INVESTIGATING THE EMERGENCE AND SPREAD OF TUBERCULOSIS IN SOUTH AFRICA

by

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Declaration

I, Tessa Jane Campbell, hereby declare that the work on which this thesis is based is my original work (except where acknowledgements indicate otherwise) and that neither the whole work nor any part of it has been, is being, or is to be submitted for another degree in this or any other university. I authorise the University to reproduce for the purpose of research either the whole or any portion of the contents in any manner whatsoever.

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ABSTRACT

Tuberculosis is the leading cause of death in South Africa and, while the disease is clearly very relevant today, little has been done to understand its emergence locally from a deep history perspective. Bioarchaeological investigations of tuberculosis have received much attention in Europe and North America but, with the exception of Egypt, the African continent has not been well-evaluated in this regard despite numerous genetic studies suggesting an African origin for the disease. This thesis aims to identify cases of skeletal tuberculosis in the archaeological record of South Africa and, aided by historical literature, trace its geographical and temporal origins in order to inform a broader local and global understanding of this ancient pathogen. Ancient DNA detection and analysis was also attempted in order to compliment skeletal diagnoses and obtain strain informative sequences. Preservation, demographic and paleopathological data were collected for 2321 individuals, representing the majority of South African Holocene and historical remains curated in local institutions, and a thorough description of this extensive dataset is presented. Only twelve individuals showing pathology suggestive of tuberculosis were identified. All potentially date to the colonial period; however, two individuals likely predate direct European contact. The association of these individuals with Iron Age populations may suggest a link between an early introduction of the disease and East African trade but caution in this interpretation is advised due to the tentative diagnoses in both cases. The skeletal record supports the major events identified in the literature; an early focus of disease along the Orange River and at the major port city of Cape Town, followed by a cluster of cases in Kimberley associated with mining activities and, finally, a spread to rural agricultural communities. Of the nine individuals subject to ancient DNA analysis using silica-based extraction protocols and quantitative PCR, M*ycobacterium tuberculosis* complex (MTBC) DNA was identified in only three. Attempts to obtain MTBC strain informative sequences through next generation sequencing were unsuccessful. This is the first study in South Africa to report the detection of ancient pathogen DNA; however, despite a possible African origin for tuberculosis, there is no evidence to support a pre-colonial presence of the disease in South Africa.

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TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

LIST OF ABBREVIATIONS

aDNA	Ancient DNA
ALB	Albany Museum
BCG	Bacille Calmette-Guerin
BRI	Bone representation index
LSA	Later Stone Age
EIA	Early Iron Age
GLD	Gladstone Cemetery
HOA	Hypertrophic osteoarthropathy
LIA	Late Iron Age
PCR	Polymerase Chain Reaction
MRCA	Most recent comment ancestor
MTBC	*Mycobacterium tuberculosis* complex
MMK	McGregor Museum
MNI	Minimum number of individuals
NDT	Neolithic demographic transition
NGS	Next generation sequencing
NMB	National Museum, Bloemfontein
NTM	Nontuberculous mycobacteria
qPCR	Quantitative PCR
SAM	IZIKO Museums of South Africa
SNP	Single nucleotide polymorphism
UCT	University of Cape Town
UP	University of Pretoria
VOC	*Vereenigde Oost-Indische Compagnie* or Dutch East India Company
WGA	Whole genome amplification
WITS	University of the Witwatersrand
WWI	World War I

CHAPTER 1: INTRODUCTION

Prior to the 1980s, tuberculosis was thought to be on the decline. Certainly, in most Western countries disease prevalence had dropped dramatically from peaks in the 17th and 18th centuries. It was hoped that the widespread use and availability of antibiotics would lead to its eventual eradication. Tuberculosis incidence began to rise worldwide in the 80s, however, spurred partly by the rise of the HIV epidemic and drug-resistant strains of tuberculosis, particularly in developing countries. These factors, in combination with increased immigration, resulted in a resurgence of disease, even in countries were long term trends of decline had been observed. The last two decades have, however, ushered in a steady decline in global tuberculosis incidence and mortality (World Health Organization [WHO], 2018). Despite this, tuberculosis remains the leading cause of death, globally, from a single infectious agent. The World Health Organization (2018) reported an estimated 10 million new cases and 1.3 million deaths in 2017. Developing countries, particularly in Asia and Africa, bear the highest burden of disease. Of the 30 high burden countries identified by the World Health Organization, South Africa is ranked 8th globally and 2nd in Africa (WHO, 2018). Within South Africa, tuberculosis is the leading cause of death, contributing to 6.5% of deaths from natural causes in 2016 (StatsSA, 2018). In 2017, an estimated 322 thousand new cases and 22 thousand deaths were reported (WHO, 2018). Nationally, the disease predominantly affects adults of reproductive age (WHO, 2018) and it is the leading cause of death among men but not women (where tuberculosis is ranked fifth) (StatsSA, 2018). The modern relevance of this disease has undoubtedly contributed to interest in understanding the emergence and evolution of what is actually an ancient disease.

Tuberculosis is an infectious disease typically transmitted from one person to another via aerosol droplets containing bacilli of the *Mycobacterium tuberculosis* complex (MTBC). According to the WHO (2018), approximately 5-10% of infected individuals will progress to active disease. An estimated 23% of the global population has a latent tuberculosis infection and is at risk of developing active disease. The lungs are the primary organ of infection but any organ of the body can be affected. Extra-pulmonary tuberculosis accounts for approximately 14% of tuberculosis cases (WHO, 2018) and skeletal tuberculosis manifests itself in 30% of extrapulmonary cases and 3% of the total (Ortner, 2003). Tuberculosis infection of the bone leads to abnormal bone changes which are typically characterized by lytic lesions but both destruction and new bone formation can be observed (Ortner, 2003). It is, therefore, in these cases, where the disease affects the bone, that we are able to recognise it in the bioarchaeological record. Bone is, however, limited in its response to insult and an observed lesion could have a number of possible aetiologies.

Initial investigations into the antiquity of tuberculosis focused on evidence from the written and skeletal records. The earliest written evidence that includes a possible description of the disease is found in an ancient Chinese medical text dating to 2700 BC (Roberts and Buikstra, 2003), while the oldest convincing skeletal evidence indicates that the disease has plagued our species for at least six millennia (Canci et al., 1996). Technological developments in DNA amplification and sequencing in the 1980s ushered in a new avenue of investigation into tuberculosis, with both phylogenetic studies of modern people and ancient DNA (aDNA) studies contributing to our understanding of tuberculosis in the past. Studies of modern (living) people have shown that Africa hosts the greatest genetic diversity of MTBC lineages, and have suggested an African origin for the disease (Comas et al., 2013; Gutierrez et al., 2005; Hershberg et al., 2008; Wirth et al., 2008). In addition, researchers have attempted to estimate when the disease emerged, trace its global dispersion and evaluate the nature host-pathogen co-evolution (see Achtman, 2016; Brites & Gagneux, 2015; Galagan, 2014); however, modern phylogenetic studies have their limitations. They are influenced by the rate of bacterial evolution, the potential effects of selective sweeps and bottlenecks, as well as ancient and modern population movements (Brites and Gagneux, 2015; Smith et al., 2009). Ancient DNA studies offer a window into the past and can aid not only in the confirmation of skeletal diagnoses but also provide insights into the source of disease, the timing of its emergence, the evolution of the bacterium itself and the nature of host-pathogen co-evolution (see Achtman, 2016; Brites & Gagneux, 2015; Galagan, 2014). However, ancient DNA studies are fraught with challenges of their own.

The antiquity of tuberculosis in South Africa has not received much attention and little is known about the emergence of the disease on the continent more broadly (Roberts and Buikstra, 2003), despite indications of a possible African origin for the disease. Literature reports suggest that tuberculosis was absent in South Africa prior to colonial settlement.

However, there are issues with some of the assumptions made, and the accounts of early European explorers, missionaries and settlers, while mostly consistent, were not always so. The skeletal record of South Africa has not been systematically investigated in this context and only a few cases of skeletal tuberculosis have been reported in the literature. Furthermore, no successful ancient pathogen DNA studies have been reported in South Africa to date.

THIS STUDY

This study aims to trace the emergence and spread of tuberculosis in South Africa using the historical literature, skeletal record and ancient DNA. More specifically, the aims are to:

- identify the earliest reports in the historical literature, examine the case for precolonial tuberculosis and identify the historical factors that have led to the current epidemic.
- conduct a thorough survey of South African Holocene and historical skeletal material to identify possible cases of skeletal tuberculosis.
- collect preservation, sex, age and pathology data on the surveyed skeletal remains in order to frame the discussion of identified tuberculosis cases.
- identify the earliest skeletal evidence of tuberculosis in South Africa, describe any patterns observed among all cases and relate these to the modern and historical literature.
- attempt to confirm paleopathological diagnoses of identified skeletal cases with ancient MTBC DNA analyses.
- obtain lineage/strain informative sequences, if possible, to gain insight into the origin(s) of the infecting bacillus and contribute an understanding of the timing and evolution of this pathogen.

This study primarily aims to contribute to a greater understanding of the emergence and spread of a modern health epidemic in a local context. However, it also contributes to the global understanding of these processes. Africa bears a high burden of disease and tuberculosis may potentially have an African origin, but so little is understood about its emergence on the continent.

This thesis is divided into six chapters. Chapter 2 provides a brief overview of the global history of tuberculosis based on evidence from the historical literature and bioarchaeological record. A brief outline of the peopling of South Africa follows, and provides context for a review of the South African historical literature which details the emergence of this disease. Here, the assumption that tuberculosis was absent in pre-colonial South Africa is challenged (but not necessarily discarded). Finally, the major historical events that have contributed to the current epidemic are presented. The contributions of modern and ancient molecular tuberculosis studies are detailed in Chapter 3. Specifically, this chapter begins with a brief history of ancient DNA studies and the challenges associated with analysing degraded DNA. Subsequent sections review the body of literature focused on ancient molecular tuberculosis studies specifically. This includes a review of methods used and insights gleaned about this ancient pathogen. The bioarchaeological, molecular and statistical methods used in this study are detailed in Chapter 4. In Chapter 5, the results are presented in two parts. The first part presents an analysis of the complete assemblage, while the second part provides detailed descriptions and contextual information for all cases of possible tuberculosis identified in this study, as well as the results of the molecular analyses. A similar two part format is observed for the discussion presented in Chapter 6. Here, discussion of the preservation and demographic patterns observed for the assemblage as a whole provides the context within which the discussion of identified tuberculosis cases can be framed. This is followed by a differential diagnosis of identified cases and a discussion of the historical context and significance of these finds.

A NOTE ON TERMINOLOGY

The people of South Africa are diverse and represent many different population histories. These different population histories have also affected experiences with disease and it is, therefore, necessary to distinguish among different populations, particularly with respect to tuberculosis which, past and present, does not affect all populations equally. The racial history of South Africa and the legacy of Apartheid have left its citizens critical of attempts at classification and the terms used to effect such classification. Identifying appropriate terminology is, therefore, not an easy task and it is necessary to acknowledge the problems associated with much of the terminology currently in use. Some of these terms are not appropriate for different time periods and others are not appropriate because of their derogatory connotations. Historical terms like “Bushman”, “San”, “Hottentot” and “Bantu” all have derogatory connotations and are offensive to some or all within descendant populations. One alternative approach is to use terms associated with the primary subsistence economy, like hunter-gatherer, herder and agro-pastoralist, which differed among the indigenous pre-colonial populations of South Africa. These terms are not generally viewed as offensive, but their use does become increasingly irrelevant/inappropriate after colonization with the disruption of traditional economies and increasing admixture. Furthermore, they suggest that subsistence economy was fixed and universal within a broader population, which is not supported by historical literature (Penn, 1986; Viljoen, 1996). Another approach is based on the technology associated with pre-colonial populations and includes terms like Later Stone Age (LSA) and Iron Age populations. Again these terms are not particularly offensive but are not appropriate after colonization. Other terms are based on language e.g. Khoekhoe (previously Khoikhoi) or Bantu-speaking. These are generally considered acceptable and are frequently used. Under Apartheid the racial terms “black”, “white”, “coloured” and “Indian” were used and many of these terms are still used to describe modern populations, although they are considered offensive to some. The term Khoesan (previously Khoisan) is also frequently used and is a collective name for the Khoekhoen and San – the terms preferred by modern groups. However, groups have also expressed dissatisfaction with the collective term preferring the use of individual group names (Hitchcock et al., 2006). It is, however, not always possible to distinguish between these populations in the archaeological record and past group affiliations are not always known. Lastly, the term African is used to refer to modern Bantu-speaking/Black South Africans, but the exclusive usage of this term has also been criticized. None of these terms is ideal in all circumstances and, consequently, all have been used in this thesis. To summarize, the terms “Bushman” and San refer to hunter-gatherer populations and Khoekhoen and “Hottentot” to herders. Khoesan and Later Stone Age are collective terms for these two groups. The descendants of these populations were classified as “coloured” under Apartheid, a term encompassing all lighter “brown”-skinned individuals with a high degree of admixture, but modern affiliations vary. Agro-pastoralist populations are referred to as such or as Iron Age, Bantu-speaking, Black or African. These terms are not strictly synonymous and usage depends on the context. The historically offensive terms are only used when quoting or specifically discussing historical text.

CHAPTER 2: THE HISTORY OF TUBERCULOSIS IN SOUTH AFRICA

This chapter is divided into a number of sections based on major events that have contributed to the nature and spread of tuberculosis in South Africa. In order to understand this process, it is necessary to understand the origin and movement of people across the South African landscape. A brief description of the peopling of South Africa is followed by a discussion of the current knowledge concerning the presence/absence of tuberculosis prior to colonial settlement. All subsequent sections discuss the impact of colonial settlement on the nature and spread of the disease among indigenous populations. I begin with a brief note on statistical data collection for tuberculosis among indigenous populations. This is followed by the impact of tuberculous immigrants; mining and the migrant labour system; industrialization and urbanization; and finally drug resistance and the HIV/AIDS-tuberculosis co-epidemic.

Before discussing the history of tuberculosis in South Africa it is important to outline the global history of the disease through which the South African experience can be framed.

THE GLOBAL HISTORY OF TUBERCULOSIS

TUBERCULOSIS IN ANCIENT HUMAN REMAINS

Tuberculosis is an ancient disease that, based on skeletal evidence, has plagued our species for at least six millennia. Although two earlier cases of tuberculosis have been reported, one in a *Homo erectus* specimen from Turkey (~500 ka; Kappelman et al., 2008), and another in a human specimen from Israel (ca. 9000 BP; Hershkovitz et al., 2008), the diagnoses in both instances have been questioned (Roberts et al., 2009; Stone et al., 2009; Wilbur et al., 2009). Following the geographic regions described in Roberts & Buikstra (2003), the earliest convincing evidence is concentrated in the Mediterranean region. The earliest evidence has been recovered from Italy where two specimens dating to the 4th millennium BC and 5800 ± 90 BP have been reported (Canci et al., 1996; Formicola et al., 1987). Egypt (4500 BC) and Jordan (3150 – 2200 BC) also provide early evidence (Morse et al., 1964; Ortner and Frohlich, 2008), while possible cases appear later in Spain during the Neolithic, and by 900 BC in Greece (Angel, 1984; Roberts and Buikstra, 2003). By the 4th century AD, the earliest case appears in France (Roberts and Buikstra, 2003), but evidence for tuberculosis does not appear in other countries within the Mediterranean region until later in the Medieval period (Roberts and Buikstra, 2003).

Early evidence has also been reported from Northern Europe. Poland has the oldest possible case dating to 5000 BC (Gladykowska-Rzeczycka, 1999), while evidence appears sometime later in southern Russia (1000 BC), Denmark (500 - 1 BC) and Britain (400 - 230 BC) (Bennike, 1999; Mays and Taylor, 2003; Rokhlin, 1965). Austria and Lithuania report evidence of tuberculosis by the 4th century AD. Other Northern European countries do not provide evidence of skeletal tuberculosis until the medieval period. In both the Mediterranean and Northern European regions, the frequency of skeletal tuberculosis appears to increase in the later medieval period. Roberts & Buikstra (2003) attribute this rise to an increase in urbanization and suggest that the aggregation of populations created ideal conditions for the spread of tuberculosis.

In Asia, evidence for tuberculosis does not appear until much later. The earliest reported skeletal cases date to 454 BC – 124 AD from Japan (Suzuki and Inoue, 2007). Evidence from Thailand is reported for a slightly later period (300 BC – 200 AD) (Tayles and Buckley, 2004); however, there may be earlier evidence dating to 1400 – 900 BC (Douglas, 1996; Higham, 2002; Pietrusewsky, 1976). In China, the earliest skeletal evidence for tuberculosis dates to 200 BC (Suzuki and Inoue, 2007). Evidence for the presence of tuberculosis prior to European contact has also been reported for more recent periods from Hawaii (Pietrusewsky, 1976; Pietrusewsky and Douglas, 1994; Trembly, 1997), Papua New Guinea and, possibly, Tsonga and the Solomon Islands (Stone et al., 2009).

With the exception of Egypt, there is no information concerning the presence or absence of pre-colonial skeletal tuberculosis in Africa (Roberts and Buikstra, 2003; Stone et al., 2009).

Evidence for skeletal tuberculosis pre-dating European contact has also been reported for the New World. The presence of pre-Columbian tuberculosis in the Americas was for some time a controversial topic; however, mounting skeletal and genetic evidence has largely settled this debate. The earliest New World skeletal cases are from South America, specifically, the Atacama Desert of northern Chile and coastal southern Peru (Allison et al., 1981, 1973), and date to at least AD 700. While these two regions also contain the highest frequency of South American skeletal cases of tuberculosis (Stone et al., 2009), additional evidence has been reported from Venezuela and Columbia (Roberts and Buikstra, 2003). In North America, Roberts & Buikstra (2003) identify two clusters of skeletal cases – one in eastern North America and the other in the Southwest. Evidence has also been reported from Mexico. All North American cases post-date AD 900. No skeletal cases of tuberculosis have been reported for Mesoamerica (Roberts and Buikstra, 2003).

In summary, the skeletal evidence for tuberculosis in the Old World indicates an early focus of the disease in parts of the Mediterranean and Northern Europe with a later appearance in others parts of these regions and in Asia. There is also an increase in frequency of reported cases during the later Medieval period. In the New World the earliest evidence for tuberculosis appears in South America and, later, in North America. Holloway, Henneberg, de Barros Lopes & Henneberg (2011) provide a map showing the geographical location of skeletal tuberculosis cases which indicates a notable concentration of reports from Europe and North America. (Roberts and Buikstra, 2003) suggest that regional differences are most likely a reflection of relative research intensity, but that other possibilities cannot be excluded. These include a genuine presence of the disease in some regions and absence in others, differential access to skeletal material (e.g. poor preservation, destructive mortuary rituals, ethical issues that may exist in some regions but not others), differences in host immunity (which could be environmental or genetic in origin) or differences in mycobacterial strain virulence (Roberts and Buikstra, 2003).

TUBERCULOSIS IN THE WRITTEN RECORD

Written sources can also be used to elucidate the history of tuberculosis. It is, however, important to note that not all populations developed a written language and for many its adoption has been a relatively recent event. As a result, the contribution of this resource will be limited and may provide a skewed picture. Furthermore, the reliability of diagnoses and descriptions can be questioned based on the extent of medical knowledge in the past. Thus, there is always the possibility that the descriptions/terms used were not specific to tuberculosis and that another disease may have been responsible for the observed symptoms.

The earliest written evidence alluding to tuberculosis, which also describes treatment of the disease, comes from a Chinese medical text dated to 2700 BC (Roberts and Buikstra, 2003). Other early written descriptions come from the *Rig Veda*, a Sandskrit holy text discovered in India, and an ancient Egyptian medical papyrus, both of which date to ~1500 BC (Evans, 1998). Many classical writers of ancient Rome and Greece describe the disease (Roberts and Buikstra, 2003), as do the Arabian writers, Rhazes (AD 850-953) and Avicenna (AD 980-1037) (Morse, 1967). Little is known about pulmonary tuberculosis during Medieval times; however, scrofula (a form of tuberculosis affecting the lymph nodes) appears to have been common in Europe (Chalke, 1962), with many sufferers seeking to be cured by the king's touch. The frequency of the disease is difficult to assess until the 17th century when deaths due to consumption were more routinely recorded in parts of Europe (Walford, 1878). Based on the London Bills of Mortality, tuberculosis appears to have been rife between the 17th and 19th centuries and was often alluded to in contemporary fictional writings (Chalke, 1962). Tuberculosis prevalence in England appears to have reached its peak during the 19th century (Chalke, 1962)

In the latter half of the 19th century a decline in the prevalence of tuberculosis was noted in England. In fact, while differences in timing exist, this trend has been observed throughout the developed world. Importantly, this decline precedes the use of anti-tuberculosis drugs, which were only introduced in the mid-1940s, and widespread use of the bacille Calmette-Guerin (BCG) vaccine. The reasons for this decline are debated but include reduced transmission through isolation of the sick in Sanatoria and/or improved living conditions and sanitation, reduced susceptibility to disease through improvements in diet or genetic adaptation and, finally, a reduction in *M. tuberculosis* virulence (Dye and Williams, 2010). The introduction of chemotherapeutic agents greatly reduced tuberculosis mortality and led to hopes of complete disease eradication in developed countries. However, by 1990 it was clear that tuberculosis was still a major global health concern and a re-emerging threat in developed nations. This resurgence had been spurred by the rise of the HIV/AIDS pandemic, declining socio-economic conditions in some regions of the world, increased drug resistance and ease of global migration (Dye and Williams, 2010).

Now that we have described the global history of the disease, we can examine the South African record. Before we discuss the history of tuberculosis in South Africa, it is necessary to first discuss a brief history of the country and its people.

A BRIEF HISTORY OF THE PEOPLING OF SOUTH AFRICA

When European settlers arrived at the southern tip of Africa, the region south of the Orange and Limpopo rivers (which roughly dictate the boundaries of modern day South Africa) was populated by communities that have traditionally been separated into three major groups differentiated by a combination of shared population history, language, technology, subsistence strategy and ideology. The western region of South Africa was occupied by hunter-gatherer and herder populations popularly referred to as San and KhoeKhoe, respectively (or Khoesan collectively), while the eastern region was occupied by Bantu-speaking agro-pastoralists. In the context of southern African archaeology, the Khoesan are also frequently referred to as Later Stone Age (LSA) populations and Bantu-speaking agro-pastoralists as Early/Late Iron Age (EIA/LIA) populations despite a temporal overlap. While these terms clearly do not represent true temporal “Ages” their use has persisted in southern African archaeology. This section is not intended to be an authoritative description of the history of South Africa but rather an introduction to the origin of its people, population densities and movements across the landscape through time.

South Africa has a rich skeletal record of modern human occupation. While this does not necessarily equate to population continuity through time, cranial morphometric and dental studies do suggest biological continuity among hunter-gatherer populations at least throughout the Holocene (Stynder et al., 2007a, 2007b). Prior to the adoption/arrival of food production/producers, hunter-gatherers were distributed throughout modern day South Africa and lived in small, mobile groups. Group size may have been fluid and variable but was generally small (Hobhouse, 1956; Wadley, 1987); however, the degree and uniformity of mobility, particularly during the Holocene, has been questioned. The geographic distribution and density of hunter-gatherer populations (estimated from archaeological sites) appears to have varied throughout the Holocene. Between 8 000 BP and 4 000 BP hunter-gatherer sites occur infrequently in the interior (Deacon, 1974; Mitchell, 2002; Sealy, 2016). This has been interpreted as a depopulation of the interior and may be related to climatic changes that resulted in warmer, dryer conditions in the interior during this period (Deacon, 1995). Given that there is no evidence of a concomitant increase in population density at the coast, general population decline throughout the region is assumed (Sealy, 2016). From about 4 000 BP onwards, as climatic conditions ameliorate, hunter-gatherer population densities increase (Jerardino, 2010; Parkington et al., 1988) and archaeological sites in the interior increase in frequency (Deacon, 1984, 1974). Mitchell (1997) argues that there has been too great an emphasis on drought. He highlights the climatic variability in the region during this time and argues that a greater understanding of other environmental or cultural factors is needed to understand the decrease in population density.

