusal wear, suggesting an adult age. All the long bone epiphyses were completely fused, also suggestive of an adult age. The medial ends of the clavicles and S1 and S2 of the sacrum and were unfused and the spheno-occipital synchondrosis presented with a scar line, suggesting an age younger than 25 years at the time of death. A final age estimate of between 20 and 25 was given for this individual and would therefore fall within the 20-34 year age category set by Falys and Lewis (2010).

35.2. Sex

The morphological features of the skull and pelvis were consistent with someone of male sex. The skull presented with a sloped forehead, a prominent glabella and supraorbital margin,

rounded orbital margins, large mastoid processes, and a well-developed nuchal crest and mental eminence. The pelvis presented with a narrow greater sciatic notch and no pre-auricular sulcus. Metric analysis of the femoral measurements was inconclusive with Function 1 (-0.025676) suggesting female sex whereas Function 2 (0.69854) suggested a male sex. The molecular sex results (Gibbon *et al.*, 2010) for this individual, however, also suggested a male sex and therefore this individual was classified as male.

35.3. Stature

The stature was calculated by using the maximum length of the femur and tibia. A stature of 163.14 ± 3.91 cm was obtained.

35.4. Dentition

Most of the teeth were present except for the upper central and lateral incisors, the upper right third molar, the upper right first premolar, the upper left second premolar, the lower central and lateral incisors, the lower right premolars, and the lower left canine which were lost post-mortem. Occlusal wear with small dentine patches were noted on the upper and lower first molars. Possible periodontal disease as suggested by the regression of the alveolar bone was also observed.

35.5. Trauma and pathology

A possible peri-mortem wedge fracture of T12 and a peri-mortem spiral fracture of the right tibia were observed. An ante-mortem compression fracture was also observed on the left parietal. Pathology included porotic hyperostosis (degree score 1 to 2; healed) of especially the parietal and occipital bones, spina bifida occulta, and a notochord defect in T7 to T11.

35.6. Summary

Adult male, 20-34 years, 163.14 ± 3.91 cm tall. Peri-mortem and ante-mortem fractures, porotic hyperostosis, spina bifida occulta and notochord defects were present.

36. A1031

36.1. Age

The upper and lower third molars were in complete occlusion, suggesting an age older than 18 years. The medial ends of the clavicles were unfused along with S1 and S2 of the sacrum, suggesting an age younger than 25 years at the time of death. The epiphyseal line was also still visible on the proximal humerus, suggesting an age of around 21 at the time of death. A final age estimate of between 20 and 25 was given for this individual. That would fall within the 20-34 year age category provided by Falys and Lewis (2010).

36.2. Sex

Overall the skeletal remains were robust with marked muscle attachments, suggestive of a more masculine build. The skull presented with a sloped forehead, whereas the sacrum was long and narrow with a gradual curvature suggestive of a male individual. Metric analysis of the femoral dimensions Function 1 (0.9566) also suggested a male sex for this individual whereas humeral dimensions Function 2 (-0.388268) suggested a female sex. Unfortunately no molecular sex results could be obtained for this individual (Gibbon *et al.*, 2010). Based on the morphological and femoral discriminant function analysis this individual was classified as male.

36.3. Stature

The stature was calculated using the maximum lengths of the femur and tibia. A stature of 164.24 ± 3.91 cm was obtained.

36.4. Dentition

Most of the maxillary teeth were lost post-mortem except for the upper right second and third molar. Almost all the mandibular teeth were present except for the central and lateral incisors which were also lost post-mortem. Dental pathology included small bands of dental calculus on the lingual surfaces of the lower left and right molars as well as multiple linear enamel hypoplasia defects on the lower canines.

36.5. Trauma and pathology

An ante-mortem fracture of the left foot was observed and affected the first metatarsal and associated medial cuneiform. Periostitis was identified on the femur and tibia (medially). Schmorl's nodes were identified on L1 to L4 with notochord defects of T10 to T12.

36.6. Summary

Adult male, 20-34 years, 164.24 ± 3.91 cm tall. Dental calculus, linear enamel hypoplasia, ante-mortem fracture, periostitis, Schmorl's nodes and notochord defects were present.

Table 1: Skeletal inventory of 36 Chinese miner's skeletons
*** damaged and fragmented**

Skeletal element	Chinese A996	Chinese A997	Chinese A998	Chinese A999	Chinese A1000	Chinese A1001	Chinese A1002	Chinese A1003	Chinese A1004
Skull	1*	1*	1	1	1	1*	1*	1	1*
Mandible	1	1*	1	1	1	1*	1	1	1*
Cervical	5	7	7	0	7	1	5	3	4
Thoracic	12	12	7	11	12	12	2	12	9
Lumbar	5	5	5	2	5	5	2	5	5
Sacrum	0	1	1*	1*	1*	1*	1*	1	1*
Manubrium	1	1	0	0	1	0	0	1	0
Sternum	0	0	0	0	1	0	0	1	0
Right os coxae	1*	1*	1*	1*	0	1*	1*	1	1*
Left os coxae	0	1*	1*	0	1*	1*	1*	1	1*
Right clavicle	1	1	1*	1	1	1	0	1	1
Left clavicle	1	1	1*	1	1	1	1	0	1
Right scapula	1*	1*	1*	1	1	1*	0	1	1*
Left scapula	1*	1*	1*	0	1	1*	1*	1*	1*
Right ribs	11	12	3	9	10	10	4	9	12
Left ribs	12	11	3	7	12	10	1	10	8
Right humerus	1	1	1*	0	0	1	1*	1	1*
Left humerus	1	1	1*	0	1	0	1*	0	1*
Right radius	1	1	1*	0	0	1	0	1	1
Left radius	1	1	1*	1*	1	0	1*	0	1*

Right ulna	1	1	0	0	0	1	1*	1	1*
Left ulna	1	1	1*	1*	1	0	1*	0	1*
Right femur	1	1	1*	1*	0	1	1*	1	1
Left femur	1	1	1*	1*	1	1	1*	1	1*
Right tibia	1	1	1*	1*	0	1	1*	1	1*
Left tibia	0	1	1*	1*	1	1	1*	1	1*
Right fibula	0	1	1*	1*	0	1*	1*	1	1*
Left fibula	0	1	0	1*	1	1*	1*	1	1*
Right patella	0	1	0	1	0	0	0	0	1
Left patella	0	0	0	0	0	0	0	0	1
Right carpals	1	1	0	0	0	0	4 (unsided)	1 (two unsided)	8
Left carpals	0	3	0	0	0	0			8
Right metacarpals	3	3	0	0	0	2	7 (unsided)	0	5
Left metacarpals	1	3	0	1	1	0		2	4
Right tarsals	3	4	0	4	1	1 (one unsided)	0	0	5
Left tarsals	1	5	0	1	2	1	0	0	7
Right metatarsals	0	5	0	1	0	2	2 (unsided)	0	3
Left metatarsals	1	3	0	0	0	0		0	5
Phalanges	0	5	0	5	1	3	9	0	15