The earliest evidence for the production of food appears in South Africa at ~2 000 BP (Henshilwood, 1996; Pleurdeau et al., 2012; Webley, 2001) in the form of imported domesticated livestock and thin-walled pottery. The hunter-gatherers of southern Africa did not develop an independent means of food production. Instead, food production in southern Africa is associated with both LSA herder and Iron-Age, agro-pastoralist populations. Analysis of the archaeological evidence indicates that the livestock and pottery that had reached the western region of South Africa at ~2 000 BP cannot be attributed to EIA agro-pastoralists who first appear ~500 years later in the eastern region of the country. Given that the domesticated livestock was not of local origin, there has been considerable debate concerning the origin of herders in South Africa. This debate has centred around two main arguments: (1) that LSA herders represent an immigrant population that brought a "package" of livestock and ceramics with them (Ehret, 1982; Elphick, 1985, 1977; Smith, 2008; Walker, 1983), or (2) that LSA herders represent local hunter-gatherer populations that adopted livestock and ceramics through some form of cultural diffusion (Deacon et al., 1978; Sadr, 1998). Genetic (Breton et al., 2014; Henn et al., 2008; Macholdt et al., 2015, 2014; Pickrell et al., 2014, 2012; Ranciaro et al., 2014; Tishkoff et al., 2009), linguistic (Güldemann, 2008; Haacke, 2008) and archaeological evidence (e.g. distinctive rock-art, large OES beads size, high density of ceramics, domesticate remains and informal lithic artifacts, and simultaneous arrival of both livestock and ceramics) (Smith et al., 1991; Smith and Ouzman, 2004) have been put forward as evidence for the migration of LSA herders into South Africa. Furthermore, Smith (2016) has argued that the conflicting world views associated with hunting and herding would preclude an easy adoption of stock-keeping by hunter-gatherers. Conversely, similarities between hunter-gatherers and herders, with respect to material culture (Schrire, 1992) and population continuity (Morris, 2003; Soodyall et al., 2008; Stynder, 2009; Stynder et al., 2007a, 2007b; Uren et al., 2016), as well as the diversity in the decoration of thin-walled pottery (Sadr and Sampson, 2006) argue against the arrival of a large immigrant population and rather suggest an adoption of certain cultural attributes by local hunter-gatherer populations. A number of scholars have suggested that the available genetic, linguistic and archaeological evidence is best explained by combining these arguments i.e. that livestock and pottery-making were adopted by some local hunter-gatherer populations, with a small input from immigrants (Deacon, 1984; Parkington and Hall, 2012; Sadr, 2018, 2013). Based on historical observations herder settlement sizes were variable, consisting of 30-50 huts on average (Hale, 2007) but there are descriptions of larger settlements (a few hundred huts) with large numbers of stock (Boonzaier et al., 1997; Elphick, 1985; Kolbe, 1917). It is difficult to determine whether this was also the case in the first millennium due to the ephemeral nature of archaeological herder sites and the difficulty in distinguishing between hunter-gatherer and herder sites (Sadr, 2008; Smith, 2008). Based on archaeological and historical sources the pre-colonial distribution of herder populations were concentrated along the western and southern coastal regions of South Africa, the Orange River valley and Limpopo region. Herder occupation of parts of the interior and along the eastern coast was possibly restricted by inferior grazing opportunities and the presence of migrating EIA agro-pastoralists. However; our knowledge of the distribution of herder populations may be limited by our ability to detect these sites archaeologically.

As was mentioned previously, the Iron Age populations of South Africa were also food producers who, in addition to herding livestock, also cultivated summer rainfall crops. In this case the immigration of a morphologically and genetically distinct population with a "package" of livestock, agriculture, ceramics and iron technology is well supported (de Filippo et al., 2012; Huffman, 1982; Tishkoff et al., 2009). Archaeological, genetic and linguistic evidence support a West/Central African origin for these migrating populations (Huffman, 1982; Salas et al., 2002) and, although numerous scenarios (differing in the routes followed and timing of migrations) have been proposed, a recent genetic study has shown greatest support for the West-East-South model (Li et al., 2014). This model proposes an eastwards migration to East Africa before a southerly migration into Southern Africa; however, the authors note this model was only marginally better supported than the other models under investigation. In southern Africa the archaeological evidence is best explained through multiple migration events by people sharing a recent common ancestry (Parkington and Hall, 2012). Based on ceramics analyses, the earliest evidence for EIA populations in South Africa is dated to AD 250-450 (Hall and Vogel, 1980; Parkington and Hall, 2012). These early 1st millennium sites were initially restricted to the eastern coast - favouring high rainfall regions and proximity to sources of iron ore. During this period population densities were probably low and villages unevenly distributed across the landscape (Parkington and Hall, 2012). Around 900 AD a new Iron Age population, responsible for Zhizo ceramics, migrated into the region where the Shasha and Limpopo rivers meet. This was followed, in the 11th century, by the migration of new populations, thought to be ancestral Shona-speakers, into the region. Importantly, during this period (900-1290 AD), the communities of the Shasha-Limpopo river valley underwent an increase in political, social and economic organization that has largely been attributed to an increased intensification of long distance trade with the East African coast and an increase in population density. The result was the development of the Zimbabwe Culture; a period that has been further subdivided into three based on its major capitals – Mapangubwe (AD 1220-1290), Great Zimbabwe (AD 1290-1450) and Khami (AD 1450-1820) (Huffman, 2000, but see Chirikure et al., 2017 for a critique). Elsewhere, new populations had migrated into South Africa. Ancestral Nguni-speakers had reached the coastal regions of KwaZulu-Natal by AD 1100 and, by AD 1300, ancestral Sotho/Tswana-speakers had reached the region surrounding the Soutpansberg. This was followed by steady expansion southwards throughout the 14th and 15th centuries. By the 17th century Sotho/Tswana settlements had extended south of the Vaal River into the grasslands of the Free State. Similarly, between the 14th and 18th centuries, Nguni settlements continued to expand, first extending further inland from the KwaZulu-Natal coast and later moving across the escarpment where they encountered Sotho/Tswana communities (Huffman, 2004). Further westerly migration was restricted by the boundaries of the summer rainfall region, which is bordered by the dry Karoo, while the southerly migration of Nguni farmers extended up to the Great Fish River. The 16th to 19th centuries also saw the development of new socio-political identities some of which can be traced to modern times through colonial text and indigenous oral histories. Early in the 18th century a trend of increasing settlement size began to emerge. By the late 18th and early 19th centuries, this settlement aggregation culminated in the large Tswana towns observed by early European travellers (Huffman, 1986; Maggs, 1976). As an example, estimated population sizes for Marothodi, Molokwane and Kaditshwene (early 19th century Tswana settlements) were 7 000, 12 000 and 16 000, respectively (Campbell, 1822; Mason, 1986; Pistorius, 1992). This aggregation has conventionally been identified as a defensive response to the *Mfecane*/*Difaqane* (time of troubles) - a period of great unrest among the LIA populations of South Africa. The origins of this period of war, famine and mass displacement can be found in the preceding century. The introduction of maize, population growth, climate change and the expanding influence of Europeans (both from the Cape Colony and Delagoa Bay) may have all contributed to the events of the 19th century (Eldredge, 1992; Hannaford et al., 2014).

In 1487, the Portuguese mariner, Bartholomew Dias, set off on an expedition to find a trade route to the East and, early in 1488, rounded the southern tip of Africa travelling as far north as Algoa Bay on the South African coast before returning to Portugal (see Thompson, 2001). Ten years later, Vasco da Gama succeeded in reaching India – opening up a sea route to the East that allowed direct European trade for the next few centuries. Thus, the Portuguese were the first documented European sailors to reach the South African coast and interact with the local indigenous populations they encountered. Portuguese interest, however, was with trade in the East and along the East African coast and their interaction with the indigenous people of South Africa was limited (largely due to negative experiences navigating the coastline and violent interactions with the indigenous people they encountered) (Thompson, 2001). During the 16th century Portuguese vessels made regular trips to the East and, by the end of the century, Dutch, English, French and Scandinavian trading vessels were also making use of this sea route. Occasionally, ships were damaged and lost or would land along the southern coast to replenish supplies and barter with the indigenous people; after spending months stranded at Table Bay, it was shipwrecked Dutch mariners who suggested occupation of the region. Thus, in 1652, tasked by the Dutch East India Company (*Vereenigde Oost-indische Compagnie* or VOC) to establish a refreshment station, the first Dutch settlers arrived at Table Bay. A large settlement at the Cape was never the initial intention of the *Heeren XVII* (the VOC board of directors); however, the size of the colony began to increase steadily after a number of company employees, released from their contracts, were granted free-holdings and the VOC approved the importation of slaves. The slave population of the Cape comprised a broad mix of Africans and Asians from East Africa, Madagascar, India and Indonesia (Shell, 1994). In 1707, there were 700 company employees and 2 000 free-burgers (Thompson, 2001). By 1711, the number of slaves exceeded those of free-burgers (Worden, 1985). Both populations continued to increase at a roughly similar rate but the free-burger population never again exceeded that of the slave population while under Dutch rule (Worden, 1985) and by 1793, there were 14 747 slaves and 13 830 free-burgers on record in the Cape Colony. Similarly the area of occupation had expanded from the Cape Peninsula in 1769 to much of the Western Cape by 1779. Much of this land appropriation was done by the *trekboers*, mobile pastoralist farmers of European descent, who occupied most of the land outside of the arable Southwest. The Colony remained under Dutch rule until it was captured by the British, first in 1795-1803 (whereafter, it returned to Dutch rule) and then again from 1806. At the time of initial British occupation, Cape Town was still the only official port of entry into the country. Under British rule the colony continued to expand. Close to four thousand immigrants from Britain, Wales, Scotland and Ireland – who later became known as the 1820 settlers - were granted land along the eastern frontier as part of the British plan to drive out the Xhosa, bolster the number of white settlers at the frontier zone and appease social and economic unease in the United Kingdom (UK) (Thompson, 2001). At first many towns were established in proximity to the coast linked to ports or at defensive outposts. Later with the migration of the *Voortrekkers* and the discovery of minerals, European migration into the interior increased dramatically. The *Voortrekkers* were a group of Europeans (largely of Dutch, German and French Huguenot descent) that embarked on a relatively large scale emigration (amounting to ~10% of the white population of the Cape Colony) in the 1830's and 1840's to escape the British-dominated politics of the Cape colony. The *Voortrekkers* and their descendants spread over much of the country eventually establishing the independent *Boer* republics of Natalia (Natal), the South African Republic (Transvaal) and the Orange Free State. All three republics were eventually annexed by the British and together with the Cape colony were united in 1910 when the Union of South Africa was formed. After the British annexation of Natal in 1844, a large number of Afrikaaners (as South Africans of Dutch descent became known) left in protest, and many British immigrants began to settle in the region. Furthermore, from 1860, a large number of Indians were brought to Natal to work as indentured labourers on the sugar plantations. Most remained in South Africa after their service had ended, resulting in a sizeable Indian population. In 1867 diamonds were discovered in Kimberley, and in 1886 gold in the Witwatersrand of the Transvaal. The discovery of minerals resulted in a dramatic influx of immigrants into the interior and mining became the driving force of the South African economy. The mining industry spurred larger scale industrialization in South Africa, the development of the rail system and a need for cheap labour. With continued industrialization of the Union, racial division of the labour force extended from the mining industry to the manufacturing industry and policies of racial segregation and discrimination became more entrenched. The election of the National Party in 1948 saw these policies become formalized in law under Apartheid. In 1961 South Africa became a republic cutting its last major political ties with Britain. Finally, in 1994, the first free and fair democratic election, in which all South African citizens could vote, saw an end to the legal oppression of its non-white citizens.

The colonial period was a very turbulent time for indigenous populations. The Khoesan, under pressure from the expanding colonial frontier in the southwest and LIA argro-pastoralists in the east (and the resultant loss of land, warfare and disease (notably smallpox)), either perished, fled (usually to environmentally less hospitable areas) or were assimilated into neighbouring Iron Age communities or the lower classes of settler society. By the early 20th century the Khoekhoen had largely ceased to exist as an independent political entity within South Africa. While the San, greatly reduced in number, continued to reside in the northern-most regions of South Africa, Botswana and Namibia. Under Apartheid the remaining South African Khoesan were designated "Coloured" – a racial category established by the Apartheid government referring to individuals of mixed (European, Khoesan, Bantu-speaking and slave) ancestry. Bantu-speaking agropastoralists also faced increasing pressure from the expanding Cape Colony and Portuguese influence in Mozambique, population increase and climate change (Eldredge, 1992). This culminated in the much publicised '*Mfecane*/*Difaqane*' – a period of great unrest in the first half of the 19th century that resulted in massive population displacement, famine and war. As a consequence many were forced to seek subsistence in the Cape Colony. As the 19th century proceeded, Africans found themselves increasingly drawn into colonial activities and interests; and African social, economic and political autonomy was being eroded on multiple fronts. The discovery of minerals in Kimberly and the Witwatersrand, in addition to drawing large numbers of white immigrants, also attracted many black workers and throughout the 19th and 20th centuries urbanization continued. This was fuelled in part by a number of policies and laws that served to dispossess indigenous peoples of their land and restrict the subsistence options of the rural population. The native reserves, and later under Apartheid, the ten homelands were created in an attempt to limit black occupation in the urban centres and control the labour force. Even within the cities racial groups were segregated with coloured and black townships often located around the outskirts. Today, even after the first democratic elections of 1994, the social, economic and physical segregation largely persists as a legacy of Apartheid.

This section has served to highlight the complexity of interaction among different groups on the South African landscape through time as a foundation for understanding the emergence and spread of tuberculosis.

PRE-COLONIAL TUBERCULOSIS IN SOUTH AFRICA?

It is clear from the global history of tuberculosis that the disease has plagued humans for at least 6 millennia and was present in North Africa by 4500 BC. However, little is known about the history of the disease in sub-Saharan Africa (Roberts and Buikstra, 2003). The indigenous people of South Africa did not develop a writing system. Therefore, to investigate the presence of pre-colonial tuberculosis we must rely on early explorer, missionary and settler accounts. These accounts are based on the observations of individuals or their recordings of oral histories, and are, therefore, by definition anecdotal. In addition, a number of articles and reports have investigated the topic of pre-colonial tuberculosis in South Africa (Collins, 1982; McVicar, 1908; Metcalf, 1991; Packard, 1989a; TBRC, 1932; U.G., 1914; Wilcocks, 1962). The contributions of these sources, and others, to our understanding of pre-colonial tuberculosis in South Africa, will be discussed below.

Most early travellers make no reference to tuberculosis when discussing the indigenous populations of South Africa. Some note that disease in general is rare, while others specify that chronic disease is rare (de Saint-Pierre, 1773; Kolben, 1927). Furthermore, those that do list observed ailments do not mention tuberculosis. For example, Barrow, who in 1798 travelled to the South African interior, writes that tuberculosis is frequently observed in the European population but does not mention the disease when discussing the health of the indigenous communities he encountered (Barrow, 1801). Similarly, Dr Lichtenstein (1812), who recorded his travels from 1803 to 1806, also does not mention tuberculosis when discussing the illnesses affecting the Xhosa, for which he says diseases are rare. A few historical sources do specifically note the absence of tuberculosis among indigenous populations. Dr Livingstone (1857), in his *Missionary travels and researches in South Africa*, specifically notes the absence of tuberculosis among the communities he met in the interior; however, instances like these are rare.

The earliest reference to the disease can be found in the writings of Cornelius de Jong (1802), the Commander of a Dutch man-of-war, who visited the Cape between 1791 and 1795. He lists consumption as one of the most prevalent diseases encountered at the Cape; however, it is unclear from this description whether he is referring to a specific segment of the population (Europeans or those of European descent) or to the Cape population in general. Captain Robert Percival (1804), who visited the Cape in 1796-1801, writes that, “Consumption and ulcers seemed the only distempers attended to any extent with fatal consequences to our countrymen.” Thus, the earliest historical references to tuberculosis likely refer to disease in the European population. Referring to the Khoesan, Laidler and Gelfand (1971) state that pulmonary consumption was frequent in the late 18th century and, by 1821, was present in the Koranna tribes of the Orange River. The earliest reference to tuberculosis in the Bantu-speaking population is in JWD Moodie’s *Ten years in South Africa 1820-1830* (Packard, 1989a). Here Moodie describes an encounter, near Grahamstown, with the son of the Chief of Tzatzoe who was dying of consumption (Moodie, 1835). Moodie also reports that the disease is uncommon among those of European descent but is common among the Khoesan.

McVicar's (1908) thesis was the first comprehensive report on the subject of pre-colonial tuberculosis among the indigenous populations of South Africa. In his enquiries he sourced or requested reports from hospitals and journals, as well as doctors, missionaries and officials based in the major towns and selected rural areas in South Africa. These reports generally state that, outside of the Cape Colony, tuberculosis was unknown or uncommon among the indigenous population when these sources first had contact with them (the date varies from region to region – 60 to 15 years prior). Most note that the frequency of disease had only begun to increase relatively recently, in the last decade or so, and that tuberculosis would soon become a serious problem among the rural population if the current trend continued unabated. The presence of tuberculosis among the indigenous people of the Cape Colony, prior to large scale European contact, was more difficult to assess given the longer history of European settlement in this region. Despite this, McVicar concludes that tuberculosis may have been present among the Khoesan prior to European contact given the high prevalence of, and their long history with, the disease.

Evidence from Natal provides another exception to the general conclusion that tuberculosis was absent prior to European contact. In that region, reports on the presence of tuberculosis prior to European settlement were varied. While many came to the same general conclusion as stated above (that tuberculosis was unknown until recently), some sources claimed that the disease was known to the Zulu of Natal prior to European contact. For example, (McVicar, 1908) includes two sources in his report that state tuberculosis was present among the Zulu when they first arrived, 40 – 50 years earlier. Others note that, when questioned, individuals state the disease "has always been with us" or has been with them "as far back as can be traced" and is called *isifuba*. McVicar notes that most of the evidence supporting pre-colonial tuberculosis comes from the coastal regions, while most of the contradicting evidence hails from further inland. In his concluding remarks, he dismisses the evidence favouring a pre-colonial presence of tuberculosis arguing that the linguistic terms are too generalized and that the uneven distribution is indicative of a recently introduced disease. In support of this, he highlights the uniform distribution of tuberculosis in countries (such as England) where tuberculosis is well established. He does, however, concede that tuberculosis may have arrived in Natal with the immigration of the bantu-speaking population who, he believes, likely acquired the disease from the Indians or Portuguese further north along the east coast. McVicar, addressing the reasons for the rapid increase in tuberculosis among indigenous populations in recent times, states that within the Cape Colony, the rising prevalence can be attributed to an increase in the number of tuberculous immigrants entering the port and visiting the warm and arid regions in search of a climatic cure for their disease. However, in regions not frequented by consumptive immigrants and, in particular, the rural areas, the rise in prevalence was repeatedly attributed to the return of men from the mines who, having contracted the disease at their workplace, proceeded to infect their families and others in the community.

A few years later, in 1912, the Tuberculosis Commission was established to investigate the extent of tuberculosis in South Africa and to find solutions to stem the alarming rise in prevalence among the indigenous population. That same year the Commission published its first report dealing with the admission of tuberculous immigrants. In this report the Commission states:

> From the evidence before us it would appear that although Consumption had existed to some degree for long past in the South African Sub-Continent, it is only within recent years that it has laid its grip upon certain portions of the community and assumed its present menacing proportions ….

No further details are provided in this report. However, two years later, in the 1914 Report of the Tuberculosis Commission, the issue of pre-colonial tuberculosis was dealt with in much greater detail. The commission, in its opening statement, states:

> In view of its extensive occurrence from time immemorial in Europe and in Asia, it is reasonable to suppose that the disease must have been occasionally introduced into South Africa by Arab traders and other immigrants from the North and the East, communication with which regions had been carried on for many centuries. And later on, after the discovery of the Cape route by Bartholomew Diaz in 1486, Europeans must have brought it from time to time to the Cape and East Coast ports.

This statement acknowledges earlier opportunities for disease transmission that precede the 17th century Dutch settlement at the Cape. The commission, however, did not believe that the disease existed to any great extent among the indigenous population and provide three reasons for this supposition. Firstly, if it had, the early explorers, missionaries and settlers would have mentioned it in their writings. Secondly, the 'prominence' (i.e. high frequency of occurrence) of tuberculosis in South Africa was a comparatively recent event. Lastly, while they concede that tuberculous sailors and settlers probably visited the Cape early on, the large scale importation of tuberculosis through the immigration of consumptives seeking a climatic cure was also a relatively recent practice, having only begun 30-35 years earlier. The committee provided a more extensive review of the early literature than that provided by McVicar but embraced a similar method for obtaining reports from contemporary sources. The conclusions of these reports were identical to those in McVicar's thesis. Again, emphasising that tuberculosis was rare or unknown among the indigenous populations prior to European contact and that, in certain regions, prevalence was now rising at an alarming rate. A number of reports from Grahamstown indicate tuberculosis was present 30-50 years earlier but it should be noted that Grahamstown, like Cape Town, has had a relatively long history of European occupation. Furthermore, these reports indicate prevalence was higher among communities with greater sustained contact with Europeans. The commission provides only one contradictory report from Natal. The source had been visiting this region for the past 40 years and states that *isifuba* was "the great complaint" among the Zulu of Eshowe. In conclusion, the commission states that tuberculosis was not prevalent in South Africa a few generations prior to the publication of the report (1914) and that the belief in its recent introduction is too widespread to disregard. Prevalence among the European population was always low (if imported cases are excluded) and the worst affected indigenous communities were those with the longest history of close European contact, i.e. the Khoesan and their descendants of mixed ancestry. The commission, discussing the causes for the rising prevalence of tuberculosis, attribute this to the immigration of consumptives from Europe and the advent of industrialization. Despite the frequent assertion by contemporary sources in the report that returning mineworkers were the main source of infection in rural communities, the commission does not address this issue. The mines, as a cause for the spread of tuberculosis, are only referred to as the main stimulus of industrialization and the increase in urbanization and mobility that occurred as a result.

The Tuberculosis Research Committee was established in 1925, partially funded and commissioned by the government and the Chamber of Mines, and published their first report in 1932. The purview of this Committee was much the same as that of the Tuberculosis Commission, which was unable to reach a unanimous conclusion on how to deal with the problem of tuberculosis in the indigenous population or provide recommendations. The report of the Tuberculosis Research Committee (TBRC, 1932) was the first to discuss in detail the “virgin soil” hypothesis and racial susceptibility of the indigenous population. These hypotheses argue that ‘isolated’ communities, having little or no experience with a particular disease, are usually more adversely affected when exposure occurs than those communities with a long history of the disease due to their immunological naivety or inherent, heritable susceptibility. In essence, the Committee discusses the concepts of genetic adaptation and acquired immunity and the role of these processes in understanding the susceptibility of the indigenous population to tuberculosis. While the Committee favoured the idea that the indigenous populations of South Africa represented an ‘isolated’ population with little or no experience of tuberculosis, they reach no firm conclusion on the idea of racial susceptibility and do not overlook the role of changing environmental circumstances on the morbidity and mortality of the disease in the indigenous populations. The report adds little else to the discussion of pre-colonial tuberculosis and most of what follows in the report is a review of previously published reports. The Committee's conclusions are in line with those of previous reports stating that the disease would have been noted by early observers had it been present. In addition, however, they state that the conditions necessary for the maintenance and spread of the disease were not present prior to large scale European settlement and that the nature of tuberculosis among Africans is not characteristic of a population with a long history of the disease.