Skeletal elements	Chinese A1005	Chinese A1006	Chinese A1007	Chinese A1008	Chinese A1009	Chinese A1010	Chinese A1011	Chinese A1012	Chinese A1013
Skull	1*	1*	1*	1*	1*	1*	1	1*	1*
Mandible	1*	1*	1*	1	1*	1*	1*	1*	1*
Cervical	4	1	1	7	4	0	7	2	4
Thoracic	11	10	12	12	12	12	12	0	9
Lumbar	5	5	5	5	5	5	5	0	5
Sacrum	1	1*	1	1	1*	1	1	0	1*
Manubrium	0	0	0	0	1	1	1	0	0
Sternum	0	1	0	1	0	0	1*	0	1*
Right os coxae	1*	1*	1*	1*	1*	1*	1*	1*	1*
Left os coxae	1*	1*	1*	1*	1*	1*	1*	1*	1*
Right clavicle	1*	1* (unsided)	1	1	1	1	1*	1*	1*
Left clavicle	1		1	1	1	1	1	0	1*
Right scapula	1*	0	1*	1	1*	1*	1*	0	1*
Left scapula	1*	1*	1*	1*	1*	1*	1*	0	0
Right ribs	6	4	12	12	11	6	9	0	2
Left ribs	9	4	10	12	12	7	11	0	3
Right humerus	1*	1*	1	0	1*	0	1	0	1*
Left humerus	1*	1*	1	0	1*	0	1*	1*	1*
Right radius	1*	1*	1	1	1	0	1	1*	1*
Left radius	1	1*	1	1	1	0	1*	1*	1
Right ulna	1*	1*	1	1	1*	1*	1	1*	1*
Left ulna	1*	1*	1	1	1*	0	1*	1*	1

Right femur	1	1	1	1	1	1	1	1*	1
Left femur	1	1*	1	1*	1*	1	1*	1*	1
Right tibia	1	1	1	1	1	1	1	1	1
Left tibia	1*	1	1	1	1*	1	1	1*	1
Right fibula	1	1	1	0	1*	1	1	1*	1
Left fibula	1*	1	1	0	1*	1	1*	1*	1*
Right patella	1	0	0	1	0	0	1	0	1
Left patella	1	0	0	1	0	0	1	1	1
Right carpals	0	0	0	8	0	0	6	0	8* (unsided)
Left carpals	1	0	0	8	2	0	7	0	
Right metacarpals	0	2	1	5	2	0	5	0	4
Left metacarpals	0	0	3	5	0	1	5	0	5
Right tarsals	4	2	2	7	7	0	7	1	7
Left tarsals	3	1	2	7	6	0	7	2	7
Right metatarsals	4	0	0	5	5	0	4	2	5
Left metatarsals	0	1	1	5	5	1	3	0	5
Phalanges	1	0	0	22	13	0	15	0	11

Skeletal elements	Chinese A1014	Chinese A1015	Chinese A1016	Chinese A1017	Chinese A1018	Chinese A1019	Chinese A1020	Chinese A1021	Chinese A1022
Skull	1*	1*	1*	1*	1*	1*	1*	1*	1*
Mandible	1*	1*	1*	1*	1*	1*	1*	1*	1
Cervical	2	5	0	0	0	4	two neural arches	3	7
Thoracic	9	12	3	0	0	7	10	7	12
Lumbar	5	5	4	1	0	5	5	5	5
Sacrum	1*	0	1*	1*	0	1	1*	1*	1*
Manubrium	0	0	0	0	0	0	0	0	0
Sternum	0	0	0	0	0	0	0	0	1
Right os coxae	1*	1*	1*	1*	1*	1*	1*	1*	1*
Left os coxae	1*	1*	1*	1*	1*	1*	1*	0	1*
Right clavicle	1	1	1*	0	0	1	1*	1*	1
Left clavicle	1	1	1*	0	0	1*	1*	1*	1
Right scapula	1*	1*	0	1*	1*	1*	0	1*	1
Left scapula	1*	1*	1*	0	1*	0	1*	1*	1
Right ribs	6	3	0	0	ten fragments	6	4	one fragment	11
Left ribs	5	2	0	0		2	2		11
Right humerus	1*	1*	1*	1*	1*	1*	1*	1*	1*
Left humerus	1*	1*	1*	1*	1*	0	0	1*	1*
Right radius	1	1	1*	0	0	1	1*	1*	1
Left radius	1	1	1*	0	0	1	1*	1*	1

Right ulna	1*	1*	1*	1*	0	1*	1*	1*	1
Left ulna	1*	1*	1*	1*	0	1*	1*	1*	1
Right femur	1	1*	1*	1*	1*	1	1*	1*	1
Left femur	1	1*	1*	1*	1*	1	1*	1*	1
Right tibia	1	1*	1	1*	1*	1	1*	1*	1
Left tibia	0	1*	1	1*	1*	1*	1*	1*	1
Right fibula	1	1*	1	1*	0	1*	1*	1*	1
Left fibula	1	1*	1	1*	0	1*	1*	1*	1*
Right patella	0	0	0	1	0	1	1	0	0
Left patella	1	0	0	0	0	1	0	0	1
Right carpals	2	one unsided*	0	0	0	0	seven unsided*	0	1
Left carpals	0		0	0	0	0		0	1
Right metacarpals	5	five unsided*	0	0	0	seven unsided*	six unsided*	0	two unsided*
Left metacarpals	4		0	0	0			0	
Right tarsals	6	nine unsided*	2	ten unsided*	seven unsided*	five unsided*	two unsided*	1	1
Left tarsals	4		2					0	2
Right metatarsals	1	six unsided*	six unsided*	nine unsided*	0	six unsided*	0	0	0
Left metatarsals	0				0		0	0	0
Phalanges	2	6	0	1	0	3	8	0	1

Skeletal elements	Chinese A1023	Chinese A1024	Chinese A1025	Chinese A1026	Chinese A1027	Chinese A1028	Chinese A1029	Chinese A1030	Chinese A1031
Skull	1*	1*	1*	1*	1*	1*	1*	1	1*
Mandible	1*	1*	1*	1*	1*	1*	1*	1	1*
Cervical	7	3	2	2	0	0	7	6	5
Thoracic	12	0	8	0	two neural arches	six neural arches	10	12	12
Lumbar	5	0	1	1	0	2	5	5	5
Sacrum	1	0	0	1*	0	1*	1*	1	1
Manubrium	1	0	0	0	0	0	0	0	1
Sternum	1	0	0	0	0	0	0	0	1
Right os coxae	1*	0	1*	1*	1*	1*	1*	1*	0
Left os coxae	1*	0	0	1*	0	1*	1*	1*	0
Right clavicle	1	1	1*	0	0	0	1	1	1
Left clavicle	1	0	0	0	0	0	1	1	1
Right scapula	1	0	0	0	0	0	1	0	1*
Left scapula	0	0	0	0	0	0	1	1	1*
Right ribs	11	1	fragmented	two fragments	two fragments	fragmented	2	12	10
Left ribs	11	2					1	11	11
Right humerus	1	0	1*	0	0	1*	0	0	1
Left humerus	1*	1	1*	0	1*	1*	1	0	1
Right radius	1	0	1*	0	0	0	0	1	1
Left radius	1	1	1*	0	1*	1*	0	1	1*

Right ulna	1	0	1*	0	0	0	0	1	1
Left ulna	1*	1	1*	0	1*	1*	0	1	1*
Right femur	1	0	1*	0	1*	1*	1*	1	1
Left femur	1*	0	1*	0	1*	1*	1*	1*	1
Right tibia	1	0	1*	0	1*	1*	0	1	1
Left tibia	1*	0	1*	1	1*	1*	1*	1	1
Right fibula	1	0	0	1	1*	0	1*	1*	0
Left fibula	1*	0	0	1*	1*	0	1*	0	1
Right patella	0	1	0	0	0	0	0	0	0
Left patella	0	0	0	1	0	1	0	0	1
Right carpals	0	0	two unsided*	3	0	0	0	0	0
Left carpals	0	2		0	0	0	1	0	0
Right metacarpals	0	0	0	1	0	0	0	0	4
Left metacarpals	0	5	5	0	0	0	0	3	0
Right tarsals	1	0	two unsided*	6	2 (four unsided*)	0	2 (five unsided*)	1 (two unsided*)	thirteen unsided*
Left tarsals	1	3		0	2	0	2		
Right metatarsals	0	0	0	3	one unsided*	0	0	0	5
Left metatarsals	0	4	0	0		0	0	0	5
Phalanges	1	8	2	5	0	0	1	3	7