A number of review articles and book chapters deal with the subject as well. Collins (1982), in his paper on *The history of southern Africa's first tuberculosis epidemic*, also states that, while the East African and Portuguese trade may have provided opportunities for occasional transmission of tuberculosis, the, "… conditions favourable for the spread of the disease did not exist," attributing this to the lack of "overcrowded cities or industrial complexes" and "major outside intrusion into the interior". Metcalf (1991) asserts that tuberculosis is a relatively new disease in southern Africa and considers it unlikely that the disease existed to any great extent prior to European colonization.

Reports for other regions in Africa, although usually less detailed and widespread, are very similar to those just described for South Africa. While temporal variation in the onset of the epidemic exists from region to region, most reports state that tuberculosis was unknown or rare among indigenous populations when first encountered by European observers. The above is true for Swaziland, German South West Africa (Namibia), Bechuanaland (Botswana), Portuguese East Africa (Mozambique), Southern Rhodesia (Zimbabwe), Northern Rhodesia (Zambia), Kenya, British Central Africa (Nyasaland/Malawi), Uganda and Nigeria (Collins, 1982; McVicar, 1908; U.G., 1914). A pre-Colonial presence of tuberculosis is conceded for countries involved in Indian Ocean trade i.e. Uganda, Mozambique and Zimbabwe (although it should be noted that the idea of an Arabic or Phoenician origin for Great Zimbabwe was prevalent at the time). While in some regions the spread of the disease is linked to European contact, in others the rise in tuberculosis prevalence was mainly attributed to the return of workers from the South African mines and increased urbanization resulting from industrialization.

In summary, all previous reports conclude that tuberculosis was either absent or was present but at very low prevalence prior to colonialization. In addition, all the reports acknowledge that transmission opportunities existed prior to colonial settlement at the Cape. While early reports argue that if the disease was present, it was at too low a level to be noticed by early travellers, missionaries and settlers, later reports argue that the conditions necessary for the maintenance and spread of the disease were lacking. The uneven distribution of tuberculosis across the landscape and the susceptibility of the indigenous population, it is argued, provide additional support for the idea that tuberculosis was a new disease to the indigenous people of South Africa and was only introduced with European settlement. Much has changed since the publication of these reports with regards to our understanding of the global and genetic history of tuberculosis, the factors that influence its maintenance and spread, the archaeology of pre-contact populations and the influence of racial biases in attributing disease prevalence/susceptibility. The following section discusses the validity of the explanations for the lack of tuberculosis put forward in the reports, in light of this more recent evidence.

PRE-COLONIAL OPPORTUNITIES FOR DISEASE TRANSMISSION

GENETIC EVIDENCE FOR THE AFRICAN ORIGIN OF TUBERCULOSIS

Genetic studies on modern *Mycobacterium tuberculosis* complex (MTBC) strains have identified Africa as the region with greatest genetic diversity and this has led many researcher to suggest an African origin for the MTBC (Comas et al., 2015, 2013; Gutierrez et al., 2005; Hershberg et al., 2008; Wirth et al., 2008). Specifically, East Africa has been identified as the most likely region of origin. However, it should be noted that The timing of the emergence of the MTBC has not yet been resolved (Brites and Gagneux, 2015). Hershberg et al. (2008), Wirth et al. (2008) and Comas et al. (2013) have hypothesized that the spread of tuberculosis is associated with the ancient global dispersal of modern humans out of Africa. Hershberg et al. (2008) describe an out-of-Africa-back-to-Africa scenario, whereby ancient strains leave Africa with the migration of modern humans. After a period of population growth, modern strains are introduced "back" to Africa and dispersed globally, largely as result of the extensive trade and migration that occurred after the age of exploration, to result in the current global distribution of modern strains. Two studies have provided dating estimates that support the above scenario. Comas et al. (2013) date the emergence of the MTBC to 70 000 yrs ago, while Wirth et al. (2008) produce a more recent date of 40 000 yrs ago. Bos et al. (2014), using ancient genomes to calibrate phylogenetic estimates, produce a much more recent date for the emergence of the MTBC. The substitution rates reported in this analysis are closer to those of modern epidemiological studies, and dating estimates limit the emergence of the MTBC to within the last 6000 yrs. Regardless of the length of association between the disease and its human host, tuberculosis appears to have an African origin despite the dearth of pre-colonial skeletal evidence for the disease in Africa. Thus, tuberculosis could have conceivably reached South Africa by pre-colonial transmission among adjacent populations, inter-regional trade networks or migrating agro-pastoralists.

PRE-COLONIAL TRADE NETWORKS

The Indian Ocean trade network provides one plausible route for pre-colonial disease transmission to South Africa. Archaeological investigations indicate that exotic trade items made their way to southern Africa by the 8th century AD (Wood, 2011). These items include beads and later ceramics from the Middle East, India, China and South East Asia (Wood, 2000). Pre-European trade was dominated by Arab traders largely from the Persian Gulf, who are known to have traded as far south as Mozambique (Wood, 2000). While in the northern regions of East Africa the penetration of trade items into the interior was poor, this was not the case in southern Africa (Wood, 2012). Here trade items, which likely entered at ports along the Mozambican coast (Sinclair, 1982; Wood, 2000), were highly sought after in the interior and facilitated the establishment of urban centres near the confluence of the Shashe and Limpopo rivers where South Africa, Botswana and Zimbabwe meet. At the end of the 15th century, the Portuguese had discovered the southern sea route to the east and, by the 16th century, had entered the Indian Ocean trade network. The

Portuguese dominated trade until the 17th century, whereafter Dutch and English commercial vessels took control of Indian Ocean trade (Chaudhuri, 1985). Within South Africa, the Bantu-speaking communities of the Shashe-Limpopo valley were predominantly involved in this external trade and there is no evidence of pre-European trade along the South African coast. After the Portuguese discovery of the southern sea route, occasional trade and shipwrecks along the South African coast resulted in European interaction with coastal South African populations.

Thus, South Africa was not isolated from Europe, Asia, or of course the rest of Africa prior to Dutch settlement at the Cape. Despite this, little is known about the history of tuberculosis throughout sub-Saharan Africa; however, the disease is known to have been well established in Eurasia and North Africa in antiquity (Roberts and Buikstra, 2003). While direct contact with foreigners may have been limited to the coastal regions of East and later South Africa, tuberculosis could have followed trade routes into the interior. Thus, given this early trade, it is possible that tuberculosis was periodically introduced to the indigenous people of South Africa prior to colonization.

TUBERCULOSIS AND EARLY EUROPEAN OBSERVERS

It was noted in many of the early reports that the presence of tuberculosis would have been mentioned by early observers had it been present to any great extent. While this view certainly cannot be excluded, Packard (1989a) highlights a number of issues which should be kept in mind when basing inferences on these accounts. Firstly, in most cases information concerning the prevalence of tuberculosis was not actively sought; rather, medical information was collected “passively”. For example, a disease was noted only if it was observed, although, in the case of tuberculosis, the absence of the disease was specifically noted in some early accounts. Even in these cases, it is not always possible to determine whether the observer was allowed access to sick individuals or if statements were based solely on observations. Secondly, indigenous people were often wary of western medicine preferring, instead, to seek cure and council from traditional healers. Thus, many were hesitant to seek European care and, as a result, cases of tuberculosis may have gone undetected. Lastly, Packard questions the reliability of these observations given that many observers were not medically trained, the aetiology of the disease was not understood and reliable methods for diagnosis were not available at that time.

An additional criticism is put forward by the Tuberculosis Commission (U.G., 1914) who, referring to their contemporary sources, state:

> Such evidence as the foregoing is open, of course, to the criticism that it mainly consists of impressions – in some cases, perhaps of the impressions of impressionable persons. And in this connection due allowance must be made for the fact that in recent years the subject of tuberculosis and its prevention has become a public topic all over the world, so that the most impartial observer may well be biased in his appreciation of the present as compared with the past.

This criticism speaks both to Packard's point that information was rarely actively sought and highlights another issue i.e. observers and their observations need not be impartial and may be influenced by the prevailing consciousness of the disease at a particular point in time.

CONDITIONS NECESSARY FOR THE MAINTENANCE AND SPREAD OF TUBERCULOSIS

As was mentioned above, there were opportunities for pre-colonial disease transmission but it is not clear whether these events would be self-limiting or would result in endemic disease. The transmission dynamics of tuberculosis are complex and its persistence in a population is dependent on a number of factors. In modern populations these include the nature, duration and frequency of contact; the physical environment in which contact occurs; population size and density; demographic, environmental and socioeconomic factors; factors affecting host susceptibility (e.g. host genetics, nutritional status and concurrent disease burden) and pathogen virulence (Lönnroth et al., 2009; Ozcaglar et al., 2012). Many of these factors are also relevant to past populations but are more difficult to assess. Both population size/density and the nature of the contact were implicated in historical and review texts as limiting factors in the acquisition and spread of tuberculosis. However, as is detailed below, these factors do not clearly play a limiting role in the South African context.

POPULATION SIZE AND DENSITY

Small population size and density has been used as an argument against the presence of tuberculosis in pre-urban societies, such as the pre-colonial hunter-gather groups of South Africa. Tuberculosis bears the hallmarks of a disease associated with both high and low population densities. Transmission patterns of modern tuberculosis indicate a disease dependant on high population size and density, while the long periods of latency indicate a transmission mechanism evolved under small population size and density (Blaser and Kirschner, 2007). Comas et al. (2013) suggest that initially small population sizes favoured disease transmission from one generation to the next. After the Neolithic demographic transition (NDT), larger, denser populations favoured a horizontal mode of transmission. If an older date for the emergence of the MTBC is accepted (Comas et al., 2013; Wirth et al., 2008), then the divergence of most lineages predates the NDT and small population size need not have been a major limiting factor. However, if the more recent date for the emergence of tuberculosis is accepted (Bos et al., 2014), then this would suggest that larger, more sedentary populations associated with the development of domestication and agriculture were necessary for the maintenance of disease. In contrast, transmission patterns of modern tuberculosis indicate that it is a crowd disease, dependant on high population size and density. After the Neolithic demographic transition (NDT), larger, denser populations likely favoured a horizontal mode of transmission (Comas et al., 2013).

Although pre-European southern African populations have traditionally been perceived as rural in character, archaeological investigations of the past few decades have revealed that this is not the case. Manyanga et al. (2010), however, note that pre-European urbanism has received relatively little recognition. For example, colonial authorities failed to recognise monumental architecture (e.g. Great Zimbabwe) as African in origin, instead attributing these structures/empires to foreign people. Throughout its occupation southern African settlement size and structure has varied among different populations and through space and time. Khoesan populations are typically small and fairly to highly mobile, whereas Bantu-speaking populations are usually more sedentary with greater temporospatial variation in population size and structure (Huffman, 1986). Early Iron Age settlements were relatively small and dispersed and this type of settlement has persisted to the present. However, by AD 900 larger, concentrated settlements began to appear (Manyanga et al., 2010). Schroda and K2 are two of the earlier larger settlements with an estimated population of 300-500 and 1000-2000 individuals, respectively (Huffman, 2000). Mapungubwe (AD 1220-1300) marks the development of the first major southern African state with an estimated 5000 individuals residing in its capital (Huffman, 2000). Greater still is the population estimate (between 11 000 and 18 000 individuals) for Great Zimbabwe (AD 1280-1550) (Manyanga et al., 2010). Chirikure et al. (2018) has, however, questioned the traditional view of linear state succession with large state capitals, arguing for a more complex and dynamic relationship among states with spatiotemporal overlap for most sites. In this argument, while larger populations are observed, the large population sizes reported for these sites (e.g. Huffman, 2000) do not represent single synchronous occupations but instead represent temporally overlapping populations. Population sizes for Tswana towns of the early 19th century were also relatively large, for example, Campbell (1822) estimated 16 000 individuals resided at Kaditshwene. Thus, sizable urban settlements did exist prior to colonization, although, not quite as large as has been previously reported. These larger populations could, in principle, have favoured increased transmission of tuberculosis.

THE NATURE OF CONTACT

The nature, duration, frequency and context of contact between susceptible and infectious individuals are also all important variables impacting the maintenance of tuberculosis in a population. These variables are difficult to determine in past populations, and South Africa is no exception. Prior to Portuguese entry into Indian Ocean trade, external trade with the southern African interior was controlled by the Swahili and direct interactions with foreign traders were likely limited to the East African coast. That said, little is known about the volume, conduct and organization of this trade until the mid-16th century (Pikirayi, 1993). After their arrival at the end of the 15th century, the Portuguese began to seize control of strategic Indian Ocean trading ports. To usurp the Swahili and gain control of the southern African gold trade a number of forts were established along the Mozambican coast and Portuguese presence eventually extended into the southern African interior (Pikirayi, 1993). Portuguese activities, however, were focused further north in what is now Mozambique and Zimbabwe. It is possible that as a result of this pre-European and early European trade, tuberculosis may have travelled along trade networks to reach the indigenous people of South Africa. Direct contact between South African indigenous communities and Europeans was likely limited to coastal regions, where shipwrecked sailors or traders would occasionally land, after the discovery of the sea route to the east. These interactions were varied – at times favourable, and other times violent. Thus, the nature, duration, frequency and context of contact with tuberculous individuals is a difficult subject to assess and would have likely also varied.

It should, however, be noted that the transmission dynamics of tuberculosis are complex and disease persistence is dependent on more than just population size/density. While relatively large urban populations may have existed, it is always possible that these were not sufficiently large or stable. Alternatively, many of the other factors necessary for disease persistence may not have been present.

DISTRIBUTION OF THE DISEASE ACROSS THE LANDSCAPE

McVicar (1908) argued that the uneven distribution of tuberculosis was indicative of its recent introduction. In support of this argument he states that in countries, such as England, where tuberculosis is endemic the disease is uniformly distributed. However, no statistical evidence is provided in support of that statement. The relationship between the spatial distribution of tuberculosis and endemicity is difficult to evaluate. In modern urban populations clustering exists and is often a reflection of socioeconomic geographic differences (Maciel et al., 2010; Munch et al., 2003). Zaman et al. (2006) investigated the geospatial prevalence of tuberculosis in a rural Indian population and also identified clustering, although the factors responsible were not determined. In light of the modern epidemiological understanding of tuberculosis transmission dynamics, McVicar’s argument is not valid. More likely, the geospatial patterning of tuberculosis is dependent on multiple factors including the nature and extent of interactions between populations, population size and socioeconomic status. Furthermore, geospatial pattern is also likely impacted by the reliability of historical observations.

THE VIRGIN SOIL HYPOTHESIS AND RACIAL SUSCEPTIBILITY

Prior to the discovery of the tubercule bacillus the aetiology of tuberculosis was a debated topic. The views of the 18th and 19th centuries included an external contagious source, morbid spontaneity (disease arising spontaneously within the body), a hereditary defect, atmospheric imbalances and stress (see Barnes, 2000; Kaufmann and Schaible, 2005; Murray, 2004). Even after Jean Antoine (Villemin, 1865) had demonstrated the successful transmission (by inoculation) of tuberculosis from humans to rabbits, scepticism of an external contagious source for tuberculosis remained (Murray, 2004a). It was only after Robert Koch's discovery, in 1882, of the microorganism responsible for the disease, that an external infectious source for tuberculosis was accepted by the broader scientific community (Murray, 2004a).

The existence of an external infectious source for tuberculosis did not, however, explain differences (real or perceived) in the impact of the disease among different population groups, including the often devastating effect of tuberculosis on newly contacted or colonized indigenous populations compared to their European counterparts. Attempts to explain these differences were framed by the prevailing scientific views of the time and included concepts of acquired immunity and racial susceptibility. Central to both arguments was that isolated indigenous communities lacked exposure to the disease and were, as a result, more adversely affected than their European counterparts who had exposure to or a longer history with the disease. Darwinian evolutionary explanations argued that susceptibility to disease was a heritable trait and in communities lacking exposure to tuberculosis, susceptible individuals had not yet been removed from the population through natural selection (see Packard, 1989; Worboys, 1999). This argument was particularly enticing after research indicated that the decline in tuberculosis in Europe preceded the improvements in standard of living and sanitation, to which this decline was frequently attributed (see Worboys, 2010). Critics, however, argued that this decline was too rapid to be explained through natural selection. Alternative explanations, which arose from vaccination studies and the field of immunology, stressed the role of acquired immunity. The "primitive tuberculosis" or "virgin soil" theory was popularized by the English physician, Lyle Cummins, who became one of the foremost experts on tuberculosis in the early 20th century. Cummins drew parallels between childhood tuberculosis and the experiences of African populations (Cummins, 1929). He argued that all infants represented "virgin soils" but through adequate childhood exposure were able to acquire immunity to the disease, which resulted in reduced prevalence and a more chronic expression of disease in adulthood. Conversely, isolated African populations lacked exposure and, therefore, immunity to the disease which explained the high prevalence, as well as, the acute and disseminated form. Cummins' view on the role of heritable susceptibility varied throughout his career. He initially favoured the argument of racial susceptibility then shifted to an argument based exclusively on acquired immunity. Later, in the 1930's he incorporated some aspect of inherited susceptibility only to reject it again a decade later (Worboys, 1999). Neither argument could ignore the role of socio-environmental conditions but rather incorporated these as mediating factors.

In South Africa, ideas of "primitive tuberculosis" and racial susceptibility were pervasive and persisted long after environmental arguments began to dominate views abroad. Packard (1989a) has argued that these "physiological" views suited the labour policies of the mining industry and, later, the separatist policies of the Apartheid government. Thus, they were promoted by the medical practitioners' dependant on these institutions for their livelihood. Furthermore, these views encouraged a fatalistic attitude to tuberculosis control (Packard, 1989a; Worboys, 1999). If the cause of the tuberculosis epidemic among Africans was due to an inherent physiological weakness, then intervention was futile. African populations would, given time, eventually acclimatise or become more resistant through natural selection. However, acceptance of these theories of racial susceptibility and acquired immunity was not universal. Others argued that poverty and the conditions it created were overwhelmingly the primary reason for the differences observed between races, irrespective of the roles of acquired immunity and racial susceptibility. McVicar (1908), for example, stated: "If racial predisposition be a factor in the spread of tuberculosis among the Native races of South Africa, I think it can safely be relegated to a secondary place, and the sanitary reformer may comfort himself with the reflection that, difficult though his task may be, he is at least not fighting against fate.” Furthermore, the generalizations on which these physiological theories were based were called into question. For example, Allan (1924) noted that in the rural “reserves”, in places where living conditions were better than in the cities, Africans were better able to resist infection and the disease tended to follow a more chronic course similar to that observed among Europeans. Others more openly rejected notions of racial susceptibility arguing for a solely socio-environmental cause for observed racial differences (Dormer et al., 1943; Dormer and Wiles, 1946). Echoing McVicars remarks, (Dormer and Wiles, 1946) state: “The fatalistic attitude which explains the severity of tuberculosis on the basis of racial susceptibility should be abandoned. The real causes are clear, and they can be remedied.” By the 1940’s environmental arguments began to emerge as the predominant view abroad. Physiological views fell out of favour as the validity of biological race concepts, particularly using skin colour as a determinant, were increasingly questioned and epidemiological studies increasingly linked socio-economic factors to differences among populations (Jones, 2003; McMillen, 2008; Worboys, 1999). Jones (2003) and McMillen (2008), referring to Native American populations, discuss these criticisms in greater detail but many of their arguments are applicable to African populations as well.

More recently, advances in the field of genetics have revived interest in the role of genetic susceptibility. Polymorphisms in a number of genes have been linked to greater tuberculosis susceptibility/resistance (see Azad et al., 2012) and some have suggested that differences in the frequencies of these polymorphisms may explain racial differences in susceptibility (Bellamy, 1998). It should be noted that the effect of these variants is modest and hints at epistasis among or polygenic inheritance of susceptibility genes. Furthermore, in most cases, the biological mechanism through which these genetic variants effect susceptibility/resistance remains elusive (Azad et al., 2012; Thye and Meyer, 2012). Without dismissing the role of a genetic component to tuberculosis susceptibility and disease progression, critics caution that many of these modern studies ignore the past discourse on racial differences in tuberculosis, make uncritical use of earlier research to support their arguments, underplay the role of socio-economic factors, and, finally, that these studies might undermine efforts to combat the disease on a social front (Barnes, 2000; Jones, 2003; McMillen, 2008).

Today there is a greater appreciation of the multifactorial aetiology of tuberculosis which involves a relatively poorly understood interplay between socio-environmental factors, as well as host and pathogen genetics. Epidemiological studies have indicated that, in many cases, differences in disease susceptibility, previously attributed to racial differences, can now be explained by differences in the risk of infection and disease progression created by socio-economic inequalities. Despite this, the assumption that certain populations with high tuberculosis prevalence and mortality represent ‘isolated’ populations, lacking significant exposure to the disease, is still uncritically accepted by many. Colonization resulted in both direct and indirect disruptions to the lives of indigenous populations. It is impossible to distinguish between the effects of the adverse conditions experience by indigenous populations after colonization and any inherent genetic or immunological susceptibility. In light of this, racial susceptibility and “primitive tuberculosis” cannot be accepted as supporting evidence for the absence of tuberculosis in South Africa. Based on phylogenetic dating estimates, Comas et al. (2015) have rejected the “virgin soil” hypothesis in sub-Saharan Africa (specifically East and West Africa) showing that the age of the most-recent-common-ancestor of some African MTBC lineages predates large-scale European contact. While this evidence may not directly extend to South Africa, this study does highlight the problem of associating a high prevalence and rapid/severe course of tuberculosis after colonization with a precolonial absence of the disease.

CONCLUDING REMARKS

The intention of this critique has not been to argue for a precolonial presence of tuberculosis in South Africa. Rather, the aim has been to highlight that arguments for its absence are largely based on an absence of evidence and to repeat the mantra “absence of evidence is not evidence of absence.” Furthermore, while many of the additional arguments may hold true, the premise on which they are based is often problematic. To conclude, McVicar (1908) in his thesis stated:

> So far as I am aware there is no actual evidence as to what diseases the Bushmen had previous to their contact with other races … but in view of the wide spread of tuberculosis through the world, it seems somewhat rash to conclude in total absence of proof, that it did not exist among any given people.

I adopt a similarly cautious approach and argue that in the absence of evidence all that can be claimed is that tuberculosis was either never present among the indigenous people of South Africa, was periodically introduced but did not become endemic or was present but went undetected by early settlers, explorers and missionaries.

HISTORICAL EVENTS AND PROCESSES THAT HAVE CONTRIBUTED TO THE MODERN TUBERCULOSIS EPIDEMIC

Arguably, the four historical events/processes that have contributed most to the modern tuberculosis epidemic have been the importation of disease by European immigrants, mining and the migrant labour system, urbanization and the HIV/AIDS co-epidemic. In the early colonial years, imported tuberculosis was a major threat to colonists and Khoesan/Khoesan-descendant populations. It was, however, the discovery of minerals in the late 1800s that played a major role in spreading the disease to the black population and disseminating the disease to the rural areas. The role of the mining industry in the tuberculosis epidemic is given particular attention here, due to the emphasis placed on its role in the historical literature, and the role it has played in shaping South African labour policies and migration. The rise in notifications after World War I (WWI) has been attributed to continued urbanization, and the decline of notifications in the mid-1960s, to the population segregation policies of the Apartheid government. Finally, the more recent role of the HIV/AIDS epidemic, as well as the threat posed by drug-resistant tuberculosis, is briefly discussed. Other factors, e.g. dairy industry, schools, railways, jails, ports, etc., have also been implicated historically (McVicar, 1908; U.G., 1914), but the contribution of these factors to the tuberculosis epidemic are typically more localized. Much of this discussion has been influenced by the writings of Packard (1989a), whose contributions to understanding the political economy of this disease must be acknowledged.