Table 2: Cranial measurements of the 36 Chinese individuals. Buikstra & Ubelaker (1994)

All measurements in mm.
- skeletal element was absent or damaged and therefore measurements could not be obtained
*** indicates right side**

Skeletal dimension	A996	A997	A998	A999	A1000	A1001	A1002	A1003
Max. cranial length	174	166	198	193	179	174	181	178
Max. cranial breadth	133	139	139	141	149	136	129	139
Bizygomatic diam.	-	-	131	138	147	129	-	139
Basion-bregma height	138	147	154	144	146	142	141	134
Cranial base length	94	96	112	104	104	105	105	100
Basion-prosthion length	91	87	104	102	104	101	92	93
Maxillo-alveolar breadth	62.94	65.65	65.32	63.95	62.63	64.18	62.50	62.58
Maxillo-alveolar length	94.63	48.29	55.05	58.61	53.34	52.38	48.28	51.92
Biauricular breadth	119.17	119.43	123.32	130.52	113.93	116.26	19.72	122.78
Upper facial height	73.63	75.96	78.51	73.83	70.73	71.69	70.83	71.05
Min. frontal breadth	87.20	93.74	98.52	96.68	90.60	86.08	85.75	91.72

Upper facial breadth	94.43	106.75	108.86	105.22	102.09	97.51	95.44	101.71
Nasal height	53.15	53.21	55.18	51.89	49.50	55.30	50.69	50.79
Nasal breadth	25.70	24.85	28.26	25.95	24.24	25.75	23.78	25.39
Orbital breadth	38.73	36.89	38.37	37.32	38.62	37.68	37.47	41.43
Orbital height	34.97	36.64	37.64	35.97	39.45	31.66	40.88	34.05
Biorbital breadth	88.06	97.62	99.59	92.51	93.60	88.79	58.75	94.26
Interorbital breadth	23.71	23.64	29.39	24.41	24.40	24.01	20.51	23.51
Frontal chord	112.98	118.12	119.99	114.62	108.59	108.85	107.89	106.30
Parietal chord	104.48	112.57	129.45	104.46	119.14	110.05	116.75	121.71
Occipital chord	93.75	88.64	101.29	116.80	95.32	98.44	92.47	86.48
Foramen magnum length	35.41	34.73	34.35	37.91	36.31	45.29	41.66	32.14
Foramen magnum breadth	28.01	28.80	28.56	31.89	28.56	32.05	29.35	24.39
Mastoid length	-	33.55	30.21*	24.59	29.88	29.48*	-	26.62
Chin height	33.59	33.54	37.84	35.32	33.93	33.76	31.83	35.68
Height of mandibular body	30.53	30.90	39.29	31.23	32.22	32.76	27.31	34.06

Breadth of mandibular body	11.53	10.31	16.66	17.02	12.66	14.03	11.77	15.26
Bigonial width	97.91	90.19	105.40	107.56	106.52	98.62	95.07	108.71
Bicondylar breadth	104.99	113	116.08	122.46	134.93	110.47	103.89	116.45
Min. ramus breadth	32.35	32.55	35.65	36	35.01	31.60	30.07	32.99
Max. ramus breadth	41.63*	42.29*	44.82	41.19	44.83	42.94*	39.04	43.65
Max. ramus height	63.43	70.78*	71.22	73.25	67.91	69.95*	65.31	64.31
Mandibular length	106	103	113	101	105	110	102	98
Biasterionic breadth	112.57	101.55	115	121	117	107	-	102

Skeletal dimension	**A1004**	**A1005**	**A1006**	**A1007**	**A1008**	**A1009**	**A1010**	**A1011**
Max. cranial length	-	175	166	186	-	169	-	176
Max. cranial breadth	134	140	139	-	-	146	131	137
Bizygomatic diam.	-	-	-	134	133	-	-	127
Basion-bregma height	123	136	-	138	-	142	-	136
Cranial base length	101	103	-	107	103	-	-	97

Basion-prosthion length	102	98	-	88	96	-	-	88
Maxillo-alveolar breadth	61.13	63.93	61.36	60.21	60.71	-	63.38	61.73
Maxillo-alveolar length	49.84	47.79	-	49.61	51.93	-	46.72	48.60
Biauricular breadth	124.26	127.72	120.73	120.59	119.04	-	-	115.97
Upper facial height	76.97	70.13	70.33	75.89	73.86	-	64.29	66.95
Min. frontal breadth	85.60	89.49	87.05	92	93.48	90.62	88.38	89.62
Upper facial breadth	94.41	96.73	95.93	101.45	96.66	-	89.11	95.56
Nasal height	55.07	50.61	53.81	55.69	53.35	-	51.42	46.84
Nasal breadth	22.50	23.68	24.33	24.63	25.30	-	23.40	22.10
Orbital breadth	37.83	37.19	37.44	36.12*	34.64	-	37.92	37.75
Orbital height	39.36	39.75	39.20	41.09*	38.12	-	37.37	40.02
Biorbital breadth	87.38	89.97	86.60	89.57	89.17	-	89.59	85.03
Interorbital breadth	21.10	24.13	19.39	24.69	27.08	-	22.91	15.30
Frontal chord	109.49	111.52	100.28	113.94	-	106.12	-	106.47
Parietal chord	-	100.56	106.60	116.12	-	105.01	-	116.14

Occipital chord	-	94.80	-	92.22	93.46	101.62	89.63	87.57
Foramen magnum length	-	33.08	-	35.68	32.82	33.44	37.62	33.07
Foramen magnum breadth	-	28.89	-	27.08	39.41	31.44	31.28	29.52
Mastoid length	-	20.59	28.97	30.55	36.53	30.47	25.54*	28.50
Chin height	34.23	31.46	30.20	33.31	39.36	36.82	29.60	-
Height of mandibular body	27.75	29.60	31.47	31.64	33.33	31.78	28.82	30.81
Breadth of mandibular body	14.59	14.40	12.88	10.91	14.25	14.01	12.74	11.58
Bigonial width	-	89.81	95.52	100.67	105.55	95.95	-	93.42
Bicondylar breadth	-	110.92	-	-	117.87	112.49	-	-
Min. ramus breadth	34.84	39.96	37.05	32.86*	35.06	33.40	36.55	30.52
Max. ramus breadth	-	48.67*	43.91	45.70*	45.61	43.65*	47.61*	38.40
Max. ramus height	59.17	62..55	63.55	73.50*	68.77	60.07*	63.09	65.44
Mandibular length	-	107	-	102	106	99	-	96
Biasterionic breadth	-	107	-	112	112	-	109	98