A NOTE ON STATISTICS

The reporting of statistics for indigenous populations has always been poor. Before the Union of South Africa was formed in 1910, statistics were reported for each colony. Death registration was only made compulsory in the Cape Colony in 1895 (McVicar, 1908). Tuberculosis was made a notifiable disease in 1904, first in the Cape Colony and later that same year in Natal (TBRC, 1932). Even after the colonies were united, the collection of statistical data for tuberculosis was poor and it was only from 1920 that tuberculosis was made notifiable for the entire Union (Collins, 1982). Despite this, the true impact of disease among indigenous populations remained largely unknown due, partly, to their exclusion from census areas and poor access to medical facilities (Packard, 1989a). Census data for the entire population of South Africa were not collected in any systematic way until 1996.

Figure 1 illustrates the trends in case notifications from 1921 to 1976 as reported by the Department of Health, and includes notifications from the Republic of South Africa and the South African Black states until their “independence” in 1976/7 (Küstner, 1979). A steady increase in case notifications are noted from 1920, when national notifications were first instituted, to the 1950s. This is followed by a steep increase in notifications in the 1950s and a steep decline in the 1960s. Trends in death notifications, from 1945 to 1977, are presented in Figure 2. A sharp decline in mortality is noted in the 1950s after the introduction of the anti-tuberculosis drug, Isoniazid. As was noted above these statistics may not be truly reflective of the extent of tuberculosis among indigenous South Africans. The issues associated with these trends, as well as the events/processes underlying and preceding them are discussed below.

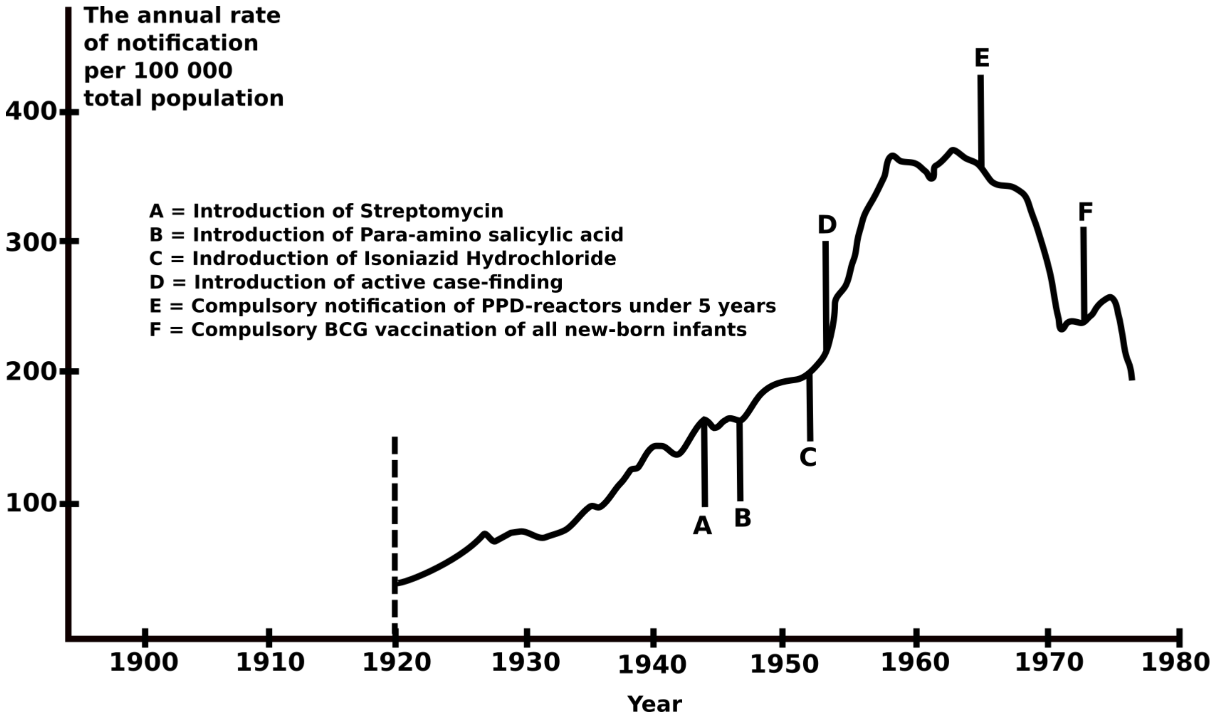


Figure 1: “Annual rate of notifications for all forms of tuberculosis per 100 000 total population, 1921-1976” (reproduced from Küstner, 1979).

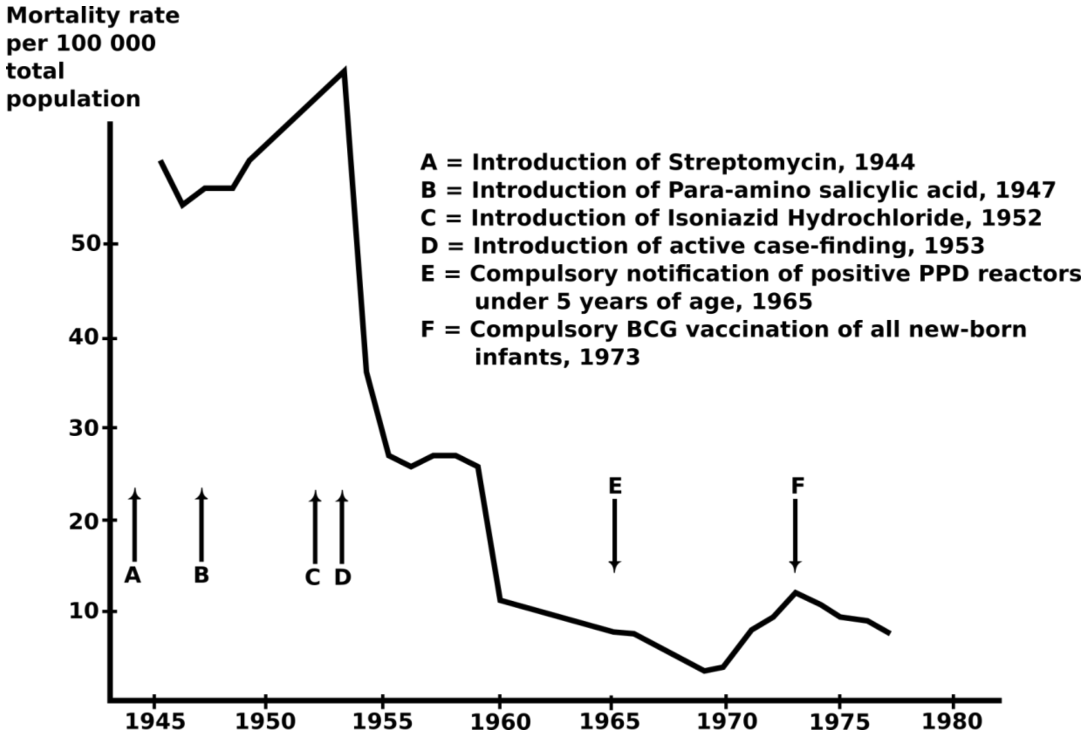


Figure 2: “Notified deaths from all forms of tuberculosis per annum per 100 000 total population, 1945-1977” (reproduced from Küstner, 1979).

IMMIGRANTS AND TUBERCULOSIS

Whether or not tuberculosis was present in pre-colonial South Africa, the importation of the disease by European immigrants undoubtedly contributed to its spread among the indigenous population. Tuberculosis was rife in 17th century Europe and it is likely that some of the Europeans who made their way to the Cape were infected with the disease. The role of imported slaves, convicts and indentured labourers from the East has not been explored in this regard, but given the antiquity of the disease in this region they may have also contributed to the spread of tuberculosis among the indigenous population. It was not until South Africa became a popular destination for consumptives, seeking a climatic cure for the disease, that large numbers of sick European immigrants ventured to the southern tip of Africa. In fact, during the late 19th century books and articles promoting the Cape as a health resort for consumptives began to circulate abroad (see Metcalf, 1991; Packard, 1989). The sea voyage from Europe was long and arduous. Conditions aboard shipping vessels (e.g. poor diet, confined living quarters, etc.) were often highly conducive to the spread of disease among crew and passengers; furthermore, they likely encouraged disease progression and the reactivation of latent infection. Many of the sick who survived the trip were too ill to journey elsewhere and would die shortly after reaching the port city (Ramsbottom, 1905). The desired destination for most, however, was the dry, arid interior, and many of these towns (e.g. Craddock, Beaufort West, Kimberley, Harrismith, Middelburg) became popular destinations for tuberculous immigrants (Metcalf, 1991).

The threat of imported tuberculosis to the local population was not immediately recognized. It was only after Koch's discovery in 1882 that the infectious nature of tuberculosis was fully appreciated, and even after this, adequate precautions to prevent the spread of infection were not frequently observed (Packard, 1989a; U.G., 1914). European immigrants who were sufficiently wealthy could avoid working and lived in private housing; however, this was rare. Most immigrants were not as fortunate, could not afford private housing and would eventually have to find employment (Ramsbottom, 1905). The better educated could, for example, serve as clerks or tutors but for many others manual labour was the only option (Ramsbottom, 1905). Those in poor physical condition were ill-suited for regular employment and, consequently, were forced to survive on meagre wages and live in overcrowded and unsanitary boarding houses. Isolation of the sick was uncommon and, as a result, these individuals were able to spread the disease to those sharing working or living quarters (Ramsbottom, 1905); thus, posing a risk to the local population. Coincident with the rise in popularity of South Africa as a health resort was the development of sanatoria. In the interior, numerous small sanatoria (in essence guest farms popular among the sick) were established. The state-run Nelspoort Sanatorium, established in 1924, was the only South African example of the archetypical sanatorium of the era. After World War II, a number of tuberculosis state hospitals, modelled on sanatoria, were established in the major cities (Dubovsky, 1991). During this period, in Europe and America, the sanatoria movement was already in decline, largely as a result of a decrease in tuberculosis prevalence.

At the beginning of the 20th century tuberculosis was made notifiable, which shed some light on the extent and distribution of disease prevalence and mortality. Excluding the major cities, the towns frequented by tuberculous immigrants had the highest tuberculosis prevalence and mortality rates (U.G., 1914). Among the white population tuberculosis rates were typically low, often lower than those reported for England and Wales (McVicar, 1908; U.G., 1914). Furthermore, the majority of deaths were attributed to imported cases (McVicar, 1908). This is not to suggest that sick immigrants did not pose a threat to the local population. Many tuberculous immigrants would find employment as tutors on farms in the interior and spread the disease to the farmers and their family. Among the indigenous population, circumstances were very different. Tuberculosis mortality rates were reportedly four to six times that of the European population (McVicar, 1908; U.G., 1914) and by the early 19th century, it was noted that the Khoesan and their descendants were severely affected and bore the heaviest burden of disease. While poverty was likely a major contributor to the heavy disease burden in these communities, tuberculous immigrants provided a source of infection. In the report of the Tuberculosis Commission (U.G., 1914) Mr A. Patterson, the town clerk of Craddock, expressed concern over the practice of selling the clothes and property of deceased tuberculous immigrants to indigenous communities. He also noted that in the town “servant girls” would take food up to the sick and eat whatever leftovers remained. The chairman of the Tuberculosis Commission (U.G., 1914) had, ~20 years earlier at the main hotel in Beaufort West, witnessed consumptives “siting all day on the *stoep*, expectorating into the adjacent open water-furrow which was the only source of water supply of many dwellings and of the extensive coloured location just below”. With the exception of the major urban centres, tuberculosis was reportedly rare among the Bantu-speaking population prior to the late 19th century and sick immigrants are not considered a major contributor to the spread of the disease among them.

The first report of the Tuberculosis Commission (U.G., 1912) addressed the issue of tuberculous immigrants and provided recommendations for their admission into South Africa. The main concerns raised in the report were the threat these individuals posed to the local population and the burden that their illness/death placed on the state. While the Commission ultimately viewed the complete exclusion of tuberculous immigrants from the country as the best way to deal with this problem, they rejected this solution viewing it as both unfeasible (given the limited resources of the state) and unethical (should repatriation of the sick equate to a death sentence / the climate actually provide a cure). Instead, the commission made a number of recommendations which included discouraging entry of tuberculous individuals as far as possible, restricting ports of entry, charging an administration fee (to defer costs), issuing permits (dependent upon the immigrants ability to prove they have the means to support themselves and their submission to monitoring for a period of 3 years), conducting medical examinations at the point of embarkation and the port of entry and, finally, making repatriation the burden of the shipping companies (in hope of forcing them to ensure passengers had sufficient funds for the return journey and enforce medical examinations at the point of embarkation), as well as requiring shipping vessels to ensure sick passengers are isolated and that cabins are properly disinfected afterwards. Some of these recommendations were dealt with in the 1913 Immigrants Regulation Act; however, this Act had limited impact on the spread of tuberculosis, as the Sanatoria movement and the popularity of the climatic cure had already begun to decline abroad (Metcalf, 1991).

In summary, the threat of imported tuberculosis from European immigrants was largely limited to the local white population, the Khoesan and their mixed-ancestry descendants. The impact of this source of infection was not largely felt by the Bantu-speaking population. The popularity of South Africa among tuberculous immigrants seeking a climatic cure for the disease undoubtedly contributed to the increasing prevalence of the disease among the local population. However, the relevance of imported tuberculosis preceding this period should not be overlooked. McVicar (1908) noted that the high tuberculosis prevalence among the Khoesan-descendant population of the Cape Colony preceded the period when South Africa was popular as a health resort.

TUBERCULOSIS, MINING AND THE MIGRANT LABOUR SYSTEM

"To say that the mining industry has played a defining role in the course of South Africa's history, at least during the last century and a half would be no great exaggeration. Nowhere else in the world has a mineral revolution proved so influential in weaving the political, economic and social fabric of a society." (Davenport, 2013)

The discovery of minerals was as important to the history of tuberculosis as it was to the political, economic and social history of South Africa. While it is clear that tuberculosis had become a problem in the major urban centres relatively early in its colonial history, the discovery of minerals probably represents the single most important factor for the rise of the tuberculosis epidemic among black South Africans at the turn of the 19th century. The mines have played such an instrumental role in the story of tuberculosis in South Africa due the poor conditions in the mines, their size, the large number of workers they employ, their high employee turnover and their role in disseminating the disease to the rural areas (Packard, 1989a). While reported mortality rates in the mines are typically lower than those in the major cities, these values are not a true reflection of mining related tuberculosis mortality. Many sick miners were repatriated to their homes and thus excluded from the mine statistics, whereas, black workers in the cities were more likely to remain in the city and be included in statistics (Packard, 1989a).

THE HISTORY OF MINING IN SOUTH AFRICA

Iron-age communities were exploiting South Africa's mineral resources long before the arrival of Europeans (Chirikure, 2015; Miller et al., 2000). However, sustained European commercial mining operations did not commence until almost two centuries after the arrival of the first European settlers (Davenport, 2013). Copper from the Namaqualand mountains was the first mineral to be commercially exploited by colonial settlers. Earlier ventures did not spur successful, long-term mining operations but by the mid-19th century the Cape Colony was in the midst of a mineral investment frenzy (Smalberger, 1975). After the copper bubble burst, many of the remaining mining companies ultimately failed owing to a poor understanding of geology, how copper was formed and a lack of infrastructure. However, a few copper mining operations were able to persevere and, in all, Namaqualand's copper resources were exploited for 150 years until reserves were recently exhausted (Davenport, 2013).

While copper was the first mineral to be commercially exploited, it was the discovery of Diamonds in 1867 that resulted in South Africa's first mineral revolution. The initial alluvial diggings were focused along the banks of the Vaal River in the region of Pniel and Barkley West (originally KlipDrift) (Payton, 1872). This initial burst of mining activity might have petered out too, had it not been for the discovery of nearby diamondiferous pipes further south of the river – of which Kimberley Mine is probably the most famous (Davenport, 2013). These 'dry diggings' attracted locals and foreigners alike, as Chapman (1872) describes, "There were faces of every conceivable cast and colour … in fact a smattering of people from every nation on the face of the earth." Initially, numerous independent stakes were claimed and dug haphazardly and, consequently, as surface deposits depleted and digging went deeper, chaos ensued (Worger, 1987). In the early years of the diamond rush the interests of independent diggers were fiercely upheld. However, in the face of ever-deepening operations, the independent, uncoordinated system that had arisen became less fruitful and more dangerous. Following legislative changes favouring capitalist interests, claims were consolidated and monopolies began to emerge. The diamond rush ushered in a mineral and industrial revolution; however, it was the discovery of gold on the Witwatersrand in 1886 that had the greatest effect on the development of South Africa's political, economic and social landscape.

The diamond rush encouraged further prospecting of the South African interior and reports of gold deposits began to emerge soon after (Williams, 1948). Similar to the diamond discoveries, gold diggings were initially focused on alluvial deposits and also followed a “diggers’ democracy” where independent diggings were favoured (Davenport, 2013). Prior to the discovery of the Witwatersrand gold, numerous diggings had already emerged. For example, Pilgrim’s Rest and Barberton had both been sites of a gold rush attracting a large and varied crowd of local and foreign diggers, as well as those engaged in ancillary enterprises (Joyce, 1986; Mathers, 1970; McDonald, 1985). Rich deposits in Barberton sparked an investment frenzy but neither the gold mining nor the investment boom were sustainable and, as a result, were short lived (Davenport, 2013). While some of these earlier diggings did eventually result in established mines, it was the discovery of ‘payable’ gold on the Witwatersrand that sparked a long and successful gold mining industry. For the most part, South African gold ore is of a relatively low grade and considerable capital is necessary to extract the ore (Davenport, 2013). Thus, from early on it became evident that the mining of small independent claims would not be profitable and, as a result, the consolidation of claims occurred much more rapidly on the goldfields of the Witwatersrand than occurred on the diamond fields of Kimberley (Cartwright, 1965). Furthermore, this capital was readily available from the mining magnates who had made their wealth in Kimberly diamonds (Wheatcroft, 1986).

Early in its development, the South African mining industry became reliant on cheap black labour. A sentiment of racial superiority among colonists was present long before the discovery of diamonds. The view that manual labour was menial, and, therefore, an undignified occupation for white men, had become pervasive among South Africa’s local inhabitants (Sparks, 1990). Thus, from the onset of the diamond diggings teams of black labourers supervised by white men was a fairly common occurrence (Chapman, 1872; Davenport, 2013). Black men were initially attracted to the diamond fields in the hopes of earning money to buy cattle and European goods (including clothes, liquor, tobacco and guns) (Davenport, 2013). In the earlier years of the diamond diggings black labourers were sufficiently scarce and sought after that they could pick and choose who to work for (based on who provided better treatment/conditions) and negotiate for better wages. However, as costs began to increase with ever-deepening operations, so did the need to control and secure a reliable source of cheap labour (Davenport, 2013). Because most of the machinery and skilled labour had to be imported (often at great expense) and the diamond price prior to the De Beers monopoly was dependant on demand, the only aspect of operations fully under the control of operators was the cost of labour (Davenport, 2013). The ability to exploit this labour came with the consolidation of the mines and the emergence of powerful mining magnates who had political sway in passing legislation (van der Horst, 1942). Specifically, pass laws were enacted to control the movement of labour and the creation of hut and poll taxes forced Africans into the wage labour market (Nkosi, 2011; van der Horst, 1942; Worger, 1987). The development of closed mining compounds also served to control the movement and permanency of the labour force, as well as curtail theft and illicit trade (Williams, 2011). Many of these developments emerged at the diamond mines which served as a model for the Witwatersrand goldfields and laid the foundations for the racial stratification of labour that would persist in South Africa for almost a century (Davenport, 2013; Wilson, 1972). The experience of white miners was significantly different. White miners were able to unionize and petition for the establishment of job colour bar, to protect their position in industry (Hutt, 1964). Consequently, they enjoyed higher wages and certain privileges that were not available to black workers. Furthermore, they were not required or compelled to live in all male compounds and could, therefore, settle nearby with their families. Thus, the experience of tuberculosis differed greatly between white and black men employed on the mines; although tuberculosis rates were still high among white mine workers.

For black mineworkers, nearly every aspect of their mining experience, from recruitment to the provision of accommodation and food and, lastly, the conditions within the mine itself, encouraged the development of tuberculosis. Furthermore, the migrant labour system that had emerged, in conjunction with declining conditions, fuelled the spread of disease to the rural areas.

LABOUR RECRUITMENT AND THE PHYSICAL CONDITION OF WORKERS ON ARRIVAL AT THE MINES

The rapid expansion of the mining industry and escalating costs of extracting deep, relatively low grade ore, led to an increasing demand for cheap unskilled labour (Feinstein, 2005). Africans were, however, reluctant to work on the mines due to the low wages offered and poor conditions on the mines (Packard, 1989a; Wilson, 1972). Thus, labour shortages were a perennial problem for mine owners (Harington et al., 2004; Wilson, 1972). In order to meet this demand, labour recruiters / recruitment agencies became a necessary part of the mining industry (Wilson, 1972). Initially, the recruitment process was not regulated or centralized and this in addition to intense competition among recruiters was blamed for the poor condition of recruits at arrival on the mines (Packard, 1989a). To begin with, many recruits were forced into labour due to declining conditions in the rural areas (Bundy, 1972). For example, drought and rinderpest epidemics had rendered many under- or malnourished and/or unable to afford paying their taxes. Once recruited, the conditions in which workers travelled to the mines were typically less than ideal (Packard, 1989a). In such instances where the recruitment agency paid for the travel, meals were often inadequate or not provided and recruits making their own way to the mines were typically unable or unwilling to spend their advances on food. In fact, mine managers often complained that scurvy, malaria, pneumonia and general exhaustion were common among new recruits (Packard, 1989a). In addition, the mixing of sick and healthy individuals during transit also increased the chances of spreading communicable diseases, including tuberculosis. The mines and, later, recruitment agencies performed health examinations of new recruits; however, these were typically cursory and did not prevent the employment of workers who were in a poor state of health (Packard, 1989a). Furthermore, competition among recruiters and differing standards at different mines exacerbated this problem. For example, Dr Miller, medical officer for East Rand Property Mines, in 1904 described the poor condition of 106 mining recruits sent to the mine and passed as fit by the Witwatersrand Native Labour Association (WNLA). On arrival 10 of these individuals were suffering from pulmonary tuberculosis and were returned to WNLA. Subsequently, these individuals were recertified as fit and sent to another mine (Packard, 1989a). As a consequence of these factors, new recruits arriving at the mines were often in such a poor state of health that their resistance to tuberculosis was low or they had already been infected during transit.

CONDITIONS ON THE MINES

Conditions on the mines and in the mining compounds were ideal for the spread of tuberculosis and acted to both facilitate transmission and increase susceptibility. Within the mining compounds, workers resided in all male hostels. Severe overcrowding was a common feature of these hostels in the first few decades of the industry (U.G., 1914). The

Tuberculosis Commission observed men sleeping on hostel floors and outside in the courtyards due to overcrowding and inadequate provisioning of beds (U.G., 1914). This problem was exacerbated when government legislation regulating working hours came into effect. To offset this reduction in working hours, mine owners would often hire more workers without a concurrent increase in housing or infrastructure (U.G., 1914). Under such close quarters, regular contact with sick individuals would have greatly increased the risk of infection for others. Nutrition within the compounds was also problematic. The diets of mineworkers were often insufficient in quantity or lacking in nutritional value. In the closed compounds of the diamond mines, workers could purchase meals from the merchant on site (Gorgas, 1914); however, many workers chose to limit the percentage of wages spent on food opting instead to consume less (Packard, 1989a). On the Goldfields many compounds provided meals as part of the wage package, but again these were typically inadequate. Provisioned meals were usually limited to one or two per day and were largely based on maize (Gorgas, 1914). Vegetables and meat constituted a minor portion of food rations. Furthermore, the quantity of food provided was not always sufficient to meet the energy requirements for such physically demanding work. The result is that many mine workers were under- or malnourished, a fact attested to by the relatively high rates of scurvy in the mines (U.G., 1914). Another factor that may have contributed to the high incidence rates of tuberculosis in the early years of the industry was the lack of basic sanitation both within the compounds and the mines (Gorgas, 1914). This lack of sanitation may have resulted in a high rate of associated diseases (e.g. dysentery, typhoid and intestinal parasites) (L'Abbé et al., 2003; Packard, 1989a; Turrell, 1984) and together with an inadequate diet would likely have had a negative effect on the immune status of workers and increased the risk of contracting tuberculosis.

There are a number of factors related to the working environment within the mine that may have also promoted the spread of tuberculosis. The humid, dusty, poorly ventilated and confined environment of the mine stope provided ideal conditions for the spread of tuberculosis. In addition, the work is physically taxing and, before legislation was passed to regulate shift lengths, working hours were long (Packard, 1989a; U.G., 1914). Miners often worked overtime during periods of heavy production, labour shortages or, simply, to meet quotas and, prior to 1910, working double shifts was common (U.G., 1914). Drilling and blasting in mine stopes produces a large amount of fine particulate matter. The regular inhalation of this material can result in the formation of pneumoconiosis of which silicosis is one of the most prevalent forms. A number of studies have reported an increased risk of developing tuberculosis in miners suffering from silicosis (Corbett et al., 1999; Cowie, 1994; Kleinschmidt and Churchyard, 1997). Even in the absence of silicosis, exposure to silica dust is associated with an increased risk of developing tuberculosis (Hnizdo and Murray, 1998; TeWaterNaude et al., 2006). After 1910, to reduce the amount of dust produced, axial water-feed drills were introduced; however, this had the unintended consequence of increasing humidity within the underground stopes (Irvine, 1932; Packard, 1989a). Greater humidity, in turn, resulted in an increased the risk of heat exhaustion (Martinson, 1976), as well as, exacerbating lung complaints (possibly through increasing pathogen survival and droplet stability) (Naumova, 2006). Rapid changes in temperature presented another risk to mineworkers. Prior to 1911, few mines provided shelters and changing houses. Thus, after a shift, wet with perspiration and water from the drills, workers were often exposed to cold temperatures/drafts, sometimes for long periods while they waited to be transported to the surface of the mine (Katz, 1980). Since mine entrances are not always near the compounds, this would have also been a problem for workers making their way to and from the compounds and those waiting to be lowered into the mine shaft. This problem would have been further exacerbated in inclement weather.

Thus, there were a number of factors existing within the mines, mine compounds and even before workers reached the mines, that may have acted in concert to produce high rates of tuberculosis on the mines. High tuberculosis morbidity and mortality are not always reflected in mine statistics owing largely to the repatriation of sick workers from the mines back to the rural areas and, therefore, the exclusion of these individuals from mine statistics (Packard, 1989a).

SPREAD OF TUBERCULOSIS TO THE RURAL AREAS

The reasons for the rapid spread of tuberculosis to the rural areas and variations in disease distribution are diverse but have largely been attributed to the return of migrant labourers from the mines and declining conditions in the rural areas (Packard, 1989a).

The development of a migrant labour system is not unique to South Africa. What separates the South African experience is the length of time over which this system has remained relatively stable. Initial resistance to permanent urban settlement by rurally sourced labour is common in any newly industrialized country, but gradually people begin to settle near the workplace (Wilson, 1972). In South Africa, this process was interrupted and a system of oscillating migration became established through the agency of industry and government which actively sought to prevent the permanent settlement of Africans in urban centres (Wilson, 1972). Wages could remain low (below the amount necessary for the reproduction of labour) only by ensuring that workers continued to have a rural economic base, which would bear the primary responsibility for the reproduction of labour, while also serving as a repository for workers who were no longer productive due to advanced age, disability or illness (Packard, 1989b). It should be mentioned that the mining industry has not been the only driver of labour migration. Oscillating migration to the major urban centres, where tuberculosis had become established relatively early, was also common before and after minerals were discovered and surely also contributed to the spread of the disease to the rural areas (Packard, 1989a). However, the role of men returning from the mines in the spreading the disease is specifically emphasized in the medical literature of the time (McVicar, 1908; Millar, 1908; U.G., 1914). Furthermore, the Tuberculosis Commission (U.G., 1914) reported a correlation between the prevalence of tuberculosis in a rural community and the extent and length of time in which that community had been participating in labour migration to the mines. In 1899, 1911 and 1936 the mines reportedly employed 90 000, 174 000 and 318 000 men, respectively (Harington et al., 2004; Wilson, 1972). Because mining contracts were short, varying between three and eighteen months, but more typically six to nine months, the annual replacement of the workforce was nearly 100% (Packard, 1989a). Thus, it is likely the sheer scale of migration induced by the mining industry ensured its overwhelming contribution to the spread of tuberculosis (Packard, 1989a). The practice of repatriating sick miners to their rural homes exacerbated the problem. Numerous cases have been reported where a repatriated miner with tuberculosis would infect his entire family, the members of which would, in a relatively short space of time, succumb to the disease as well (Millar, 1908; TBRC, 1932; U.G., 1914). The Chamber of Mines justified both the system of oscillating migration and the repatriation of sick miners by arguing that workers would benefit from the rest and recuperation at their rural homes. Furthermore, they argued that repatriated workers wanted to return home. Given the state of medical knowledge in the early 20th century and high hospital mortality rates, an aversion to hospitals is understandable (Packard, 1989a). Consequently, the latter argument is probably true; however, little thought was given to the impact repatriation would have on the spread of tuberculosis to the rural areas. This practice did have a positive effect on mine tuberculosis mortality figures, which were frequently lower than expected since they excluded repatriated workers (Dormer and Wiles, 1946). The fate of repatriated workers with tuberculosis is difficult to assess, but Dormer and Wiles (1946) estimated that as many as 90% died of their illness, while Allan (1924) provides a more conservative estimate of 60%. While the oscillating migration and repatriation of sick mine workers contributed to the spread of tuberculosis to the rural areas, the factors that contributed to their participation in this system must still be addressed.

The practice of repatriation was based on the premise that the mineworker's rural home was a place of plenty and leisure where he could recover from illness and the physical stress of mine work. As Packard (1989b) termed it, this "myth of the 'healthy reserve'" was becoming increasingly inaccurate as the 20th century progressed. Due to the high demand for cheap labour, labour shortages were threatening the viability of the mining industry (Harington et al., 2004; Wilson, 1972). In response, a number of taxes were imposed and laws passed during the late 19th and early 20th centuries, culminating in the 1913 Land Act. These served to undermine the rural African economy and African land ownership in an attempt to force Africans into the wage economy (van der Horst, 1942). This in conjunction with a series of rinderpest epidemics and droughts, as well as the effects of the South African War, resulted in an increasingly impoverished rural 'reserve' (Packard, 1989a). These factors and others acted to reduce the ability of rural Africans to resist tuberculosis (by undermining nutrition) and aid in its spread by resulting in overcrowding, as well as encouraging participation in labour migrancy.

Rinderpest epidemics first began to affect South African cattle in the late 1890s and had a devastating effect on the African economy and diet. Cattle served not only as a vital source of milk and meat but were also important for clothing, fuel, fertilizer, tractive power (for ploughing fields) and capital (Phoofolo, 1993). Thus, these epidemics impacted more than just African health; the economic, social and political spheres of life were affected as well. The cattle population recovered rapidly after World War I and this, together with increased overcrowding in the reserves, resulted in overstocking and overgrazing (Packard, 1989a; U.G., 1932). As a result, during the 1930s, there was rapid decline in African herds and an increase in stock mortality (Fox, 1936). The loss of nutrients from livestock was, however, only part of the problem. The shift from traditional crops (such as the more drought resistant sorghum) to more economically beneficial (but less nutritious) maize ultimately had a negative effect on rural African economic viability and nutritional status. Maize was a more economically attractive crop because it could be sold in the European market, had lower labour requirements and produced a higher yield (McCann, 2001). Initially, maize formed a part of mixed rural agriculture, but with the economic opportunities provided by the growth of the mining industry the trend was increasingly towards a maize monoculture that acted to limit the nutritional variety of rural African diets (McCann, 2001). As African dependence on maize increased, the introduction of taxes on maize sales and purchases contributed to the problem (Packard, 1989a). Increased overcrowding, due to population growth and political policies, placed additional pressure on land resources in the reserves. Consequently, rural farmers chose to forgo longer fallow periods and crop rotation to increase agricultural productivity (Fox and Black, 1939; Packard, 1989a). These problems were further exacerbated by the droughts that occurred in the late 1920s and early 1930s (Packard, 1989a; Rouault and Richard, 2003). By the 1920s, the rate of land degradation occurring in the reserves as a result of overcropping and overstocking had already become a cause for concern (Wolpe, 1972). From the 1930s, numerous reports drew attention to these factors highlighting their roles in the increasing prevalence of malnutrition through declining agricultural productivity, lack of dietary variation due to an over-reliance on maize and nutrients lost from declining herds (Fox, 1936; Fox and Black, 1939; U.G., 1946, 1932). Although, Simkins (1981) posits that rural Africans in the reserves were unable to meet their dietary requirements through agricultural production from as early as 1918 (the first year a national agricultural census was taken). Kark and Le Riche (1944) show that at best close to half of all the rural children examined in their study were suffering from obvious signs of ill-health or malnutrition; however, for most regions the percentage was greater. Furthermore, nutritional deficiency diseases, such as ariboflavinosis, pellagra and scurvy, were relatively common and a cause for concern particularly among rural African children (Fox, 1936; Kark and Le Riche, 1944; U.G., 1946). Thus, it is likely the high rates of malnutrition in the reserves would have negatively affected the immuno-competency of individuals and aided the spread of tuberculosis. Lastly, while general overcrowding in the reserves has been mentioned, the introduction of the hut tax served to worsen overcrowding within individual huts (Millar, 1908; Werner, 1905). Millar (1908), describing his experiences in the former Pondoland, notes that overcrowding was the rule and instances of about 20 people sleeping in a single hut were common. Under these overcrowded conditions, the risk of tuberculosis transmission would have been high.

It should be noted that the rate at which tuberculosis spread was not uniform across all rural areas, within the same area or through time (Packard, 1989a). Considerable variation existed in a region's experience with, for example, drought, livestock and human diseases, political policies, labour migration, etc. While there are no adequate statistics for the rural areas, what information is available seems to indicate that the former Ciskei and Transkei (Eastern Cape) were the worst affected regions initially. Reserves within the former Transvaal, Orange Free State and Natal fared better, at least until World War II, as did the regions outside of South Africa's borders from which mine labour was sourced (Allan, 1924; Packard, 1989a). Given that levels of wealth and participation in the wage economy differed across individuals and communities, differences in the spread of tuberculosis occurred even within the same region (Packard, 1989a). Temporal differences within a region occurred as well. For example, in Natal many medical practitioners observed a decline in tuberculosis prevalence in the 1920s after an initial increase at the turn of the century. A series of rinderpest epidemics and crops losses due to locusts, were blamed for this initial increase and the decline attributed to the amelioration of conditions after 1920 (Allan, 1924).

The declining conditions in the rural 'reserves' did not go unnoticed. The poor state of new recruits led the Chamber of Mines to establish a commission to investigate conditions in the reserves (Fox and Black, 1939), and the South African government led similar inquiries (U.G., 1946, 1932). The picture that emerged was one of general poverty and malnutrition. Yet nothing was done to lessen the plight of the increasing impoverished rural Africans, and the mines continued to support their programme of repatriation. While tuberculosis prevalence continued to rise, the role of the mining industry in disseminating the disease to the rural areas diminished with time. Dr Allan speculated that tuberculosis had become endemic in the former Transkei and Ciskei by the 1920s (TBRC, 1932), and by the onset of World War II, this was likely the case for the other rural areas as well (Packard, 1989a) to the extent that returning mineworkers no longer posed a major threat.

URBANIZATION AND APARTHEID

World War I ushered in a period of industrial growth (Feinstein, 2005), and this, in combination with laws designed to force Africans into the labour market, increasingly drew Africans to the cities (Packard, 1989a). Tuberculosis mortality continued to rise during the war; however, the post-war period was marked by a decrease in mortality despite an economic recession. The Second World War, like its predecessor, spark a period of economic growth and the expansion of the manufacturing industry again facilitated the migration of rural Africans to urban centres (Feinstein, 2005; Packard, 1989a). The combination of insufficient affordable housing and low wages resulted in a housing crisis that persists to this day. Africans in the cities had little choice but to live in overcrowded slums or erect their own informal shelters on the outskirts of town. Poor nutrition and lack of sanitation infrastructure further contributed to poor health and, in combination with overcrowding, created ideal conditions for the spread of tuberculosis (Packard, 1989a). The government’s response to this crisis effectively redistributed the problem instead of dealing with the underlying causes of poor health among Africans (Packard, 1989a).

The victory of the National Party in the 1948 elections ushered in a period defined by policies of racial segregation that formed the foundation of Apartheid ideology. The marked rise in tuberculosis notifications during the 1950s had been partly attributed to increased efforts in case-finding (Collins, 1982). Packard (1989a), while acknowledging this, stresses that the rise in notifications was underpinned by an actual increase in tuberculosis incidence, fuelled by a worsening housing crisis, poverty and inadequate tuberculosis control programmes. While the decline in tuberculosis notifications that began in the 1960 has often been interpreted as a decline in tuberculosis incidence, Packard (1989a) has suggested an alternative explanation for what he terms the “Great Disappearing Act”. The forced removal of millions of Africans out of urban centres to the “reserves” under Apartheid effectively relocated the disease. Thus, the decline in tuberculosis notifications did not represent a decrease in disease incidence but rather a removal of the most marginalized segment of the population from statistical records. The decline in death notifications has, however, been attributed to a decline in mortality following the introduction of chemotherapeutic agents (Collins, 1982; Packard, 1989a).

HIV AND MULTI-DRUG RESISTANT TUBERCULOSIS

More recently, the emergence of the HIV epidemic has spurred an increase in tuberculosis. The first case of HIV was reported in South Africa in 1982 (Karim et al., 2009). While the disease was initially identified in homosexual men, by the 1990s it had established itself more broadly within the population (Karim et al., 2009). Under Apartheid little was done to limit the spread of the disease, which was branded a “black” disease. Post-Apartheid control of HIV was initially hampered by a lack of adequate health services and the lack of a clear HIV control strategy that was fuelled by the denial of the cause of AIDS by government leadership (Karim et al., 2009). Consequently, HIV mortality continued to rise throughout the 90s and rose to a peak in 2006 (Bradshaw et al., 2016). The steady decline in mortality that has been observed since, has been attributed to the implementation of more effective antiretroviral roll-out strategies (Bradshaw et al., 2016); however, HIV-related tuberculosis mortality remains a serious threat to public health.

While most publications report separate statistics for HIV-negative and HIV-positive tuberculosis cases, the reality is that in many cases an individual’s HIV status is not known or not reported. In a study on the South African population, Bradshaw et al. (2016) report that an estimated 93% of HIV-related deaths are misattributed to other causes, of which tuberculosis accounts for 23%. Karim et al., 2009 estimate that half of the tuberculosis incidence can be attributed to HIV co-morbidity. Clearly, these two epidemics are inextricably linked.

The emergence and increasing prevalence of drug-resistant, multidrug-resistant and extremely drug-resistant forms of tuberculosis are a further burden to an already strained public health system. Treatments are expensive and require a disproportionate allocation of the tuberculosis budget (Pooran et al., 2013). Drug-resistance also threatens to undermine the small gains that have been made in tuberculosis control during the past decade. In South Africa, the primary source of drug-resistance appears to be transmission, rather than treatment failure or default (Gandhi et al., 2006; Shah et al., 2017). As with tuberculosis in general, HIV co-infection appears to play an important role in the acquisition of drug-resistant tuberculosis and the associated mortality. Many studies have reported a high rate of HIV co-infection among drug-resistant cases of tuberculosis (e.g. Gandhi et al., 2006; Shah et al., 2017) and while some studies have reported high mortality among HIV co-infected individuals other have revealed poor treatment outcomes regardless of HIV status (Pietersen et al., 2014).

In summary, four major events/processes have been identified that have significantly contributed to the tuberculosis epidemic in South Africa. The importation of tuberculosis by European immigrants was an important factor early in the colonial period; however, the effects of this source on the indigenous population, while not negligible, were relatively limited. In contrast, the development of the mining industry and the system of oscillating migration played a key role in the emergence of the tuberculosis epidemic, as well as its spread to and eventual endemicity in the rural areas. Urbanization and the creation of overcrowded slums and informal settlements, in conjunction with poor nutrition and sanitation, contributed to the increasing incidence of tuberculosis throughout the 20th century. While statistics showed a decline in tuberculosis notification during Apartheid, this decline was more apparent than real, and the disease likely continued to spread unabated in the rural reserves. Finally, the rise of the HIV epidemic and a growing threat of drug-resistance continues to challenge effective tuberculosis control.

CHAPTER 3: MODERN AND ANCIENT MOLECULAR CONTRIBUTIONS TO UNDERSTANDING THE EMERGENCE AND SPREAD OF TUBERCULOSIS

The previous chapter has detailed the contribution of the literature and the paleopathological record to our understanding of the emergence, spread and evolution of tuberculosis. However, these sources can only provide information on the disease - its aetiology, epidemiology and the human experience of it through time. Molecular tools, on the other hand, can provide information on the pathogen responsible. This can be achieved either by studying phylogenetic relationships among extant organisms or by studying past organisms through ancient DNA and biochemical analyses.

The causative agent of tuberculosis in humans is the rod-shaped, aerobic, slow-growing, acid-fast bacillus *Mycobacterium tuberculosis* (*M. tuberculosis*) - identified and isolated by Koch in 1882 (Koch, 1882). The *M. tuberculosis* genome is a circular chromosome approximately 4.4 million base pairs in length with approximately 4000 protein coding genes (Cole et al., 1998). *M. tuberculosis* belongs to the genus Mycobacteria (phylum: Actinobacteria, class: Actinobacteria, subclass: Actinobacteridae, order: Actinomycetales, suborder: Corynebacterineae, family: Mycobacteriaceae), which includes over 170 species (Forbes, 2017). The mycobacteria are traditionally divided into fast- or slow-growing species based on their growth rate in culture; however, this divide is also supported by phylogenetic analyses (Tortoli et al., 2017; Tsukamura, 1967; Wee et al., 2017). Most mycobacterial species are not typically pathogenic and are free-living, environmental bacilli (Falkinham, 2015). While some of the slow-growing mycobacteria are obligate human and animal pathogens, many of the other mycobacteria may cause opportunistic infections in humans (Falkinham, 2015). Of the obligate human pathogens, *M. tuberculosis* and *M. leprae* (the causative agent of leprosy) are the most well-known and historically important.

THE *MYCOBACTERIUM TUBERCULOSIS* COMPLEX (MTBC)

The MTBC is a group of closely related organisms that cause tuberculosis in humans and animals. It includes the human-adapted *M. tuberculosis* and *M. africanum*, as well as *M. bovis*, *M. caprae*, *M. microti*, *M. pinnipedii*, *M. mungi*, *M. orygis*, *M. suricattae,* the chimpanzee bacillus and dassie bacillus, which are adapted to or have been isolated from a broad range of mammalian hosts (see Brites et al., 2018). Although once considered a member of the MTBC, *M. canettii* is generally no longer included. It is thought to be an environmental bacterium that only occasionally causes disease in humans (Koeck et al., 2011), is genetically more distant to the other MTBC members and has been described as more closely resembling the MTBC-progenitor (Gutierrez et al., 2005). Phylogenetic studies reveal 99.9% sequence identity among members of the MTBC (Smith et al., 2009), and researchers have only recently begun to resolve the phylogenetic relationships within the MTBC. The earliest of these studies began in the late 1990s and differences in molecular targets, as well as technological and methodological advancements over time, have resulted in a number of different classification schemes. The most recent classification, based on whole-genome comparisons, has separated the human-adapted *M. tuberculosis* and *M. africanum* into seven lineages that broadly correspond to the their geographic origins (Firdessa et al., 2013; Gagneux et al., 2006; Gagneux and Small, 2007). The *M. africanum* lineages 5 and 6 form a monophyletic clade with the animal-adapted strains and this clade is distinct from the *M. tuberculosis* lineages 1-4 and 7 (Brites et al., 2018). Gagneux and Small (2007) tabulate the older and current phylogenetic classification schemes and this is reproduced in Table 1. The characterization of lineage 7 is relatively recent and is not included in the table.

Table 1: Comparison of terminology and molecular markers for six main lineages of M tuberculosis and Mycobacterium africanum (reproduced from Gagneux and Small, 2007**)**

	Lineage 1	Lineage 2	Lineage 3	Lineage 4	Lineage 5	Lineage 6
SNP[a] (Sreevatsan et al., 1997)	Principal genetic group 1	Principal genetic group 1	Principal genetic group 1	Principal genetic groups 2 and 3	Principal genetic group 1	Principal genetic group 1
SNP (Baker et al., 2004)	Lineage IV	Lineage I	Lineage III	Lineage III	Not done	Not done
LSP[b] (Gagneux et al., 2006)	Indo-Oceanic lineage	East Asian lineage	East African-Indian lineage	Euro-American lineage	West-African lineage I	West-African lineage II
SNP (Gutacker et al., 2006)	Cluster I	Cluster II	Cluster II.A	Clusters III-VII	Not done	Not done
SNP (Filliol et al., 2006)	Cluster group 1	Cluster group 2	Cluster group 3a	Cluster groups 3b-6b	Not done	Not done
Spoligotyping (Brudey et al., 2006)	EAI	Beijing	CAS	Haarlem, LAM, T, X	AFRI2	AFRI1
LSP marker	RD239	RD105	RD750	*Psk*15/1 Δ7bp	RD711	RD702
SNP marker	*OxyR* C37T	Rv3815c G81A	*RpoB* T2646G	*KatG* T1388G, *RpoB* C3243T	Not known	Not known
Geographical association	East Africa, southeast Asia, south India	East Asia, Russia, South Africa	East Africa, north India, Pakistan	Americas, Europe, north Africa, middle east	Ghana, Benin, Nigeria, Cameroon	Senegal, Guinea-Bissau, The Gambia
Comments	Similar to ancestor based on presence of TbD1				Traditionally known as *M. aficanum* subtype 1 (Clade 1)	Traditionally known as *M. africanum* subtype 1 (Clade 2)

[a]SNP = single nucleotide polymorphism; [b]LSP= large sequence polymorphism

PHYLOGENETIC STUDIES AND THE EMERGENCE OF TUBERCULOSIS

It had long been held that human tuberculosis emerged as a consequence of zoonotic transfer from a bovine host. This zoonosis was linked to the development of domestication and agriculture and the large scale human settlement that resulted (Bates and Stead, 1993; Diamond, 1997; Kapur et al., 1994). This idea was based on the fact that *M. bovis* had a much broader species range than *M. tuberculosis* and that the earliest convincing skeletal evidence appeared around the time of domestication. It was also believed that the disease could not be maintained in the much smaller pre-agriculture and pre-domestication populations. However, genetic studies have shown *M. bovis* is the more derived ecotype (e.g. Brosch et al., 2002; Gordon et al., 1999), dispelling the notion that *M. tuberculosis* is derived from a zoonotic transfer of *M. bovis*. The evolutionary history of the MTBC is, however, not yet resolved. As was discussed above, the animal ecotypes are more closely related to the human-adapted *M. africanum* lineages 5 and 6. Given that *M. tuberculosis* and *M. africanum* are human-adapted, one possible scenario is that the progenitor to the MTBC was a human pathogen and host “jumps” occurred after the divergence of lineages 5 from lineage 6 and the animal clades (Brites et al., 2018). Alternatively, the progenitor was capable of infecting a broad range of hosts with subsequent host specializations accounting for the current pattern of host-adaptations. Regarding the geographical origin, as was discussed in Chapter 2, genetic studies have suggested an African origin for the MTBC; however, the timing of its emergence has not yet been resolved (see Brites and Gagneux, 2015).