Skeletal dimension	A1012	A1013	A1014	A1015	A1016	A1017	A1018	A1019
Max. cranial length	-	-	186	-	-	-	-	-
Max. cranial breadth	-	-	-	-	138	-	-	-
Bizygomatic diam.	-	-	-	-	-	-	-	-
Basion-bregma height	-	-	-	-	-	-	-	-
Cranial base length	-	106	-	-	-	-	-	-
Basion-prosthion length	87	94	-	-	-	-	-	-
Maxillo-alveolar breadth	63.72	63.01	63.64	-	61.56	58.40	-	-
Maxillo-alveolar length	44.61	50.64	54.52	-	48.79	49.66	-	-
Biauricular breadth	121.10	114	-	-	109.62	-	-	-
Upper facial height	-	69.30	76.25	-	68.37	65.33	-	-
Min. frontal breadth	-	89.92	92.39	84.37	-	-	-	-
Upper facial breadth	-	95.12	101.64	-	-	98.44	-	-
Nasal height	-	51.70	55.26	-	45.63	45.43	-	-

Nasal breadth	28.81	23.65	25.55	-	23.90	24.98	-	-
Orbital breadth	-	35.29	39.64	-	37.12	38.77	-	-
Orbital height	-	36.63	36.95	-	39.29	36.25	-	-
Biorbital breadth	-	84.02	91.98	-	87.98	87.70	-	-
Interorbital breadth	-	22.11	21.67	-	21.83	22.18	-	-
Frontal chord	-	-	113.28	-	-	-	-	-
Parietal chord	-	-	120.58	-	-	-	-	-
Occipital chord	89.53	-	-	-	-	-	-	-
Foramen magnum length	29.28	33.46	-	-	-	-	-	-
Foramen magnum breadth	23.96	29.12	-	-	-	-	-	-
Mastoid length	25.52	-	29.81	-	-	-	-	-
Chin height	31.54	31.03	-	-	33.02	33.44	32.76	33.78
Height of mandibular body	29.85	29.31	-	31.59	31.44*	30.52	29.40	32.56
Breadth of mandibular body	12.14	12.33	-	13.09	12.06*	12.29	13.73	9.98

Bigonial width	96.73	-	-	-	-	103.18	-	-
Bicondylar breadth	-	-	-	-	-	-	-	-
Min. ramus breadth	32.43	-	-	30.68	32.37*	34.92	86.00	-
Max. ramus breadth	41.60	-	-	-	-	43.24	-	-
Max. ramus height	67.06	-	-	-	-	61.62	-	-
Mandibular length	99	-	-	-	-	103	-	-
Biasterionic breadth	102	-	-	-	100	-	-	-

Skeletal dimension	**A1020**	**A1021**	**A1022**	**A1023**	**A1024**	**A1025**	**A1026**	**A1027**
Max. cranial length	-	-	-	166	-	-	-	-
Max. cranial breadth	-	-	136	133	-	-	-	-
Bizygomatic diam.	-	-	-	-	-	-	-	-
Basion-bregma height	-	-	140	145	-	-	-	-
Cranial base length	-	-	-	97	-	-	-	-

Basion-prosthion length	-	-	-	91	-	-	-	-
Maxillo-alveolar breadth	-	57.51	57.84	64.60	-	-	-	-
Maxillo-alveolar length	-	45.74	-	44.79	-	-	-	-
Biauricular breadth	-	-	-	117.18	-	-	-	-
Upper facial height	-	73.34	71.30	66.32	-	-	-	-
Min. frontal breadth	-	95.87	-	86.31	-	-	-	-
Upper facial breadth	-	-	-	97.37	-	-	-	-
Nasal height	-	53.94	50.79	47.20	-	-	-	-
Nasal breadth	-	22.22	22.29	25.73	-	-	-	-
Orbital breadth	-	40.36	40.97*	38.52	-	-	-	-
Orbital height	-	42.32	36.38*	38.97	-	-	-	-
Biorbital breadth	-	-	-	90.71	-	-	-	-
Interorbital breadth	-	17.41	17.64	23.20	-	-	-	-
Frontal chord	-	-	-	111.28	-	-	-	-
Parietal chord	-	-	118.95	112.82	-	-	-	-

Occipital chord	-	-	101.67	86.04	-	-	-	-
Foramen magnum length	-	-	37.32	36.48	-	-	-	-
Foramen magnum breadth	-	-	31.63	28.15	-	-	-	-
Mastoid length	-	-	27.17*	21.18	-	-	-	-
Chin height	31.27	-	36.17	34.44	-	-	-	-
Height of mandibular body	26.92	34.67*	34.18	31.80	-	-	-	-
Breadth of mandibular body	12.22	10.30*	10.38	13.13	-	-	-	-
Bigonial width	-	89.37	93.58	94.82	-	-	-	-
Bicondylar breadth	-	-	114.28	-	-	-	-	-
Min. ramus breadth	-	30.36	30.76	32.80	-	-	-	-
Max. ramus breadth	-	40.57	40.33	39.62*	-	-	-	-
Max. ramus height	-	65.40*	58.61	63.83*	-	-	-	-
Mandibular length	-	107	103	91	-	-	-	-
Biasterionic breadth	-	-	-	104	-	-	-	-

Skeletal dimension	A1028	A1029	A1030	A1031
Max. cranial length	-	-	185	-
Max. cranial breadth	-	-	142	-
Bizygomatic diam.	-	-	146	-
Basion-bregma height	-	149	146	-
Cranial base length	-	106	110	-
Basion-prosthion length	-	102	104	-
Maxillo-alveolar breadth	-	64.26	67.48	-
Maxillo-alveolar length	-	53.15	52.49	-
Biauricular breadth	-	-	128.38	-
Upper facial height	-	69.17	82.34	-
Min. frontal breadth	-	91.93	105.61	-
Upper facial breadth	-	102	112.27	-
Nasal height	-	48.21	56.11	-

Nasal breadth	-	23.65	27.82	-
Orbital breadth	-	40.25	38.48	-
Orbital height	-	34.65	40.57	-
Biorbital breadth	-	89.03	98.84	-
Interorbital breadth	-	22.35	25.95	-
Frontal chord	-	111.98	110	-
Parietal chord	-	110.28	119.19	-
Occipital chord	-	-	91.65	-
Foramen magnum length	-	34.44	36.18	-
Foramen magnum breadth	-	29.23	29.21	-
Mastoid length	-	-	27.86	-
Chin height	-	36.05	38.42	-
Height of mandibular body	-	32.36	34.02	31.24
Breadth of mandibular body	-	12.39	18.18	9.55

Bigonial width	-	93.35	96.43	-
Bicondylar breadth	-	102.23	113.68	-
Min. ramus breadth	-	33.54	33.63	30.08
Max. ramus breadth	-	43.28	45.40	45.03
Max. ramus height	-	61.28	62.06	57.89
Mandibular length	-	97	112	-
Biasterionic breadth	-	-	122	-

Table 3: Post-cranial measurements of the 36 Chinese individuals.