ANCIENT DNA

Ancient DNA (aDNA) is broadly defined as DNA recovered from preserved biological remains (Cooper and Wayne, 1998). The remains investigated in aDNA studies span a broad temporal range and a variety of source materials. At the more recent end of the temporal spectrum are seeds, hair, feathers and faecal material recovered from extant, free-ranging organisms (sometimes referred to as non-invasive sampling), as well as degraded biological remains recovered from forensic cases. These investigations are at times considered a part of the aDNA field (Cooper and Wayne, 1998; Wayne et al., 1999) because they can deal with similarly degraded DNA. However, more typically aDNA studies refer to DNA recovered from museum and archaeological material in the form of preserved soft tissue (naturally or artificially), bone, teeth, coprolites, hair, feathers, eggshells, seeds, pollen, other plant remains, soil sediments and ice cores (see Cappellini et al., 2018). There have even been claims of palaeontological finds greater than a million years old; however, these have largely been discredited (see Austin et al., 1997; Hebsgaard et al., 2005). In 1984, the first successful case of aDNA retrieval was reported from a museum specimen of the extinct equid, the quagga (Higuchi et al., 1984). Since then, aDNA investigations into the past have spanned a broad range of organisms and topics that include: phylogenetic relationships of extinct organisms, the origin and expansion of plant and animal domesticates, population history and phylogeography, kinship and familial relationships, migration and admixture, paleoecology and paleoenvironment, ancient microbial, floral and faunal biodiversity, behaviour and diet, the diagnosis, origin and spread of disease, tempo and mode of mutations in populations, the origins of life (see Briggs and Summons, 2014; Cappellini et al., 2018; Kaestle and Horsburgh, 2002; Pääbo et al., 2004; Wayne et al., 1999).

Early aDNA studies were dependant on bacterial cloning to amplify the ancient endogenous host DNA (Higuchi et al., 1984; Pääbo, 1985); however, it was only after the development of the Polymerase Chain Reaction (PCR) that aDNA research began to increase in popularity. Initially, aDNA studies targeted multi-copy molecular targets. The premise behind this was that these loci would be more abundant in ancient remains and would, therefore, be more easily detected. The intracellular organelles, mitochondria and chloroplasts, (and thus, their respective genomes) are present in 100s to 1000s of copies per cell. Thus, these became popular targets for aDNA studies. In contrast, nuclear DNA is present in much fewer copies per cell (eukaryotic cells are typically diploid with exceptions, including polyploid plants). Prior to the development of next-generation sequencing technologies, PCR enabled the targeting of nuclear loci, which although used less frequently, had met with some success (e.g. Greenwood et al., 1999; Huynen et al., 2003; Stone et al., 1996).

Ancient DNA studies have entered a new era with the advent of high-throughput, mass-parallel, next-generation sequencing (NGS). These second generation sequencing technologies have enabled the amplification and sequencing of multiple DNA fragments simultaneously, potentially generating millions of short sequence reads from a single sample (see Marciniak et al., 2015). NGS has allowed access to much more genomic information than was possible with conventional PCR-Sanger sequencing based studies. In the NGS era, the number of mitochondrial and complete genomes produced by aDNA studies have increased dramatically (Hofreiter et al., 2015; Paijmans et al., 2012). NGS technologies are particularly well-suited to the analysis of short DNA fragments which are typical of degraded DNA. Furthermore, they have provided novel methods for evaluating the authenticity of ancient sequences and identifying DNA contamination (see Key et al., 2017). Many of the initial challenges confronting aDNA studies using NGS technologies are being overcome. Advances in bioinformatics have helped overcome some of the challenges associated with the analysis of millions of DNA sequences that are potentially generated per run (see Hofreiter et al., 2015; Key et al., 2017). Low endogenous DNA yields from the target organism are being combated through the development of target capture and enrichment approaches in an attempt to increase the yield of informative sequences (see Briggs et al., 2009; Knapp and Hofreiter, 2010; Marciniak et al., 2015). Costs have decreased significantly due to technological improvements, and new molecular methods such as barcoding/tagging of samples (which allow multiple samples to be pooled and analysed simultaneously in a single run) (Marciniak et al., 2015; Meyer et al., 2007). Yet despite the growing popularity of NGS in aDNA studies, conventional PCR-based approaches remain useful particularly as a screening tool (Harkins et al., 2015).

There are a number of challenges associated with aDNA studies and some of these are briefly discussed below.

ANCIENT DNA DAMAGE AND PRESERVATION

In a living organism DNA damage is repaired by various mechanisms (Lindahl, 1993); however, after death DNA damage begins to accumulate with the result that only a small number of ancient specimens will contain amplifiable endogenous DNA (Binladen et al., 2006; Höss et al., 1996; Pääbo, 1989). Furthermore, those specimens will typically not contain DNA fragments larger than 150bp; however, fragments up to 500bp can be obtained in well preserved specimens (Pääbo, 1989). After death the [cellular] membranes break down and DNA degradation is initiated by endogenous nucleases. The DNA is further degraded by the action of microrganisms within the digestive tract, as well as those present in the burial environment (Eglinton and Logan, 1991). Damage also results from the spontaneous oxidation and hydrolysis of DNA (Höss et al., 1996; Lindahl, 1993). These processes result in a variety of products that can be divided into two classes, those that effectively act to prevent PCR amplification (i.e. strand nicks, breaks, baseless sites, certain modified bases and crosslinks) and those that result in erroneous bases in the sequenced product (termed miscoding lesions) (Lindahl, 1993; Pääbo, 1989). Mosaic/chimeric sequences, between different endogenous loci or between authentic endogenous sequences and laboratory or environmental contaminants, are another phenomenon that may arise during amplification as a result of template switching (‘jumping’ PCR) (Pääbo et al., 1990).

Oxidative and hydrolytic DNA damage occur at a predictable rate and, after the initial DNA degradation, which occurs soon after death via the action of endonucleases, microbes and fungi, chemical degradation begins to accumulate. It would, thus, seem intuitive that DNA degradation should be associated with the age of a specimen. However, numerous studies investigating the relationship between the archaeological age of a specimen and the extent of DNA damage (or recoverable fragment size as a proxy) have failed to show such a relationship (Pääbo, 1989). Instead, the rate of DNA degredation appears to be dependent on environmental factors, of which temperature is the most significant (Smith et al., 2003). Stable, cold environments appear to yield amplifiable DNA more regularly than do climatically variable or warmer environments (Höss et al., 1996; Smith et al., 2003). Burial conditions encompass a range of variables, and ideal conditions for DNA preservation include a neutral to slightly acidic pH, low chemically available water, a cold/cool stable temperature and limited microbial-mediated decay. UV damage may be less important in buried specimens but may have some bearing depending on the post-mortem and history of the remains. The conditions to which a specimen is exposed after recovery may also be an important factor for DNA preservation. For example, the removal of a specimen from an ideal burial condition to a storage facility, where ideal conditions are no longer met, will negatively impact DNA preservation. Thus, DNA preservation can be predicted to be highly variable among different geographical regions, and even within the same site. Previously most researchers viewed 100 000 yrs as the limit for DNA preservation (Höss et al., 1996; Lindahl, 1997, 1993; Pääbo, 1989). With the advent of next generation sequencing, genomes exceeding 100 kyrs BP have been successfully sequenced. These include mitochondrial and nuclear DNA from 430 kyrs hominins located in Sima de los Huesos, Spain (M. Meyer et al., 2016; Meyer et al., 2014) and a 560-780 kyrs BP middle Pleistocene horse from Canada (Orlando et al., 2013). The current view is that obtaining DNA sequences greater than 1 million years old is unlikely but given recent technological advances not impossible (Millar and Lambert, 2013).

CONTAMINATION

While DNA contamination can occur in all molecular studies, it is a particular concern for ancient DNA studies and questions of authenticity have plagued the field. Contamination may originate from various sources, as well as at various points in the post-mortem and post-recovery history of a specimen (Handt et al., 1994). Contaminant sequences can be divided into two classes, those that arise within the laboratory and those already present on the sample prior to laboratory analysis (Willerslev and Cooper, 2005). Sources of laboratory contamination include laboratory personnel (who exude their own DNA and transfer DNA from many sources), reagents (which may contain contaminants incorporated during manufacture) and amplified DNA from previous experiments (so called “carry-over” contamination) (Willerslev and Cooper, 2005; Yang and Watt, 2005). The use of dedicated facilities for ancient and modern DNA; protective clothing; unidirectional work-flow; and regular UV-crosslinking and/or bleach decontamination of surfaces, reagents and equipment, have all been employed to limit laboratory sources of contamination (Champlot et al., 2010; Cooper and Poinar, 2000). Furthermore, the routine use of multiple no-template controls is advocated to detect contamination should it occur (Cooper and Poinar, 2000).

With conventional PCR-based studies, non-laboratory/environmental sources of contamination are, however, more difficult to detect and remedy (Willerslev and Cooper, 2005). These include a variety of plant, animal and microbial DNA sources in the burial environment (NGS studies report only 1-5% endogenous DNA sequences (see Llamas et al., 2017a)) and post-recovery environment (e.g. handling during excavation, contaminants introduced during processing and curation, pests in storage facilities, handling by researchers, etc.). For the latter, special excavation and storage procedures have been advocated (Llamas et al., 2017; Yang and Watt, 2005), but this is not relevant for sources that have been housed in institutions for decades. Sample surface decontamination is often employed, but these do not remove deeply penetrating contaminants and, depending on the methods used, may negatively affect downstream analyses. Novel methods to detect endogenous contamination are, however, possible with NGS. Ancient DNA sequences produce predictable damage patterns that have been used to distinguish authentic sequences from modern contaminants (Briggs et al., 2007; Ginolhac et al., 2011); however, non-damage based methods have also been developed (see Key et al., 2017). Thus, NGS has enabled the detection and removal of contaminant sequences, greatly contributing to authentication in aDNA studies.

Early in the history of aDNA studies, numerous high profile claims were made that were later shown to be false (see Willerslev and Cooper, 2005). The growth of sequence deposits in genetic databases allowed many of these sequences to be revealed as contaminant artefacts resulting from modern contamination or mosaic/chimeric sequences from ‘jumping’ PCR. In other cases attempts to replicate these results were unsuccessful. These high profile failures led to the establishment of a set of ‘criteria of authenticity’ (Cooper and Poinar, 2000; Gilbert et al., 2005a) to be used by researchers and publishers to limit the reporting of erroneous results. NGS has provided new tools to assess the authenticity of ancient sequences, and these criteria have been updated (Key et al., 2017; Llamas et al., 2017).

ANCIENT MOLECULAR TUBERCULOSIS

The first ancient pathogen study was conducted by Spigelman and Lemma (1993) where the authors reported the detection and amplification of *M. tuberculosis* DNA. Since then, the detection of a broad range of viral and bacterial pathogen DNA has been reported (see Marciniak and Poinar, 2018; Tsangaras and Greenwood, 2018). Molecular investigations of ancient tuberculosis have far outnumbered those of any other pathogen (Marciniak and Poinar, 2018). The popularity of ancient tuberculosis research probably relates to the historical and contemporary relevance of this disease, as well as the ability to detect it in the skeletal record. The following section presents an uncritical review of ancient MTBC research; however, concerns have been raised for many of these studies. Since many of the criticisms, particularly those of the conventional PCR era, are broadly applicable to multiple studies, these criticisms will be addressed separately.

Ancient tuberculosis studies have largely focused on disease in ancient human populations, although MTBC DNA has been recovered from a North American bison (Rothschild et al., 2001, but see critisicms below) and Iroquioan dog (Bathurst and Barta, 2004). Ancient MTBC DNA has been retrieved from a variety of sources including naturally and artificially mummified soft tissue of the lungs and pleural cavity; pleural calcifications (Donoghue et al., 1998); archaeological and medical (Barnes and Thomas, 2006) skeletal remains from pathological and non-pathological bone elements; non-frozen, archival tissue samples (Zink and Nerlich, 2004); and even preserved cell cultures of the isolate from which Koch based his initial characterization (Taylor et al., 2003). Successful amplification of ancient MTBC DNA has also been reported for a variety of geographical locations. Most reports stem from European localities, including: United Kingdom (England (Barnes and Thomas, 2006; Bouwman et al., 2012; Bouwman and Brown, 2005; Gernaey et al., 2001; Mays et al., 2002, 2001; Mays and Taylor, 2003, 2002; Müller et al., 2014, 2013; Taylor et al., 2005, 1999, 1996) and Scotland (Spigelman et al., 2002)), Germany (Baron et al., 1996; Taylor et al., 2003; Zink et al., 2007, 2005), Austria (Bachmann et al., 2008), Hungary (Chan et al., 2013; Donoghue et al., 2005; Fletcher et al., 2003a, 2003b; Haas et al., 2000a; Kay et al., 2015; Masson et al., 2013; Pálfi et al., 2015; Pap et al., 1999; Zink et al., 2007), Sweden (Donoghue et al., 2005; Nuorala, 1999) and Lithuania (Faerman and Jankauskas, 2000). Fewer studies have been reported from countries in the Middle East and North Africa, including Turkey (Spigelman et al., 2002), Syria (Baker et al., 2015), Israel (Donoghue et al., 2005, 1998; Hershkovitz et al., 2008; Matheson et al., 2009), Egypt (Crubézy et al., 2006, 1998; Donoghue et al., 2010, 2005; Nerlich et al., 1997; Zink et al., 2007, 2005, 2003, 2001). Few studies have reported successful recovery of MTBC DNA from the Asian continent; however, MTBC DNA has been recovered from Siberian (Dabernat et al., 2014; Murphy et al., 2009; Taylor et al., 2007) and Bornean (Spigelman et al., 2002) specimens. *M. tuberculosis* complex DNA has also been recovered from North and South American specimens (Konomi et al., 2002) from Peru (Bos et al., 2014; Klaus et al., 2010; Salo et al., 1994), Chile (Arriaza et al., 1995; Guichón et al., 2015), Brazil (Guedes et al., 2018; Jaeger et al., 2012), the United States (Barnes and Thomas, 2006; Braun et al., 1998; Raff et al., 2006; Rothschild et al., 2001) and Canada (Bathurst and Barta, 2004; Braun et al., 1998). Ancient DNA reports on the Asian continent are sparse and do not reflect the long historical record of the disease. With the exception of Egypt, there are no reports of ancient tuberculosis DNA recovery from the African continent. While the geographical pattern of successful ancient molecular tuberculosis studies may truly reflect global differences in the presence or absence of the disease, it likely also mirrors geographical differences in the paleopathological record, reflects differences in DNA preservation among the varying climatic regions, and/or results from an uneven distribution of research. MTBC DNA has also been retrieved from a broad temporal range, spanning modern historical specimens to thousands of years. The oldest Egyptian specimen is a 5 400 yr old child, reported by (Crubézy et al., 1998), from the pre-dynastic necropolis of Adaïma. The oldest specimen obtained to date (9250-8160 BP) is from Israel (Hershkovitz et al., 2008); however, the molecular results have been disputed (see Wilbur et al., 2009). Most New World specimens have provided dates between AD 900 and AD 1200 (Arriaza et al., 1995; Bos et al., 2014; Braun et al., 1998; Klaus et al., 2010; Konomi et al., 2002; Raff et al., 2006; Salo et al., 1994). However, Rothschild et al. (2001) report *M. tuberculosis* isolates from a 17870 ± 230 BP North American bison.

MOLECULAR TARGETS IN PALEOMOLECULAR TUBERCULOSIS RESEARCH

Researchers have used a number of molecular techniques and targeted various regions of the *M. tuberculosis* genome in order to recover MTBC DNA and further elucidate the causative isolate. By far the most popular molecular target is the insertion element, IS*6110*. This is a short fragment of DNA thought to be specific to the MTBC, present in 0-27 copies among its members, and widely used in clinical settings. Thus, the multicopy nature and specificity of this locus have made it ideal for the study of ancient tuberculosis. Different assays have been developed for this target - the most popular of which is a 123 bp fragment of the insertion element developed by Eisenach et al. (1990). Many researchers have favoured a further amplification with nested, 97bp (Salo et al., 1994) or more commonly 92bp primers (Taylor et al., 1996). Still others have reported success with larger fragments of 550bp (nested 181bp) (Gernaey et al., 2001) and 245/6bp (Donoghue et al., 2005; Matheson et al., 2009; Rothschild et al., 2001; Spigelman et al., 2002) or have chosen to amplify a slightly different regions of the insertion element (Bachmann et al., 2008; Klaus et al., 2010).

Another MTBC specific insertion element, IS*1081*, has also been used to amplify a 135bp (hemi-nested 113bp) (Bachmann et al., 2008; Hershkovitz et al., 2008; Taylor et al., 2007, 2005, 2003) or a 141bp (nested 113bp) (Bouwman and Brown, 2005) fragment. Jaeger et al. (2012) used membrane hybridization to a 113bp target to identify the IS*1081* fragment in their Brazilian samples, while Harkins et al. (2015) amplified a 84bp region of this element. IS*1081* is present in fewer copies (usually only 6) (Collins and Stephens, 1991), however, it may be more useful for the detection of those *M. tuberculosis* and other MTBC members that have few copies of, or completely lack, IS*6110* elements (Yuen et al., 1993).

Other targets used to identify MTBC aDNA include the heat shock protein 65 gene (*hsp65*) (also referred to as *groEL* or the 65kDa antigen gene) (Crubézy et al., 2006, 1998; Haas et al., 2000a; Konomi et al., 2002; Nerlich et al., 1997), the 19 kDa antigen gene (Fletcher et al., 2003a, 2003b), the S12 ribosomal RNA gene (Matheson et al., 2009; Rothschild et al., 2001; Spigelman et al., 2002), the conserved membrane protein gene (*CMP*) (Hershkovitz et al., 2008) and the RopB gene (Harkins et al., 2015; Klaus et al., 2010). However, these genes are not specific to the MTBC and can be detected in other mycobacterial species. Therefore, sequencing and phylogenetic comparisons should be performed to confirm specificity of amplified products. In fact, this should be attempted for all PCR-based studies.

Another class of molecular loci targeted in ancient tuberculosis studies allow *M. tuberculosis* to be distinguished from *M. bovis* isolates. These include the analyses of SNPs in the β-subunit of the RNA polymerase gene (*rpoB*) (Barnes and Thomas, 2006; Mays et al., 2001; Taylor et al., 1999), the oxidative response regulator (*oxyR*) (Bachmann et al., 2008; Barnes and Thomas, 2006; Fletcher et al., 2003b, 2003a; Mays et al., 2002, 2001; Mays and Taylor, 2003, 2002; Taylor et al., 2003, 1999, 2007, 2005; Zink and Nerlich, 2004) and the pyrazindamidase (*pncA*) gene (Bachmann et al., 2008; Mays et al., 2001; Taylor et al., 2007, 2005, 2003). The *rpoB* and *pncA* genes are associated with resistance to the anti-tuberculosis drugs rifampicin and pyrazinamide, respectively, while the *OxyR* gene has been disrupted in *M. tuberculosis* and is a pseudogene. Deleted regions, referred to as regions of difference, have also been used to differentiate between *M. tuberculosis* and *M. boivs* isolates. A genomic region referred to as mtp40, which includes a phospholipase C gene (*plcA*), is present in ~90% of the MTBC members (Fletcher et al., 2003b) but has been lost in most *M. bovis* isolates (RD5). This region has been used in a number of ancient studies to distinguish *M. tuberculosis* from *M. bovis* isolates (Barnes and Thomas, 2006; Fletcher et al., 2003b; Mays et al., 2001; Taylor et al., 2003, 1999). Other regions of difference that have been used in a similar fashion include RD7 (Fletcher et al., 2003b; Hershkovitz et al., 2008; Mays et al., 2001; Mays and Taylor, 2003), as well as RD4, RD12, RD13 and RD17 (Taylor et al., 2007). Two *M. bovis* specific fragments have also been targeted and include that identified by Rodriguez et al. (1995) (Mays et al., 2001; Taylor et al., 1999; Zink et al., 2003) and the MPB70 antigen gene (Fletcher et al., 2003a).

Other targets have also been investigated in an attempt to distinguish among *M. tuberculosis* isolates. A deleted region (TbD1) is used to distinguish ‘ancestral’ from ‘modern’ tuberculosis isolates (Bachmann et al., 2008; Hershkovitz et al., 2008; Taylor et al., 2007, 2005, 2003). This region is retained in ‘ancestral’ *M. tuberculosis* and animal MTBC strains but deleted in ‘modern’ *M. tuberculosis* strains (Brosch et al., 2002; Sreevatsan et al., 1997). A number of studies have attempted to target SNPs within a number of genes in order to ascertain which group or lineage an isolate belonged to (Barnes and Thomas, 2006; Fletcher et al., 2003a, 2003b; Mays and Taylor, 2003; Taylor et al., 2003). Dabernat et al. (2014) and Müller et al. (2014) investigated additional SNPs using the SNaPshot technique. Fletcher et al. (2003a, 2003b) targeted the *dnaA-dnaN* region, which contains an IS*6110* insertion in isolates of the Beijing family to investigate its presence in an 18th-19th century Hungarian population. Finally, spoligotyping (spacer-oligonucleotide typing) has also been used to identify strain type based on comparisons to modern patterns (Fletcher et al., 2003b; Hershkovitz et al., 2008; Mays et al., 2001; Pálfi et al., 2015; Rothschild et al., 2001; Taylor et al., 2003, 1999; Zink et al., 2007, 2003). Ancient tuberculosis studies have revealed spoligotyping patterns identical to those in modern databases or novel patterns that have not been identified in any clinical modern MTBC isolates. However, the degraded nature of aDNA may complicate inferences from spoligotyping patterns (Stone et al., 2009). These patterns are created by the amplification of spacer regions between a series of repetitive fragments termed the direct repeat (DR) region and the presence or absence of these spacer regions is used to create a strain specific spoligotype pattern. In ancient samples these regions may fail to amplify, due to the degraded nature of the DNA, resulting in incomplete patterns with low reproducibility. The molecular targets discussed above have been used with varying success in molecular studies of ancient tuberculosis, either individually or in combination, to show multi-locus support for species specificity and/or phylogenetic inferences.

More recently, researchers have attempted to use next generation sequencing (NGS) to access genomic DNA. These have included target-enriched (Bos et al., 2014; Bouwman et al., 2012) and non-target enriched methods (Chan et al., 2013; Kay et al., 2015). NGS studies of ancient tuberculosis have utilized two different sequencing platforms, SOLiD (Bouwman et al., 2012) and Illumina (Bos et al., 2014; Chan et al., 2013; Kay et al., 2015).

ANCIENT MOLECULAR TUBERCULOSIS STUDIES: RELEVANCE

Early paleomolecular tuberculosis studies aimed to confirm paleopathological diagnoses through the detection of MTBC DNA (e.g. Arriaza et al., 1995; Salo et al., 1994; Spigelman and Lemma, 1993). While this is still the primary focus of many contemporary studies, specimens with atypical pathologies and non-pathological specimens have also been analysed. Furthermore, questions on the origin of the disease, disease co-infection, as well as, population-based analyses and the characterization of ancient isolates have also been addressed.

One of the first major contributions of paleomolecular tuberculosis research concerned the debate on the origin of tuberculosis in the New World. Salo et al. (1994) were the first to report the detection of MTBC DNA from a Peruvian mummy, and subsequent reports have confirmed the presence of tuberculosis in the New World prior to its ‘discovery’ by Columbus (Arriaza et al., 1995; Bathurst and Barta, 2004; Bos et al., 2014; Braun et al., 1998; Jaeger et al., 2012; Klaus et al., 2010; Konomi et al., 2002; Raff et al., 2006; Rothschild et al., 2001). Thus, it is possible that the disease accompanied its human host early on in the peopling of the Americas. However, Rothschild et al. (2001) reported *M. tuberculosis* DNA from a 17 000 yr-old North American bison. The authors suggest that early bovids may have been a reservoir for the disease and that this find may have bearing on how tuberculosis reached the Americas. Bos et al. (2014) report *M. pinnipedii* DNA from Peruvian samples revealing seals as a possible source of infection. Bathurst and Barta (2004) recovered MTBC DNA from a 16th century Iroquoian dog and suggest that domesticated dogs may have also served as a reservoir for the disease. Pre-colonial/European tuberculosis has also been reported in a specimen from Borneo (Spigelman et al., 2002; Spigelman and Lemma, 1993). If the Hershkovitz et al. (2008) data are accepted, then molecular support for tuberculosis in the Old World extends to 9250-8160 BP (but see Wilbur et al., 2009).