All measurements in mm.
- skeletal element was absent or damaged and therefore measurements could not be obtained
*** indicates right side**

Skeletal dimension	A996	A997	A998	A999	A1000	A1001	A1002	A1003
Clavicle max. length	146	138	-	140	151	139	120	140*
Clavicle ant.-post. diameter midshaft	9.36	10.68	13.38	12.24	11.14	10.46	12.56	9.51*
Clavicle sup.-inf. diameter midshaft	11.73	12.06	16.68	12.75	13.85	13.51	12.45	13.12*
Scapula height	-	-	-	182	185	-	-	159
Scapula breadth	-	-	-	99	123	129*	-	89*
Humerus max. length	294	299	-	-	295	326*	305*	273*
Humerus epicondylar breadth	57.54	58.09	-	-	64.27	59.55*	56.09*	52.70*
Humerus vertical diameter head	45.14	43.48*	-	-	44.68	-	-	39.85*
Humerus max. diameter midshaft	20.18	20.90	21.96	-	23.39	21.28*	21.07	21.93*
Humerus min. diameter midshaft	18.91	18.32	16.24	-	20.27	18.15*	18.06	18.53*
Radius max. length	231	231	-	-	225	229*	-	212*
Radius ant.-post. diameter midshaft	12.38	11.52	-	11.23	12.36	11.66*	11.95	11.90*
Radius med.-lat. diameter midshaft	15.74	13.91	-	16.65	16.43	15.68*	13.42	14.12*
Ulna max. length	250	247*	-	-	243	-	-	232*

Ulna ant.-post diameter	14.76	10.72	-	12.92	12.84	12.80*	12.93	12.45*
Ulna med.-lat. diameter	14.30	14.90	-	16.23	16.34	17.63*	15.14	13.52*
Ulna physiological length	221	222*	-	-	218	-	-	203*
Ulna min. circumference	42	39*	-	-	40	36*	-	36*
Sacrum anterior length	-	117	-	-	119	155	122	120
Sacrum ant.-sup. breadth	-	112	-	-	109	-	-	104
Sacrum max. transverse diameter base	-	45.88	-	-	-	50.88	49.80	46.05
Os coxae height	-	-	-	200	213	207	-	184
Os coxae iliac breadth	-	-	-	-	-	152	-	136
Os coxae pubis length	-	-	-	-	-	-	-	78.05
Os coxae ischium length	-	-	-	-	-	60.71	-	56.98
Femur max. length	433	451	461	446	428	465	445	387
Femur bicondylar length	430	450	456	443	423	461	442	384
Femur epicondylar breadth	74.37	76.86	-	80.96	87.16	77	-	71.14
Femur max. diameter femur head	44.15	45.12	-	-	48.01	48.04	48.37*	43.31

Femur ant.-post. subtrochanteric diameter	27.15	25.79	26.70	28.37	26.97	26.96	27.75	26.51
Femur med.-lat. subtrochanteric diameter	28.92	30.16	31.15	32.91	32.13	30.63	29.65	29.73
Femur ant.-post. midshaft diameter	27.51	23.12	27.35	26.68	27.68	26.97	30.85	27.44
Femur med.-lat. Midshaft diameter	28.57	28.82	26.31	37.22	26.67	24.26	28	24.05
Femur midshaft circumference	89	81	84	87	86	80	90	79
Tibia length	350	351	385	352	349	357	357	315
Tibia physiological length	-	343	374	341	337	346	344	307
Tibia max. prox. epiphyseal breadth	70.09	71.73	-	75.84*	81.86	71.81	-	65.60
Tibia max. distal epiphyseal breadth	42.24	47.01	45.57	50.35	53.58	46.27	51.05	41.69
Tibia max. diameter nutrient foramen	30.05	28.15	36.56	34.59	34.64	34.58	31.58	34.76
Tibia med.-lat. diameter nutrient foramen	22.46	22.23	24.73	26.36	25.13	26.39	24.29	23.13
Tibia circumference nutrient foramen	83	82	97	99	91	93	92	89
Fibula max. length	-	349	-	-	348	-	-	303
Fibula max. diameter midshaft	-	12.91	16.53*	13.70	14.83	18.57	13.70	14.06

Calcaneus max. length	75.71	76	82	81	82	-	-	-
Calcaneus middle breadth	37.80	-	41.33	43.96	43.82	-	-	-

Skeletal dimension	**A1004**	**A1005**	**A1006**	**A1007**	**A1008**	**A1009**	**A1010**	**A1011**
Clavicle max. length	138	140	-	137	137	129	148	126.5
Clavicle ant.-post. diameter midshaft	11.78	10.04	-	11.16	9.85	11.66	11.45	8.90
Clavicle sup.-inf. diameter midshaft	10.24	12.60	-	13.37	13.86	10.79	12.20	12.99
Scapula height	-	-	-	181	181*	-	-	-
Scapula breadth	-	-	-	-	108*	-	-	-
Humerus max. length	319	306	-	313	-	281	-	304*
Humerus epicondylar breadth	56.91	54.12	52.08*	59.92	-	54.76*	-	51.65*
Humerus vertical diameter head	-	-	-	42.87	-	-	-	41.57*
Humerus max. diameter midshaft	19.57	20.49	20.22	21.14	-	21.29	-	21.69
Humerus min. diameter midshaft	18.57	20.07	18.66	17.34	-	16.77	-	15.57
Radius max. length	243*	231	-	240	218	224	-	220*
Radius ant.-post. diameter midshaft	12.16*	10.70	11.55	10.82	13.38	13.66	-	11.44

Radius med.-lat. diameter midshaft	15.30*	14.90	14.79	11.52	16.27	14.96	-	11.95
Ulna max. length	-	-	-	260	236	-	-	237*
Ulna ant.-post diameter	12.30	12.74	13.58	13.34	12.33	11.44*	13.64*	11.74
Ulna med.-lat. diameter	14.53	14.45	13.60	11.96	15.70	14.42*	15.22*	13.69
Ulna physiological length	-	-	-	232	211	-	-	211*
Ulna min. circumference	-	-	-	41	36	36*	40*	39
Sacrum anterior length	-	125	-	133	116	112	108	97
Sacrum ant.-sup. breadth	-	100	-	119	124	-	111	94
Sacrum max. transverse diameter base	-	43.27	-	45.37	52.99	45.89	47.01	41.82
Os coxae height	-	193	-	217	209	-	200*	-
Os coxae iliac breadth	-	142	-	167	156	143*	151*	143*
Os coxae pubis length	-	-	-	-	-	-	-	-
Os coxae ischium length	-	54.37	-	61.87	66.94	55.54*	63.84*	57.09*
Femur max. length	439	432	457	459	410	403*	446	405*
Femur bicondylar length	435	431	455	457	409	401*	445	402*
Femur epicondylar breadth	76.44*	71.07	76.79	77.51	84.23*	69.77*	76.08	70.47

Femur max. diameter femur head	44.03	42.92	46.53*	44.81	48.73	42.76*	43.55	43.36
Femur ant.-post. subtrochanteric diameter	24.46	25.74	28.56	26.13	26.38	25.30*	26.39	23.09
Femur med.-lat. subtrochanteric diameter	30.99	28.91	30.33	35.75	34.29	31.41*	35.79	28.05
Femur ant.-post. midshaft diameter	23.91	27.90	24.57	24.77	27.26	25.08	26.04	23.68
Femur med.-lat. Midshaft diameter	25.76	29.07	27.29	29.51	28.39	23.95	29.17	23.32
Femur midshaft circumference	80	91	80	85	87	80	84	72
Tibia length	-	351	381	369	337	337*	365	328
Tibia physiological length	-	345	368	362	326	327*	361	316
Tibia max. prox. epiphyseal breadth	68.46*	66.70	69.38	71.18	77.03	68.71*	70.94	64.75
Tibia max. distal epiphyseal breadth	-	46.62*	46.95	47.23	51.86	48.96	47.94	43.32
Tibia max. diameter nutrient foramen	32.45	32.15	33.86	33.89	33.20	29.21	35.64	27.02
Tibia med.-lat. diameter nutrient foramen	23.38	22.71	21.69	22.84	27.49	21.77	25.44	19.46
Tibia circumference nutrient foramen	90	88	88	92	94	82	92	74
Fibula max. length	-	342*	361	356	-	-	356	310*

Fibula max. diameter midshaft	14.06	12.40	17.06	12.18	-	13.01	13.89	12.04
Calcaneus max. length	75.05*	77	80*	73	81	72.43	80	70
Calcaneus middle breadth	43.96*	38.03	32.56*	38.58	40.02	37.80	38.11	37.84

Skeletal dimension	A1012	A1013	A1014	A1015	A1016	A1017	A1018	A1019
Clavicle max. length	-	-	139	131	-	-	-	127*
Clavicle ant.-post. diameter midshaft	-	-	11.26	9.75	-	-	-	10.26
Clavicle sup.-inf. diameter midshaft	-	-	13.75	12.30	-	-	-	8.98
Scapula height	-	-	-	-	-	-	-	-
Scapula breadth	-	-	-	-	-	-	-	-
Humerus max. length	-	-	306*	307	323*	-	-	310*
Humerus epicondylar breadth	-	-	54.50*	59.12	59.64*	-	-	-
Humerus vertical diameter head	-	-	-	-	-	-	-	-
Humerus max. diameter midshaft	19.32	17.76	21.50*	23.66	20.76	20.21	22.79	20.04*
Humerus min. diameter midshaft	17.85	17.43	18.46*	30.31	17.62	23.40	17.98	15.21*
Radius max. length	-	232	236	246	-	-	-	219
Radius ant.-post. diameter midshaft	-	11.47	11.97	12.30	11	-	-	11.10
Radius med.-lat. diameter midshaft	-	13.40	15.58	15.25	14.63	-	-	13.13
Ulna max. length	-	247	-	-	-	-	-	-
Ulna ant.-post diameter	-	12.69	16.27	14.02	12.48	11.80	-	10.87
Ulna med.-lat. diameter	-	15.21	14.60	14.64	15.69	15.40	-	15.46
Ulna physiological length	-	220	-	-	-	-	-	-

Ulna min. circumference	-	36	32	39	-	-	-	36
Sacrum anterior length	-	-	-	-	-	-	-	-
Sacrum ant.-sup. breadth	-	-	-	-	-	-	-	112
Sacrum max. transverse diameter base	-	-	48.64	-	45.92	-	-	48.62
Os coxae height	-	-	-	-	-	-	-	-
Os coxae iliac breadth	-	-	140	-	-	-	-	140*
Os coxae pubis length	-	-	-	-	-	-	-	-
Os coxae ischium length	-	-	-	-	-	-	-	60.36*
Femur max. length	--	444	405	452*	451	421*	436	436
Femur bicondylar length	-	437	402	449*	450	-	431	434
Femur epicondylar breadth	-	75.67	76.58	76.75	74.26	-	-	84.62
Femur max. diameter femur head	-	47.48	42.19	-	-	-	-	46.80
Femur ant.-post. subtrochanteric diameter	25.67*	26.66	23.90	28.76	27.56	-	25.40	26.13
Femur med.-lat. subtrochanteric diameter	31.51*	33.02	30.73	32.08	30.97	-	28.98	33.57

Femur ant.-post. midshaft diameter	25.61*	23.81	26.13	29.13	28.21	25.48	28.58	25.28
Femur med.-lat. Midshaft diameter	25.44*	28.48	26.64	25.91	26.28	22.61	23.63	28.74
Femur midshaft circumference	80*	83	83	86	87	76	83	84
Tibia length	345*	361	348*	-	372	343	-	359
Tibia physiological length	338*	354	335*	-	360	333	-	349
Tibia max. prox. epiphyseal breadth	69.41	75.52	72.28*	71.68	73.41	-	-	71.60
Tibia max. distal epiphyseal breadth	47.41*	50.17	45.14*	52.34	48.30	45.83	-	48.21
Tibia max. diameter nutrient foramen	32.38	34.17	32.14*	34.46	34.18	31.15	31.92	33.50*
Tibia med.-lat. diameter nutrient foramen	25.16	25.65	23.83*	23.19	25.09	25.98	23.86	23.80*
Tibia circumference nutrient foramen	89	96	87*	96	88	88	86	91*
Fibula max. length	-	346*	328	-	357	-	-	-
Fibula max. diameter midshaft	14.36	14.68	14.60	17.79*	15.36	14.89	-	13.40
Calcaneus max. length	-	74	70	-	79	-	-	76
Calcaneus middle breadth	37.47	40.41	34.23	-	38.35	-	-	37.12

Skeletal dimension	A1020	A1021	A1022	A1023	A1024	A1025	A1026	A1027
Clavicle max. length	-	-	143	121	139*	-	-	-
Clavicle ant.-post. diameter midshaft	-	9.59*	8.57	10.80	9.81*	12.01*	-	-
Clavicle sup.-inf. diameter midshaft	9.32	13.43*	11.88	11.18	11.86*	13.00*	-	-
Scapula height	-	-	176	168*	-	-	-	-
Scapula breadth	-	-	119	91*	-	-	-	-
Humerus max. length	-	-	294	280	270	-	-	-
Humerus epicondylar breadth	-	-	56.74	54.95	55	-	-	-
Humerus vertical diameter head	-	-	-	38.31*	38.81	-	-	-
Humerus max. diameter midshaft	19.68*	18.98	19.48	23.76	22.64	-	-	20.33
Humerus min. diameter midshaft	15.27*	17.84	18.59	18.73	19.03	-	-	15.58
Radius max. length	-	-	233	214	212	-	-	-
Radius ant.-post. diameter midshaft	10.46	-	11.65	11.65	12.64	-	-	11.24
Radius med.-lat. diameter midshaft	14.13	-	13.52	15.35	14.54	-	-	13.64
Ulna max. length	-	-	250	237*	232	-	-	-
Ulna ant.-post diameter	11.65	-	11.08	12.39	12.86	-	-	11.04
Ulna med.-lat. diameter	12.44	-	14.78	13.23	13.90	-	-	14.44

Ulna physiological length	-	-	227	214*	202	-	-	-
Ulna min. circumference	-	-	35	36	36	-	-	-
Sacrum anterior length	-	-	-	132	-	-	108	-
Sacrum ant.-sup. breadth	-	-	-	103	-	-	-	-
Sacrum max. transverse diameter base	-	38.99	-	45.20	-	-	42.27	-
Os coxae height	-	-	-	-	-	-	-	-
Os coxae iliac breadth	-	-	-	134	-	-	145*	-
Os coxae pubis length	-	-	-	-	-	-	-	-
Os coxae ischium length	-	-	60.89	53.91*	-	-	59.16*	-
Femur max. length	-	-	423	410*	-	-	-	-
Femur bicondylar length	-	-	420	406*	-	-	-	-
Femur epicondylar breadth	-	-	76.69	74.56*	-	-	-	-
Femur max. diameter femur head	-	-	47.50	46.01	-	43.04	-	-
Femur ant.-post. subtrochanteric diameter	23.90	23.16	24.31	25.79*	-	23.59	-	24.33

Femur med.-lat. subtrochanteric diameter	26.83	30.82	33.79	29.92*	-	32.08	-	30.81
Femur ant.-post. midshaft diameter	24.88	23.07	25.92	26.38*	-	23.55	-	26.12
Femur med.-lat. Midshaft diameter	22.61	25.16	29.10	24.65*	-	26.40	-	26.05
Femur midshaft circumference	74	76	84	80*	-	77	-	80
Tibia length	-	-	354	327	-	-	353	-
Tibia physiological length	-	-	344	316	-	-	342	-
Tibia max. prox. epiphyseal breadth	-	-	70.91	69.11*	-	-	67.43	-
Tibia max. distal epiphyseal breadth	-	-	53.07	50.12*	-	-	47	47.29
Tibia max. diameter nutrient foramen	31.18	-	32.18	28.87	-	-	30.68	28.92
Tibia med.-lat. diameter nutrient foramen	22.43	-	23.38	23.45	-	-	21.46	22.56
Tibia circumference nutrient foramen	83	-	86	81	-	-	83	84
Fibula max. length	-	-	339*	318*	-	-	243*	-
Fibula max. diameter midshaft	12.60	-	15.14	13.59	-	-	15.34	13.58
Calcaneus max. length	-	70	86	71	64	-	47.81	-
Calcaneus middle breadth	67.78	42.11	40.29	41.29	36.55	-	37.69	-