The inclusion of rib lesions as a diagnostic criterion of tuberculosis has been much debated. Paleomolecular studies have ventured to weigh-in on this debate; however, results and interpretations have varied. Gernaey et al. (2001) sampled pathological ribs from two individuals, one of whom tested positive for the IS*6110* molecular target. The authors concluded that there may be some support for correlating pathological rib lesions and tuberculosis but caution that, based on their results alone, this correlation is tenuous. Raff et al. (2006) were able to detect TB in all samples showing proliferative rib lesions (n=5) and, therefore conclude that rib lesions can be correlated with tuberculosis infection. Mays et al.

(2002) arrive at a different conclusion. Based on their investigation of pathological lesions on the visceral rib surface of seven individuals they conclude that there is no association between rib lesions and tuberculosis, or at least the recovery of MTBC DNA, because IS*6110* detection was no more frequent in pathological cases than in non-pathological controls.

Other atypical pathological changes sometimes attributed to tuberculosis infection have also been investigated. Donoghue et al. (2010) reported the recovery of MTBC DNA from 'Dr Granville's mummy' – the first Egyptian mummy to be scientifically autopsied. Granville attributed cause of death to an ovarian tumour; however, subsequent histological analysis has indicated that the tumour was benign. Histological analysis also revealed the presence of a pulmonary exudate, which led the authors to investigate tuberculosis as the possible cause of death. Haas et al. (2000) investigated the molecular evidence of tuberculosis in Hungarian samples sub-divided into three categories – specimens showing pathology typical of tuberculosis, specimens showing pathology probably due to tubercular infection and specimens showing atypical pathologies indicating non-specific inflammatory processes. The IS*6110* fragment was detected in four of the five specimens of the last category, leading the authors to conclude that minor pathological changes may represent early stages of the disease and can be detected using molecular tools. The cause of hypertrophic osteoarthropathy has also been investigated (Hershkovitz et al., 2008; Mays and Taylor, 2002).

While initial studies focused on specimens showing preserved soft-tissue or skeletal pathologies associated with tuberculosis, later studies investigated the possibility of recovering MTBC DNA from non-pathological loci within pathological specimens, as well as from specimens lacking any skeletal pathology. For example, Baron et al. (1996) reported MTBC DNA recovered from three German autopsy specimens from both pathological and non-pathological regions of pathological individuals. Faerman et al. (1997) attempted the molecular detection of tuberculosis at different loci (pathological and non-pathological) within the same individual, as well as, in individuals without any pathological lesions and were able to detect MTBC DNA in all. In addition, Nuorala (1999) reported the detection of MTBC DNA from a single specimen with "no morphological changes typical of tuberculosis" recovered from a 17th century Swedish ship in the Baltic Sea. In their investigation of rib lesions and tuberculosis, Mays et al. (2002) report positive MTBC DNA amplification from one pathological and two non-pathological (control) specimens. The successful recovery of MTBC DNA from non-pathological skeletal specimens spurred population-based investigations of disease prevalence and the relationship between disease state and epidemiological factors. Zink et al. (2007, 2005, 2003, 2001) have investigated the presence of tuberculosis in a relatively large number of Egyptian specimens over a relatively broad geographic area. In these studies the authors attempted to recover MTBC DNA from specimens showing pathology typical of tuberculosis, atypical of tuberculosis and also included roughly equal numbers of non-pathological individuals. DNA was detected in a relatively high number of specimens of all three categories. For example, Zink et al. (2007) report MTBC DNA recovery from 9 of 11 (82%) specimens showing pathology typical of tuberculosis, 8 of 37 (22%) specimens showing atypical pathology and 20 of 112 (18%) of non-pathological specimens. Based on these investigations the authors conclude that tuberculosis was prevalent throughout ancient Egypt in both geographical and temporal contexts. Fletcher et al. (2003a) also adopted a population-based approach to their investigation of tuberculosis in a 18th -19th century Hungarian population. In this study 55% of samples were positive for MTBC DNA. The authors also compared differences in amplification success with epidemiological factors including age at death, year of birth, sex and familial relationships. In addition, the relationship between MTBC DNA recovery, sampling site and gross morphology/presence of pathology were also investigated. Of these factors, only age at death was significantly associated with positive MTBC DNA amplification, with a greater proportion of positive amplification in the 51-60 yrs age category.

MTBC DNA has also been recovered in a study adopting a multiple pathogen screening approach to diagnosing disease in specimens lacking pathological lesions. Konomi et al. (2002) tested for a number of pathogens in 12 Andean mummies of which two were positive for IS*6110* markers. Similarly, researchers investigating leprosy in ancient specimens have detected MTBC DNA. In their investigation of specimens indicating evidence of leprosy, Haas et al. (2000b) also tested for the presence of IS*6110*, which they were able to detect in one *M. leprae* negative specimen. Co-infection of *M. tuberculosis* and *M. leprae* in ancient specimens has been reported by Matheson et al. (2009) and Donoghue et al. (2005).

Donoghue et al. (2005) specifically investigated *M. tuberculosis-M. leprae* co-infection in an attempt to evaluate the role of tuberculosis on the historical decline of leprosy. Based on their results the authors conclude that tuberculosis infection did not confer immunity against leprosy (given the high rate of co-infection among the specimens investigated). They further posit that leprous individuals with depressed immunity may have been more likely to die of tuberculosis, which would effectively reduce the chances of spreading leprosy and ultimately contribute to the decline of this disease.

As was previously mentioned, IS*6110* can be detected in all MTBC members (but not necessarily in all isolates); therefore, successful amplification of IS*6110* cannot resolve which MTBC member is responsible for a positive molecular signal. The identification of a specific MTBC member from ancient samples can support or question inferences from modern phylogenetic studies concerning the evolutionary history of tuberculosis, as well as contribute to our understanding of the relevance of the non-*M. tuberculosis* members in the past. Thus, alternative molecular targets have been employed to distinguish among MTBC members. Researchers have largely focused on distinguishing between *M. tuberculosis* and *M. bovis* isolates because this has bearing on the debate concerning the origin of human tuberculosis. Taylor et al. (1999) were the first to identify a specific MTBC member from ancient specimens showing pathology associated with tuberculosis. Using a variety of molecular targets, the authors reported that *M. tuberculosis* rather than *M. bovis* was responsible for disease in the specimens under investigation. In fact, *M. tuberculosis* has been identified as the causative agent in most studies investigating the MTBC member responsible for infection (Hershkovitz et al., 2008; Mays et al., 2001; Mays and Taylor, 2002; Rothschild et al., 2001). Only three studies have reported the recovery of MTBC members other than *M. tuberculosis.* Zink et al. (2003) reported *M. africanum*-like isolates from Egyptian specimens based on spoligotyping results, while Taylor et al. (2007) reported the recovery of *M. bovis* from Iron Age Siberian specimens. Lastly, Bos et al. (2014), the only study to report *non-M. tuberculosis* isolates using NGS, identified *M. pinnipedii* from Peruvian samples. Investigations of genetic groups/lineages and strain types have also been attempted. Taylor et al. (2003) were able to attribute Koch's isolate to Sreevatsan et al. (1997) genetic groups 1 or 2, while the samples investigated by Fletcher et al. (2003a) could be attributed to groups 2 or 3. Furthermore, Fletcher et al. (2003a) investigated whether any of these isolates belonged to the Beijing family. In Fletcher et al. (2003b), the authors identified spoligotypes 53 and 50 from an 18th century Hungarian family. These spoligotypes differ only in the presence/absence of one spacer and are among the most common spoligotypes found globally today (Brudey et al., 2006). NGS has allowed the identification of lineage and strain type in four studies. With the exception of the *M. pinnipedii* strain reported by Bos et al. (2014), all other cases have been attributed to the Euro-American lineage 4, common throughout the world today. Bouwman et al. (2012) were able to retrieve MTBC genomic sequences from a 19th century burial in St. Georges Crypt, England which was closely related to the laboratory strain H37Rv. More specifically, the authors attributed these to lineage 2 (Baker et al., 2004, equivalent to lineage 4, Gagneux and Small, 2007) and strain ST14 or ST40 of the SNP cluster group six (Filliol et al., 2006). Chan et al. (2013) also report a strain from lineage 4, 7199/99, in their analysis of an individual from the 18th century Vác crypt in Hungary. Kay et al. (2015), expanding on the previous study, report 14 genomes obtained from 8 individuals all belonging to lineage 4. In some studies lineage or strain identification was not possible but the authors were able to note some phylogenetically important information. For example, Hershkovitz et al. (2008) showed the TbD1 deletion was already present by ~9 000 BP (but see Wilbur et al., 2009 for a critique), while Fletcher et al. (2003a) showed that mutations in the *katG* and *gyrA* genes were present by the 18th century.

While some lineage/strain information has been reported with conventional PCR studies, NGS promises access to a wealth of information about ancient pathogens. However, despite the growing popularity of next-generation sequencing (NGS) technologies in aDNA research in general, its application in ancient tuberculosis research has been slow. This is likely linked to the challenges facing ancient pathogen research which are outlined below. Only four studies to date have reported ancient MTBC genomic sequences. These studies have addressed a number of topics including strain continuity through time, the prevelance of mixed infections and the origin of pre-Columbian tuberculosis in the New World. They have also aided in refining molecular dating estimates. As was discussed above, three of these studies were conducted on historical 18th and 19th century individuals from Hungary (n=8) and England (n=1) (Bouwman et al., 2012; Chan et al., 2013; Kay et al., 2015). These genomes have all been attributed to the Euro-American lineage 4, and although not identical to modern strains in all cases, do suggest lineage continuity through time (Kay et al., 2015). Kay et al. (2015) report five (of eight) individuals with mixed genotype *M. tuberculosis* infections, arguing against a clonal outbreak and suggesting mixed infections were common in this 18th century Hungarian population. This supported the results of Chan et al. (2013) who, in an earlier publication, reported a mixed infection in a single individual from the same site. Bos et al. (2014) in a study that screened 68 individuals from the New World for the presence of MTBC DNA, identified *M. pinnipedii* sequences from three Peruvian individuals, suggesting seals as a possible source of pre-Columbian tuberculosis in the New World. The genomes reported in Bos et al. (2014), Chan et al. (2013) and Kay et al. (2015) were used to calculated mutation rates and estimate molecular divergence dates for the MTBC. Bos et al. (2014) and Kay et al. (2015) provide a much younger estimate (< 6 000 yrs BP) for the emergence of the MTBC than that previously reported (Comas et al., 2013; Wirth et al., 2008). Although negative results are not often reported, Guichón et al. (2015) report insufficient coverage in preliminary assays from a 14th-15th century individual from Tierra del Fuego, Chile, for full genome capture with NGS. Forst and Brown (2016) attempted to use whole genome amplification (WGA) (without NGS) to improve the yield of ancient pathogen DNA for downstream PCR analyses; however, the researchers determined that the method used in that study produced inconsistent results and did not provide a benefit over traditional PCR methods.

Non-DNA biomolecular studies investigating tuberculosis have also been conducted on ancient specimens. Histological analyses have been attempted to detect the presence of acid fast bacilli, however, MTBC members are not the only bacteria that will yield positive results when treated with acid fast stains (Arriaza et al., 1995; Salo et al., 1994; Taylor et al., 2003; Zink et al., 2005). High performance liquid chromatography (HPLC) has also proven useful for the detection of *M. tuberculosis* specific mycolic acids (Baker et al., 2015; Donoghue et al., 2010, 1998; Gernaey et al., 2001; Hershkovitz et al., 2008; Masson et al., 2013). These long-chain fatty acids are expressed on the surface of the mycobacterial cell wall. Hershkovitz et al. (2008) report the detection of mycolic acids from two Israeli specimens dated to ~9 000 BP. These are the oldest specimens for which mycolic acids have been detected. Mycolic acids have also been detected using methods other than HPLC and these include matrix-assisted laser desorption/ionization time-of-flight mass spectrometry

(MALDI TOF MS) and Fourier transform infrared spectroscopy (FT-IR) (Mark et al., 2010). Other types of *M. tuberculosis* specific biomolecules have also been successfully retrieved from ancient samples. Redman et al. (2009) report the detection of mycocerosic acids using HPLC and gas chromatography mass spectrometry (GC-MS) in their analysis of 19th century Portuguese specimens, while Boros-Major et al. (2011) analysed proteins from 8th-18th century Hungarian specimens using matrix-assisted laser desorption/ionization tandem time-of-flight mass spectrometry (MALDI TOF/TOF MS), direct sequencing and peptide mass fingerprinting (PMF).

CRITICISM OF ANCIENT PATHOGEN STUDIES

With the advent of NGS, ancient pathogen research is now considered a reputable sub-discipline of the ancient DNA field; however, this has not always been the case (Hofreiter et al., 2015; Llamas et al., 2017). Of particular concern to critics of ancient tuberculosis studies in the PCR era is the fact that many show much higher rates of successful amplification than, for example, studies of ancient mammalian DNA. In addition, the sequences obtained are almost always identical to those of modern sequences deposited in genetic databases (Willerslev and Cooper, 2005). This has led to questions concerning the authenticity of these results, which, it is argued, may represent environmental or laboratory contamination rather than authentic aDNA.

CONTAMINATION, ASSAY SPECIFICITY AND ASSAY DESIGN

The three main sources of contamination for paleopathogen studies are; i) modern counterparts affecting excavators, individuals handling specimens and/or laboratory researcher; ii) laboratory contamination ‘carried-over’ from positive controls and previous PCRs and iii) modern environmental bacteria with similar sequences or those amplified by non-specific primer annealing (Gilbert et al., 2004). Some paleomicrobiologists argue that the risk posed by laboratory-based sources of contamination are not as serious as that encountered in ancient human research and have argued for a relaxation of the criteria of authenticity (Donoghue, 2011; Taylor et al., 2010). The risk posed by sample contamination with environmental bacteria has led to questions concerning assay specificity and design

(Stone et al., 2009; Wilbur et al., 2009). Many of the molecular targets and assays used in ancient tuberculosis studies were initially developed and tested in clinical settings. However, the archaeological environment provides a variable and diverse range of modern bacterial contaminants which include many free-living, closely related mycobacteria. Assay specificity in such a context is rarely reported. This insertion element shares homology with the IS3 family and nontuberculous mycobacteria (NTM) may contain IS3-like elements as suggested by the detection of positive PCR amplification (Liébana et al., 1996) and probe cross-hybridization (Bhanu et al., 2004; Mchugh et al., 1997) in several NTM species (*M. gilvum*, *M. ulcerans*, *M. avium-M. intracellulare*, *M. fortuitum*, *M. kansasii*, and *M. malmoense*). Müller et al. (2015) have shown the lack of specificity of the Eisenach et al. (1990) IS*6110* primers (the most widely used molecular target of ancient tuberculosis studies) in an archaeological context. These primers produced amplicons of the correct size but the NTM identity was confirmed by sequencing. In addition to questions concerning the specificity of MTBC specific fragments, the use of SNPs in ancient studies has also been queried (Wilbur et al., 2009). Not all of the molecular targets used in ancient tuberculosis studies are specific to the MTBC, for example, the *rpoB*, *gryA* and *katG* genes are present in other mycobacterial species. Wilbur et al. (2009) argue that without an understanding of SNP diversity in other closely related mycobacteria, it is impossible to evaluate the authenticity of results.

Other assay-based criticisms bring to light shortcomings in assay design or interpretations of results. While some of these problems can be avoided, this may not always be the case. The IS*6110* insertion element may be the most popular molecular target among ancient tuberculosis research but it is not present in all strains of *M. tuberculosis* (although reports of *M. tuberculosis* strains lacking the IS*6110* element are relatively rare). This does reduce the ability of this assay to discriminate between the absence or presence of MTBC DNA because a negative result can be interpreted as a true absence of MTBC DNA or reflect the presence of a strain lacking the IS*6110* element (Wilbur et al., 2009). The IS*1081* insertion element is thought to be present in all MTBC members and strains; therefore, this marker has greater discriminatory ability. However, the specificity of this assay has also been brought into question (Wilbur et al., 2009). A similar argument can be made for other molecular targets that are present in some but not all of the targeted MTBC members or strains. These include the TbD1 deletion, which is present in some but not all strains of *M.*

tuberculosis, and the mtp40 fragment, which is present in most *M. tuberculosis* isolates and absent in most *M. bovis* isolates (discussed in Wilbur et al., 2009 and Taylor et al., 2003 respectively). The detection of genomic deletions is also problematic. A negative result (failed amplification) may occur if the targeted region has been deleted. Alternatively, even if the region has not been deleted, amplification may fail because the DNA is too degraded. Thus, assays based on presence/absence of a deleted region will be complicated by the fact that a negative result could indicate a true absence of the region or failed amplification due to poor DNA preservation. This can be partly resolved through the use of internal and flanking primers that amplify the targeted region when it is present or absent, respectively, but this approach has not been universally adopted. The use of spoligotyping has also been criticised (Stone et al., 2009; Wilbur et al., 2009). Degraded spacers may fail to amplify resulting in inaccurate spoligotype patterns. Furthermore, different spacers may amplify, or fail to do so, in different assays performed on the same sample. Thus, when dealing with ancient sequences, the generation of accurate and reproducible spoligotypes is hampered by low DNA quantity and quality. In addition, the evolution of this region is complex and spoligotype patterns are known to be subject to convergence (Filliol et al., 2006; Warren et al., 2002), limiting the utility of phylogenetic inferences based on this assay. Finally, the use of nested PCR in aDNA studies is problematic because the use of amplification products in the nested stage carries an increased risk of contamination (Bandelt, 2005; Barnes and Thomas, 2006).

ADHERENCE TO CRITERIA OF AUTHENTICITY

The criteria of authenticity were proposed to limit the reporting and publication of non-authentic aDNA results. The original nine criteria were proposed by Cooper and Poinar (2000) during the PCR era. These include a dedicated and physically isolated laboratory, the use of multiple no-template controls to detect contamination, an observation of appropriate molecular behaviour, reproducibility, verification of amplified PCR products through cloning, replication of results in an independent laboratory, a demonstration of biochemical preservation (e.g. amino acid preservation), quantitation to assess the likelihood of sporadic contamination and the demonstration of DNA survival in associated remains (e.g. faunal remains). Gilbert et al. (2005) advocate adding a common sense approach when assessing authenticity of aDNA research. Adherence to all of these criteria is not typically a prerequisite for publication and remains the prerogative of the researcher. Roberts and Ingham (2008) evaluated the quality of 65 ancient pathogen research papers (of which tuberculosis was the most common disease considered), published between 1993 and 2006 based on their adherence to 15 criteria. In addition to the nine criteria proposed by Cooper and Poinar (2000), publications were assessed on a further six criteria taken from published literature. The additional criteria include: sterile sampling at the excavation level, dedicated work areas for aDNA analysis, DNA records for all personnel, the use of protective clothing, the removal of sample surface contamination and the analysis of host mitochondrial and nuclear DNA. They concluded that some criteria were better adhered to than others but that, overall, the authentication criteria were poorly described. Furthermore, the adherence to and description of authentication criteria did not show a significant improvement in quality over time. Lack of basic contamination control (90%) and independent verification of results (80%) were highlighted as the criteria to which adherence was poorest. Other infrequently reported criteria included amplification of mtDNA, biochemical preservation, quantitation, and analysis of associated remains. In addition, the authors note that it was often unclear if anti-contamination procedures were genuinely not performed or whether researchers simply failed to report them and also call for greater reporting of negative results. Tsangaras and Greenwood (2012) conducted a similar evaluation using seven authentication criteria and arrived at a similar conclusion. The authors noted that 21 of the 30 studies on Mycobacteria (which included 22 studies of *M. tuberculosis* and 8 of *M. leprae*) adhered to only two or three of the criteria investigated. Based on the inconsistency between the high positive amplification rates of studies with poor adherence to authentication criteria and the low positive rates of studies with good adherence to criteria, the authors conclude “the most parsimonious explanation for this contradiction is that a large number of the positive results are contaminant derived sequences and will require verification before they can be generally accepted as authentic.”

In response to these criticisms some researchers have argued that strict adherence to the criteria of authenticity is not essential for ancient pathogen research (Donoghue, 2009; Taylor et al., 2010). Specifically, researchers have questioned the need for strong paleopathological support for a diagnosis of tuberculosis; dedicated, isolated laboratories;

amplification of host mtDNA, associated faunal remains and soil samples; DNA database generation for all lab personal and cloning and sequencing of multiple clones (Donoghue, 2011, 2009; Donoghue et al., 2009; Taylor et al., 2010). Furthermore, Donoghue et al. (2009) and Taylor et al. (2010) argue that false negatives (amplification failure in MTBC-DNA containing samples due to inadequate sample storage, extraction techniques and PCR inhibition) might explain the low success rates reported in some publications. The argument for relaxating adherence to authentication criteria for ancient MTBC research is centred on two main tenets, namely, the superior preservation of MTBC DNA and the lower risk of laboratory contamination when working with ancient pathogen DNA.

Crubézy et al. (1998) were the first to suggest a protective role for the mycobacterial cell wall, in an aDNA context, after the death of the host organism. Subsequently, Spigelman and Donoghue (1999) proposed that the hydrophobic, lipid-rich cell wall served to protect mycobacterial DNA from the initial decay process following host death and used this to explain the high success rates of ancient MTBC studies when compared to other aDNA studies. In addition, Donoghue et al. (2004) propose that the high GC-content of mycobacteria increases DNA stability and further aids preservation.

The other argument, that laboratory contamination is less of a concern for ancient MTBC studies, is based on the premise that there are fewer sources of mycobacterial contamination within the laboratory than there are human sources of contamination (Donoghue and Spigelman, 2006). For example, researchers and reagents may serve as sources of contamination for human aDNA studies and contribute to the ease at which contamination occurs in such studies, but are less likely to serve as sources of contamination for ancient pathogen studies.

If these assertions hold, then there may be merit in a call for the relaxation of authentication criteria. The arguments for the omission of debated criteria are outlined below. The generation of DNA databases for laboratory personnel and the use of dedicated, isolated laboratories for aDNA analyses are recommended to detect and limit laboratory based sources of contamination. It is argued that these are more relevant for human aDNA research than ancient MTBC research (where laboratory contamination is less of a concern and can be successfully detected by the inclusion of sufficient negative controls) (Donoghue et al., 2009; Taylor et al., 2010). The successful amplification of host mtDNA may lend support to the authenticity of any pathogen DNA recovered. This is viewed as unnecessary based on the superior preservation of MTBC DNA and reports of successful MTBC DNA amplification in samples where human DNA amplification has failed (Taylor et al., 2010). A similar argument is made against the utility of analysing associated faunal remains, since these may not always be present and will have little bearing on the recovery of ancient MTBC DNA (Taylor et al., 2010). The analysis of associated soil samples may aid in the detection of soil/environmental contaminants. Taylor et al. (2010) argue that this is relevant if the DNA of interest could be present in soil but view the long term survival of MTBC DNA in soil as highly unlikely; thus, there would be little benefit to performing this assay. Finally, cloning and the sequencing of multiple clones can detect damage-derived miscoding lesions (which may support the authenticity of sequences), as well as, the presence and/or abundance of contaminant derived sequences in a given sample. These days NGS has largely superseded cloning and provides novel methods of assessing authenticity, which are outlined below. Nevertheless, prior to the NGS era, those debating the value of cloning argued that assay specificity was adaquate and direct sequencing of amplicons was, therefore, sufficient for the well preserved MTBC DNA. Furthermore, it was argued that cloning may introduce additional polymerase-derived sequence errors and carry an increased risk of contamination.