Skeletal dimension	A1028	A1029	A1030	A1031
Clavicle max. length	-	135	135	133
Clavicle ant.-post. diameter midshaft	-	9.25	10.09	10.33
Clavicle sup.-inf. diameter midshaft	-	11.35	13.06	12.24
Scapula height	-	166	164	-
Scapula breadth	-	195	112	-
Humerus max. length	-	288	-	273
Humerus epicondylar breadth	-	55.29	-	58.93
Humerus vertical diameter head	-	39.68	-	41.10
Humerus max. diameter midshaft	-	20.35	-	22.12
Humerus min. diameter midshaft	-	20.55	-	20.92
Radius max. length	-	-	216	219*
Radius ant.-post. diameter midshaft	11.55	-	11.61	11.88
Radius med.-lat. diameter midshaft	16.38	-	14.54	14.49
Ulna max. length	-	-	239	244*
Ulna ant.-post diameter	-	-	12.16	14.25

Ulna med.-lat. diameter	-	-	15.45	13.84
Ulna physiological length	-	-	209	221
Ulna min. circumference	-	-	36	41
Sacrum anterior length	-	101	114	117
Sacrum ant.-sup. breadth	-	-	109	113
Sacrum max. transverse diameter base	45.60	44.22	50.41	48.57
Os coxae height	-	201	-	-
Os coxae iliac breadth	-	142	151*	-
Os coxae pubis length	-	-	-	-
Os coxae ischium length	-	55.43	58.05*	-
Femur max. length	402	-	417	431
Femur bicondylar length	396	-	413	426
Femur epicondylar breadth	-	-	74.47	79.77
Femur max. diameter femur head	-	42.28	45.87*	44.64
Femur ant.-post. subtrochanteric diameter	24.39	25.96	25.97	24.51

Femur med.-lat. subtrochanteric diameter	30.25	29.03	31.53	33.72
Femur ant.-post. midshaft diameter	26.19	25.74	25.26	25.59
Femur med.-lat. Midshaft diameter	27.00	25.50	28.74	28.78
Femur midshaft circumference	81	80	82	83
Tibia length	-	-	359	355
Tibia physiological length	-	-	349	343
Tibia max. prox. epiphyseal breadth	-	-	73.85	70.97
Tibia max. distal epiphyseal breadth	-	-	46.83	49.51
Tibia max. diameter nutrient foramen	-	-	32.32	32.91
Tibia med.-lat. diameter nutrient foramen	-	-	27.25	24.19
Tibia circumference nutrient foramen	-	-	87	89
Fibula max. length	-	-	-	339
Fibula max. diameter midshaft	-	-	14.06*	11.74
Calcaneus max. length	-	-	-	76
Calcaneus middle breadth	-	-	-	40.85

Table 4: Dental measurements of the 36 Chinese individuals.

All measurements were taken in mm.
- dentition was absent or damaged and therefore measurements could not be obtained
*** indicates right side. MD=mesiodistal, BL = buccolingual**

Dimension												
Maxilla	**A996**	**A997**	**A998**	**A999**	**A1000**	**A1001**	**A1002**	**A1003**	**A1004**	**A1005**	**A1006**	**A1007**
MDI1	-	-	7.53	9.20*	-	-	6.47	-	9.43	-	-	8.18*
BL I1	-	-	7.04	7.80*	-	-	5.54	-	8.37	7.00	-	7.14*
MD I2	-	-	7.02	6.88	-	-	5.64	-	7.40	6.83	-	6.82*
BL I2	-	-	5.66	6.86	-	-	5.99	-	7.48	5.76	-	6.41*
MD C	-	6.95	-	8.55	7.63*	6.80	7.58	-	7.76	6.85	-	7.53
BL C	-	7.38	8.16	9.15	7.90*	7.14	6.99	7.99	7.78	5.91	-	8.06
MD PM1	7.56	7.09	7.91	7.71	7.58	6.10	7.28	6.77	7.78	6.95	6.91	6.99
BL PM1	9.19	8.91	9.38	10.00	9.54	7.67	8.78	9.33	10.26	7.38	9.73	9.14
MD PM2	7.08	7.04	6.75	6.22	-	5.72	6.48	6.32*	6.62	5.86	6.17	6.64
BL PM2	9.19	8.90	8.92	-	9.37	8.35	8.53	9.02*	9.08	8.32	9.90	8.53
MD M1	10.00	9.70	-	11.23	10.24	10.12	10.23	9.29*	12.42	10.50	8.99	9.21
BL M1	10.41	10.57	11.34	11.31	11.27	11.74	10.64	11.02*	12.74	10.25	11.30	10.79
MD M2	9.47	10.07	10.63	10.81*	-	8.91	10.17	9.60*	10.94	9.12	-	8.60
BL M2	10.75	11.33	11.58	12.55*	11.42	11.08	11.07	10.78*	-	10.99	-	11.65
MD M3	8.92	-	-	8.89*	9.28	8.12	9.53	-	-	8.92	-	-
BL M3	10.23	-	-	12.98*	11.66	11.01	11.35	-	-	10.47	-	-
Mandible												
MD I1	-	-	5.13	5.80	-	-	-	-	5.38	6.10	-	6.07
BL I1	-	-	5.96	6.12	-	-	-	-	6.23	5.73	-	5.99
MD I2	-	-	5.68	6.92	-	-	6.11	-	6.38	5.79	-	5.74
BL I2	5.74	-	6.26	6.43	6.24*	-	5.78	-	6.40	5.88	-	5.73
MD C	6.94	-	6.96	7.40	-	6.23	6.63	-	7.63	6.24	-	6.62
BL C	7.36	7.77	7.56	8.88	-	6.56	7.62	-	8.48	7.55	-	7.51
MD PM1	6.90	-	7.45	7.21*	7.23*	6.78	6.63	6.71*	7.27	6.31	-	6.06
BL PM1	7.74	-	7.86	8.03*	8.15*	7.48	7.08	8.08*	7.65	7.17	-	6.65
MD PM2	6.76	6.89	7.32	7.60	6.92	6.47	6.87	6.78*	7.60	6.33	-	6.14
BL PM2	7.92	7.70	8.62	9.12	8.34	8.00	7.58	7.88*	8.47	7.52	-	7.77
MD M1	11.24	10.96	11.19	11.26	-	10.92	11.33	-	12.07	10.99	10.40	10.64
BL M1	11.09	11.10	11.06	10.85	-	11.09	10.75	10.61	12.33	10.56	10.75	10.60

MD M2	10.89	11.05	11.16	12.27	10.75	9.24	11.05	9.67	11.94	10.43	10.28	-
BL M2	-	10.78	10.03	11.25	11.31	9.99	10.32	10.60	11.62	10.66	10.30	10.55
MD M3	9.18	-	10.13	11.30	-	10.29*	-	9.98	-	10.45	10.38	10.27
BL M3	9.70	-	10.54	11.10	-	10.24*	-	10.24	-	9.77	10.19	9.38

Dimension												
Maxilla	**A1008**	**A1009**	**A1010**	**A1011**	**A1012**	**A1013**	**A1014**	**A1015**	**A1016**	**A1017**	**A1018**	**A1019**
MD I1	-	7.76*	8.10*	6.91	-	-	-	-	7.09	8.21	-	-
BL I1	6.77	7.19*	7.71*	7.19	-	-	-	-	7.07	6.82	-	-
MD I2	6.72	-	6.71	4.18	-	-	6.65	6.23*	6.71	-	-	6.14
BL I2	6.01	6.64	6.95	4.90	-	-	7.09	-	6.96	-	-	6.03
MD C	7.64	-	7.17	7.16	-	7.83	-	8.06	8.09	7.19	-	7.93
BL C	7.74	7.42	8.82	7.10	-	8.30	-	8.35	8.36	7.91	-	8.45
MD PM1	7.04	7.03	6.82	6.23	-	7.51	7.38	7.37	7.53	6.60	-	7.19
BL PM1	8.22	8.50	9.86	8.07	-	9.18	9.53	10.77	9.67	8.52	-	9.18
MD PM2	6.11	6.55	5.49	6.61	7.01	6.88	6.49	7.34	6.65	6.24	-	6.85
BL PM2	8.11	8.73	8.77	8.73	9.77	8.76	9.05	10.10	9.03	8.44	-	9.36
MD M1	10.26	10.91	9.73	10.31	10.06	10.44	10.67	10.18	10.52	10.45	11.50	9.93
BL M1	11.22	10.99	11.64	11.25	11.26	11.00	11.08	11.91	11.33	11.33	11.13	11.45
MD M2	8.90	9.48	9.79	9.22	10.47	9.58	9.58	9.93	-	9.36	10.39	9.39
BL M2	11.16	10.43	11.60	10.61	11.37	11.55	11.34	12.22	-	10.83	10.45	11.65
MD M3	-	-	8.65	6.92*	-	8.58	8.69	6.95*	-	-	-	-
BL M3	-	-	11.77	8.18*	-	10.69	11.84	11.66*	-	-	-	-
Mandible												
MD I1	3.51	-	4.22	5.69	-	-	-	-	-	-	-	4.92
BL I1	4.71	-	5.07	5.84	4.66	-	-	-	5.54*	4.95	-	5.93
MD I2	5.84	-	5.99	-	-	5.15	-	-	5.19	5.71	-	6.03
BL I2	5.76	-	6.80	5.60	5.58	6.59	-	-	6.16	5.38	-	6.29
MD C	6.71	6.92*	7.02	7.34	6.67	6.98	-	7.30	7.28	5.52*	6.45	6.76*
BL C	6.65	7.34*	7.84	7.59	7.66	8.12	-	8.17	8.23	7.23*	-	7.40*
MD PM1	7.69	7.13	7.16	6.57	7.29	7.62	-	7.58	7.07	6.41	-	7.10
BL PM1	7.56	6.69	7.96	7.49	8.00	7.70	-	8.41	8.95	7.57	-	7.70

MD PM2	7.89	7.07*	6.32	6.33	7.86	7.29	-	7.94	7.00	6.34	7.93	6.65
BL PM2	8.48	7.12*	8.38	7.42	8.31	7.61	-	8.65	9.21	7.88	6.66	8.07
MD M1	11.00	11.33	10.95	10.21	11.67	11.54	-	11.03	11.31	11.66	10.97	10.82
BL M1	10.69	11.31	11.68	10.48	11.49	9.82	-	10.95	11.16	10.39	11.02	10.70
MD M2	10.76	9.49	10.91	9.63	11.61	10.65	-	11.85	-	10.35	11.41	10.87
BL M2	10.39	9.86	11.49	10.42	10.71	10.45	-	10.84	-	10.13	11.02	10.09
MD M3	9.54	-	10.98	8.82	-	10.35	-	9.91	-	-	11.01	-
BL M3	9.46	-	11.79	9.54	-	9.97	-	10.67	-	-	10.71	-

Dimension												
Maxilla	**A1020**	**A1021**	**A1022**	**A1023**	**A1024**	**A1025**	**A1026**	**A1027**	**A1028**	**A1029**	**A1030**	**A1031**
MD I1	-	-	8.97	-	-	7.07	-	-	-	-	-	-
BL I1	-	-	6.68	-	-	6.91	-	-	-	-	-	-
MD I2	-	-	7.07	5.94*	-	6.95	-	-	-	5.99	-	-
BL I2	-	-	5.73	6.97*	-	6.35	-	-	-	5.61	-	-
MD C	-	-	8.44	7.59	-	7.83	-	-	-	8.33	8.10	-
BL C	-	-	8.88	8.01	-	7.93	-	-	-	8.78	8.56	-
MD PM1	-	6.42	7.82	7.04	-	7.25	-	-	-	7.71	-	-
BL PM1	-	7.44	9.65	9.45	-	9.07	-	-	-	9.23	-	-
MD PM2	-	6.42	7.05	6.45	6.01	7.14	-	-	-	6.74	7.01	-
BL PM2	-	7.83	9.59	9.23	8.75	9.41	-	-	-	9.74	9.64	-
MD M1	-	9.40	9.79	10.47	-	-	-	-	-	10.10	9.98	-
BL M1	-	10.58	11.07	11.52	-	-	-	-	-	12.96	11.48	-
MD M2	-	9.33	9.25	9.75	9.76	-	-	-	-	11.75*	9.48	9.21
BL M2	-	10.70	11.26	11.14	10.75	-	-	-	-	7.45*	11.76	10.57
MD M3	-	8.37	8.60*	-	8.48	-	-	-	-	-	8.31*	8.77
BL M3	-	9.83	10.50*	-	10.99	-	-	-	-	-	10.16*	10.60
Mandible												
MD I1	-	-	5.63	4.50*	4.63	5.38	-	-	-	4.37	-	-
BL I1	-	-	5.26	5.56*	5.95	5.31	-	-	-	5.92	-	-
MD I2	5.91*	-	5.95	5.69	4.93	-	-	-	-	6.44	-	-
BL I2	5.35*	-	5.84	5.88	5.20	-	-	-	-	6.55	-	-

MD C	6.82*	-	7.20*	6.81	-	-	-	-	-	7.51	6.85	6.91*
BL C	7.22*	-	7.71*	7.33	-	-	-	-	-	8.44	7.53	7.32*
MD PM1	6.33	6.75*	7.30	7.03	-	8.32	-	-	-	7.94	-	6.65
BL PM1	6.90	7.43*	8.40	7.53	-	8.94	-	-	-	8.17	-	7.18
MD PM2	7.06	6.76	7.38	6.33	-	7.05	-	-	-	7.68	6.90	7.06
BL PM2	7.72	7.65	8.57	7.63	-	8.52	-	-	-	8.27	7.96	7.49
MD M1	10.16	10.07	11.11	11.52	-	-	-	-	-	11.42	11.09	10.59
BL M1	10.29	10.28	10.88	10.98	-	-	-	-	-	12.05	11.34	10.15
MD M2	10.42	10.00	11.31	11.33	-	-	-	-	-	11.97*	11.01	10.26
BL M2	10.16	9.50	10.47	10.96	-	-	-	-	-	11.86*	11.12	9.61
MD M3	11.07*	9.66	-	9.97*	-	10.44	-	-	-	-	10.34	9.72
BL M3	10.19*	9.31	10.26	10.78*	-	10.64	-	-	-	-	10.77	8.98