While the assertion that mycobacterial DNA is better preserved might be true, the role of the mycobacterial cell wall and high GC-content, as pertains to DNA preservation, has not been empirically tested. Critics contend that until this can be demonstrated, it cannot be used to argue for a relaxation in authentication criteria (Stone et al., 2009; Wilbur et al., 2009). Furthermore, Stone et al. (2009) note that cases of irreproducible or failed amplifications would not support this assertion. With regards to the lower risk of laboratory contamination in ancient MTBC studies, Wilbur et al. (2009) contend that the risk posed by carry-over contamination (cross-contamination from positive samples or controls of previous reactions) should not be underestimated and that laboratory contamination in ancient pathogen studies may actually be more difficult to detect due to the lack of variation in commonly used markers (e.g. IS*6110*, IS*1081*). It is impossible to entirely prevent contaminants from entering a reaction and negative controls may not always be sufficient to detect contamination due to the carrier-effect; therefore, the ability to detect contamination after it has occurred is also important. The limited sequence variation among the MTBC makes it difficult to detect contaminant sequences and, as a result, it is argued that adherence to authentication criteria should be more, not less, strict for ancient MTBC studies.

In addition to assay specificity and adherence to authentication criteria, concerns have also been raised regarding the analysis of specimens based on questionable paleopathological diagnoses and the likelihood of DNA preservation. Wilbur et al. (2009) and Stone et al. (2009) have queried the destructive analysis of specimens based on questionable diagnoses of tuberculosis (e.g. *serpens endocrania symmetrica* and HOA). These authors argue that analyses based on such questionable diagnoses may result in the unwarranted destruction of irreplaceable samples. The likelihood of DNA preservation in ancient Egyptian specimens has been repeatedly questioned (Gilbert et al., 2005b; Marota et al., 2002). The high mean annual temperature, periodic flooding of tombs, high humidity within tombs and, possibly, mummification techniques and treatments all act to limit the survival of DNA and as a result, ancient Egyptian DNA findings are still controversial.

Given the challenges and concerns surrounding paleomicrobial research, Klaus et al. (2010), referring to PCR-based studies, recommend that molecular methods not be viewed as the final authority on a diagnosis of tuberculosis. Molecular methods should rather be used as a supporting line of evidence in conjunction with other lines of evidence (e.g. paleopathological lesions consistent with a diagnosis of tuberculosis, histological and/or other non-DNA biomolecular assays) to create a more convincing argument for the presence of tuberculosis in a given specimen.

More recently, NGS has become a valuable tool for paleomicrobial research; however, PCR-based methods are still usefull for identifying candidate samples for NGS. Specifically, quantitative PCR has been recommended as a fast and efficient screening tool that aims to reduce costs and enhance NGS efficiency by identifying candidate samples with the best chance of success (Harkins et al., 2015). NGS has also provided novel methods to evaluate the validity of aDNA. Consequently, Key et al. (2017) and Llamas et al. (2017b) report new recommendations for authenticating ancient human and pathogen molecular studies. Recommendations for the use of dedicated facilities, clean room practices and negative controls remain unchanged. With respect to negative controls, sequencing depth should be at a sufficient level to detect sporadic contamination and in the event that such contamination is detected, the mapping of contaminant sequences and evaluation of damage profiles is suggested to identify the source of contamination. The authors also caution against the use of barcoding and pooling of multiple samples and negative controls during amplification citing the risk of jumping PCR in creating chimeric sequences. Additional general recommendations include the careful selection of source material/sampling site and the use of extraction protocols that are appropriate for short, fragmented aDNA. Environmental contaminants are no less of a concern for NGS studies than they are for PCR-based studies (as outlined above) and NGS DNA libraries are typically rich in environmental bacteria. The authors, therefore, outline different procedures for conventional and competitive mapping that can be used to limit the misidentification of environmental contaminants as pathogen DNA. Studies have shown that ancient DNA exhibits a specific DNA damage profile that differs from modern contaminants (Briggs et al., 2007). Thus, an evaluation of damage profiles can aid in the authentication of ancient pathogen DNA. Similarly, read distribution uniformity, fragment length profiles and read mismatches should all conform to expectations of degraded, ancient genomes.

In this regard, Campana et al. (2014) have criticized the publication by Chan et al. (2013), who reported the *M. tuberculosis* genome of a single Hungarian individual, citing issues with their mapping methodology, which they argue could create false positives, and limited reporting which restricts the ability to assess the authenticity of their claims. These issues were partly addressed in a subsequent publication (Kay et al., 2015); however, the authors here report that damage patterns do not match those typically expected of ancient DNA. This has been observed in ancient *Mycobacterium leprae* (*M. leprae*) molecular studies and authors have attributed this to the good relative preservation of *M. leprae* DNA (Schuenemann et al., 2018, 2013); however, like with the MTBC, claims of superior preservation have not been supported empirically.

In summary, modern and ancient DNA studies have proved to be an invaluable tool for the study of organisms in the past. However, these studies are not without challenges and/or controversies. Ancient pathogen DNA studies have been particularly popular among those investigating tuberculosis in the past and a large number of studies have reported the successful amplification of ancient MTBC DNA. However, issues of DNA preservation, assay design and the authenticity of results have plagued the field. In addition to confirming paleopathological diagnoses, a number of researchers have attempted to acquire sequence information that may shed light on the subspecies, strain or lineage to which the isolate may belong. This has met with limited success and remains a challenge for researchers. The use of NGS technologies in ancient DNA studies has become increasingly popular; however, reports of ancient tuberculosis genomes have been limited. Nevertheless, NGS holds great promise and has marked the onset of a new era in ancient pathogen research, allowing more nuanced questions of the origins and nature of tuberculosis in the past and the evolution of the pathogen itself.

CHAPTER 4: MATERIALS AND METHODS

MATERIALS

Skeletal material from seven institutions was included in the analysis. Together, these institutions house the majority of archaeologically derived human skeletal material in South Africa. For more information on some of these collections please see Dayal et al. (2009), L'Abbé et al. (2005) and Morris (1992). In total 2321 individuals were examined representing 2077 unique accession numbers. There were 71 unaccessioned / unlabelled individuals. For the commingled remains, an estimated 849 individuals were present in 234 boxes. In total, an estimated 3170 individuals, including individual and commingled remains, were analysed in this study. A breakdown of the number of specimens analysed by institution is presented in Table 2. A few sources of error when estimating commingled remains must be acknowledged. There were a number of instances, particularly when multiple individuals were unearthed from the same location, where some elements had been separated into individuals and placed in separate boxes, while the unsorted remains were placed in a box together. Thus, boxes containing comingled remains may not contain additional individuals but rather unsorted/unidentified elements from existing, partially sorted, individuals. Furthermore, in some instances the same individual(s) may have been stored over multiple boxes with multiple (not always contiguous) accession numbers. I have attempted to address this where possible but associations are not always clear.

SKELETAL METHODS

All archaeologically derived, Holocene and historical human skeletal material housed in an institution were included in the analysis, with the exception of the single individual included from the University of Cape Town (UCT). This individual was identified as possible case of tuberculosis from the literature and no other cases of tuberculosis had been identified in the collection (personal communication, Alan Morris). Not all institutions were able to provide a catalogue of curated human remains; therefore, any material excluded was not done so intentionally. All information present in the institution catalogue, on boxes, labels, notes or skeletal elements was recorded. The type and completeness of information was highly variable but typically included location of burial, excavator/donor name, contact details, date of donation/excavation/accession, and much more infrequently, estimated sex and broad age-at-death. The presence and nature of any additional material stored with the accessioned human remains was also recorded. This additional material included skeletal elements from other individuals, non-human skeletal remains, and other organic and inorganic artefacts.

Where more than one individual was represented by a single accession number the following strategies were adopted. If the number of additional elements was limited these were simply recorded as additional elements from another individual. However, if there were a substantial number of additional elements the individuals were recorded as separate entries designated by letters e.g. SAM-AP … [A] and [B]. Square brackets were used for my own designations. Where multiple individuals had already been assigned a sub-accession designation, the institution designation was used. These included alphabetical, numerical and roman numerical characters. After all data were collected, a number of duplicate accessions were noted. These either represented the same individual separated over multiple boxes, in which case the entries were merged, or clearly represented different individuals from different localities (when location information was present). The latter may represent recording/transcription errors on my part or, alternatively, represent genuine, unintentional duplicate accession numbers at the institution or mislabelled boxes. Depending on the analysis these were either differentiated through the addition of symbols or were excluded from the analysis.

Table 2: Frequencies of human remains analysed at South African institutions.

Institution	Accession prefix	Number of individuals	%	Number of unique Accession IDs[a]	Number of unaccessioned remains / unlabelled items	Commingled remains: Number of accessioned items[b]	Estimated number of individuals from commingled remains[c]	Total estimated number of individuals[d]
Iziko South African Museum	SAM-AP	835	35.98	769	0	11	38	873
University of the Witwatersrand (WITS)	A	393	16.93	356	0	3	8	401
National Museum Bloemfontein	NMB	424	18.27	357	36	51	146	570
McGregor Museum	MMK/GLD	285	12.28	254	19	96	349	634
Albany Museum	ALB	280	12.06	255	5	71	299	579
University of Pretoria	UP	103	4.44	85	11	0	0	103
University of Cape Town	UCT	1	0.04	1		2	9	10
Total		2321	100	2077	71	234	849	3170

[a] Accession numbers representing multiple individuals (e.g. ALB 226 A and B) were collapsed and only counted once. These values also exclude unaccessioned /unlabelled items.

[b] These counts reflect the number of boxes examined containing commingled remains.

[c] Estimates are based on MNI and adjusted when differences in size and/or age indicated the presence of additional individuals.

[d] These values are the sum of the number of individuals (excl. commingled remains) and the estimated number of individuals from commingled remains.

SKELETAL INVENTORY

A complete inventory of skeletal elements was recorded for each specimen using the written and visual recording forms provided in Buikstra and Ubelaker (1994). The “Inventory recording form for complete skeletons” and the adult, juvenile or cranial visual recording forms were used when an accession number represented a single individual or represented multiple individuals that could be easily distinguished from one another. The “Inventory recording form for commingled remains and isolated bones” was used when the remains of multiple individuals could not be easily distinguished from one another. For commingled remains the number of individuals present was estimated by determining the Minimum Number of Individuals (MNI). The MNI was calculated by counting the most abundant element or the most abundant of sided elements (Max(L,R)). Values were adjusted when obvious age or size difference indicated that additional individuals were present that would not be accounted for by the calculation of MNI alone.

Degree of preservation was reported according to the recommendations in Buikstra and Ubelaker (1994). Scores of 1, 2 and 3 represent element or sub-element preservation of a complete (100-75%), partial (75-25%) and fragmentary/poor (<25%) nature. For bilateral elements left and right sides were scored separately. Some elements were scored collectively, e.g. ribs 3-10. In the case of these grouped elements, counts of the number of elements present were provided, as well as the average score of elements. For some elements sub-sections/segments were given individual scores, e.g. long bones were scored in five parts: the proximal and distal epiphyses were scored independently, and the shaft was divided into three sections, each of which were scored individually.

For juveniles with unfused vertebral elements the preservation scores for grouped vertebral neural arches were recorded as L and R NA halves separately. Both the number of elements and the preservation score were recorded (e.g. vert C 3-6 NA scored as 4/1 (4 elements with average preservation score of 1)) I entered this information in two ways, either as summed scores for L and R halves (which provides a more accurate reflection of the number of elements without including additional categories; but this does not allow comparison to adult grouped vertebral scores) or as minimum number of elements (by taking the largest of the sided NA halves and reflecting difference in side by reducing the preservation score, which allows comparison with adult scores but does not give an accurate reflection of number of juvenile elements present. The following grouped scores were added to this study for recording juvenile remains: vertebrae (C3-7, T1-12, L1-5). State of fusion and unfused epiphyses were recorded as comments.

SEX ESTIMATION

Sex estimation was performed following Buikstra and Ubelaker (1994). Sex estimation for adult specimens was based on the assessment of a number of morphological features of the pelvis and skull. The pelvic features assessed include the presence of the ventral arc, subpubic concavity and ischiopubic ramus ridge (Phenice, 1967); shape of the greater sciatic notch and elevation of the preauricular sulcus. The cranial and mandibular features assessed include rugosity of the nuchal crest; size of the mastoid process; shape of the supraorbital margin and prominence of the glabella and mental eminence. Scores were assigned to each morphological feature in accordance with the scheme outlined in Buikstra and Ubelaker (1994) and left and right sides were scored independently where applicable. Additional morphological features not included Buikstra and Ubelaker (1994) were also assessed; these include shape of the pubic bone (Coleman, 1969), shape of the obturator (St. Hoyme, 1984) and shape of the orbits (White and Folkens, 2000). Based on the scoring profiles each individual was assigned to one of six categories (Table 3), following Buikstra and Ubelaker (1994). Sex estimation of younger juvenile material was not attempted.

Table 3:**Sex estimation categories and descriptions (reproduced from** Buikstra and Ubelaker (1994)**)**

Category	Description
Undetermined sex	Insufficient data are available for sex deterimation
Female	There is little doubt that the structure represent female
Probably female	The structures are more likely female than male or sex determination was based on a single or a few of the less reliable morphological features (sciatic notch, preauricular sulcus, mental eminence, orbit shape)
Ambiguous sex	Sexually diagnostic feature are ambiguous
Probably male	The structures are more likely female than male or sex determination was based on a single or a few of the less reliable morphological features (sciatic notch, preauricular sulcus, mental eminence, orbit shape)
Male	There is little doubt that the structures represent a male

AGE-AT-DEATH ESTIMATION

Age-at-death estimations for adults were based on multiple assessments using morphological features of the pubic symphyseal face (Brooks and Suchey, 1990; Katz and Suchey, 1986; Todd, 1921, 1920), the auricular surface of the ilium (Lovejoy et al., 1985; Osborne et al., 2004), the sternal surface of the ribs (İşcan et al., 1993, 1985, 1984; Oettlé and Steyn, 2000) and cranial suture closure of the endocranium (Buikstra and Ubelaker, 1994), ectocranium (Meindl and Lovejoy, 1985) and palate (Mann et al., 1991).

For the pelvic features, left and right sides were scored separately. Where phase scores differed between the left and right sides, or features resembled more than one phase / age indicator (i.e. were contradictory), the age range was expanded to include both phase age ranges. For the Suchey-Brooks method, the 95% range and mean were used to aid the age-at-death estimation (Brooks and Suchey, 1990). For the auricular surface, the method of Lovejoy et al. (1985) was used. The age ranges produced by the prediction intervals of the 6 phase category for Osborne et al. (2004) were also used.

The right 4th rib was used for assessment of age-at-death from the sternal end of the ribs. This was substituted by the left 4th rib if the right side was absent or damaged. If both left and right 4th ribs were absent/damaged, the 3rd or 5th ribs were substituted. This method was only used when ribs 3-5 could be identified. Age-at-death estimations from the sternal rib ends were not made for the entire sample; observations were not recorded for material examined at the Iziko South African museum, as the method was only included for use at a later stage. Işcan et al. (1987) noted that population differences may exist in age-determination using their method. This was investigated by Oettlé and Steyn (2000) in a black South African population. These authors found that the Iscan phases estimated this age-at-death of this group poorly and provided new method to age black South Africans. Thus, the Oettlé and Steyn (2000) method was also used to aid the age assessment.

Cranial suture closure assessments for bilateral measures were based on the left side unless damaged/obscured, in which case the right side was substituted. Suture closure was scored on a scale of 0 to 3 as recommended in Buikstra and Ubelaker (1994). Composite Scores and age ranges of the ectocranial vault and latero-anterior sites were determined based on Meindl and Lovejoy (1985). The age ranges as determined by Mann et al. (1991), as well as broad age ranges as suggested by Buikstra and Ubelaker (1994), were used for the palate. For endocranial suture closure, individuals were placed into broad age categories as suggested by Buikstra and Ubelaker (1994). Numerous studies have indicated that cranial suture closure methods do not perform well (see Falys and Lewis, 2011). Consequently, these methods, while recorded whenever possible, were only relied upon when the pelvis and ribs were not sufficiently well preserved.

Juvenile age-at-death assessments were based on dental eruption patterns utilizing the chart in Ubelaker (1989), as well as, spheno-occipital and epiphyseal union (Schaeffer et al., 2009). Tooth formation, although considered a more reliable indicator of chronological age, was difficult to score consistently as teeth were often fixed in alveolar sockets. Consequently, this method was not included.

Note that many of these methods were not developed on South African populations and while some have been tested and either accepted or the methods revised, others have not; thus caution must be applied when interpreting results. Furthermore, these test and revisions typically apply to modern South African populations and may not be appropriate for the use on Holocene and historical South African populations. Age-estimation methods for the Khoesan in particular are poorly represented and this may lead to greater inaccuracy in age-estimations for this population.

Final age assessments for individuals were reported in a number of ways. Age was reported as mature if all epiphyses were fused and immature if not. In addition, broad age categories of pre-natal (before birth), infancy (birth to 1 yr), early childhood (2-5 yrs), late childhood (6-9 yrs), adolescence (10-19 yrs), young adult (20-34 yrs), middle adult (35-49 yrs) and old adult (50+ yrs) were also reported. The juvenile categories were adopted from Scheuer and Black (2004, Table 1.4.), while the adult age ranges were adopted from Buikstra and Ubelaker (1994). In this classification scheme, the age of 20 is used as the cut off between adult and juvenile. The appropriate age category was determined based on the assessment of multiple age indicators. Methods based on the pubic symphyseal face were considered more reliable than those of the auricular surface. The rib-based methods were infrequently used due to preservation issues. And all other methods were considered more reliable than those based on cranial suture closure. Where age estimates could not be readily confined to a single category, multiple categories were combined and all were reported (e.g. middle to old adult).

CHRONOLOGICAL DATE

All dates were obtained from reports in the literature and were derived from a combination of absolute and relative methods. Uncalibrated radiocarbon dates were binned into 1000 yr categories. Relative dates were based on a variety of sources including historical records, associated archaeological material and known periods of occupation of sites. The specificity of these dates varies considerably with narrow age ranges associated with more recent interments, when historical records are available, to very broad age range associating individuals with a particular time period or cultural group (e.g. Late Iron Age).

BURIAL LOCATION

Location information was sourced from institutional catalogues, information provided in notes stored with the remains, information written on the remains and literature reports. Co-ordinates provided are not necessarily specific to the exact location of the remains but do indicate the general area in which they were found. Locations were plotted in Google Maps using the My Maps feature and coordinates were exported in KLM files. Maps were created in QGIS (V1.8.0, QGIS Development Team, 2015). Map backgrounds/ vector data were sourced from Natural Earth (2015) and current provincial boundaries from the Demarcation Board (2015). Additional image editing was performed in Inkscape (Inkscape Team, 2016) and GIMP (GIMP Team, 2013).

PATHOLOGY

When possible, pathology was classified into the following categories: trauma, developmental disorders, joint disease, metabolic disease, infectious disease and neoplastic disease. Uncategorized pathology was recorded as abnormal bone loss, abnormal bone formation, abnormal bone shape or abnormal bone size. A large number of journal articles covering both the clinical and paleopathological literature, as well as commonly used reference books including Aufderheide and Rodríguez Martín (1998), Barnes (1994), Buikstra and Ubelaker (1994), (Grauer, 2012), Katzenberg and Saunders (2008), Mann and Hunt (2005), Ortner (2003), Pinhasi and Mays (2008) and Waldron (2009a) were consulted in order to aid in the descriptions and diagnoses of pathology and anatomical anomalies/disorders.

TRAUMA

Only trauma present on well preserved elements (>75% complete) is reported.
Criteria for fractures include visible fracture line/non-union or noticeable change in angle.
Cranial depressions were not included as fractures if they were small, there were no visible

fracture lines and no corresponding protrusion of the internal table. Pathological fractures are reported but not included in analysis.

For each lesion, the type of trauma was recorded. This includes fracture, dislocation, amputation, heterotopic ossification and osteochondritis dissecans. The element, side (where applicable) and sub-element/segment were recorded, as was a description of the lesion location including any additional relevant features. Additional information recorded for fractures included whether the fracture was simple or comminuted and complete or incomplete. Furthermore, the type of fracture was recorded and this included penetrating, crush (depression/compression), transverse, spiral, oblique, greenstick, avulsion, stress and pathological fractures. Any predisposing/pre-existing conditions that may have resulted in the observed lesion were noted. Complications arising from the traumatic lesion were noted and included osteoarthritis, infection, ankylosis, nerve injury, haematoma ossification, non-union and malunion. In addition, the type of fracture malunion was recorded. Types of malunion included angulation, displacement/translation, distraction, shortening and rotation.

DEVELOPMENTAL DISORDERS

Persistent sutures, bipartite elements and accessory ossicles were only diagnosed in adult individuals. Styloid processes exceeding 30mm were considered abnormal in length (Eagle, 1943). Kumar and Tubbs (2011) suggest that all anomalies involving a failure of fusion in the posterior spine be referred to as spina bifida or spinal dysraphisms. Failure of fusion was referred to as a cleft when occurring on the anterior or posterior arch of the 1st cervical vertebra, the zygapophyseal facets or any other non-spinal location. Noticeable anterior or lateral loss of vertebral height, without evidence of trauma, was referred to as vertebral body hypoplasia. Similarly, diminished or absent structures, with no evidence of trauma or pathological changes to the bone, were considered hypoplastic or aplastic, respectively. Fused elements were considered developmental in origin if there was no evidence of infection, joint disease or trauma. Similarly, bipartite elements and accessory ossicles were diagnosed when there was no evidence of trauma; however, fractures should still be considered as an alternative diagnosis.

JOINT DISEASE

The frequency of osteoarthritis was investigated by noting the presence and extent of marginal osteophytes, surface osteophytes, pitting, alteration of the surface contour, eburnation and grooving. Waldron (2009a) suggests osteoarthritis only be diagnosed when eburnation or two other features associated with the disease, as listed above, are noted. Intervertebral disc disease was diagnosed when both pitting and marginal osteophytes were present (Waldron, 2009). In addition, the number of affected joints was recorded as monoarticular (if a single joint was affected), pauciarticular (if fewer than 5 joints were affected) and polyarticular (if 5 or more joints were affected).

OTHER

The presence of other pathological conditions either did not lend themselves to inclusion into broad categories, occurred in low frequencies or lacked a secure/confident diagnosis. These are presented in the results.

DATA MANIPULATION AND STATISTICAL METHODS

Data manipulation and analyses were performed in excel (Microsoft, 2010) and R (Version 3.4.2, R Core Team, 2017). Initial data collection and basic data cleaning were performed in excel. Most of the subsequent data manipulations and analyses were performed in R using the base, plyr (Wickham, 2011) and reshape2 (Wickham, 2007) packages. Preservation scores for which the element or side were uncertain, as well as age and sex determinations based on partially damaged or pathological elements, were removed from the dataset for subsequent analyses.

In order to generate descriptive statistics, grouped, segment and sided element preservation scores were adjusted when necessary. Firstly, all scores were converted to proportions as follows: score 1 = 1, score 2 = 0.74 and score 3 = 0.25. Grouped scores (average score for a set of grouped elements) were adjusted to reflect the number of elements present. The product of the average score (now converted to a proportion) and number of elements present was then divided by the total number of possible elements in the set. Values greater than or equal to 0.75 are given a new score of 1, those less than or equal to 0.25 are given a new score of 3 and the remaining values, a score of 2. This adjustment was performed for the carpals, tarsals, metacarpals, metatarsals, hand and foot phalanges. Where parts of an element, i.e. element segments, were scored separately, the weighted, converted scores were summed and the resulting values evaluated as described for grouped elements. Element segments were weighted by equal proportions (see Table 4 for proportions) and this was applied to the os coxa, humerus, radius, ulna, femur, tibia and fibula. The sternal manubrium, body and xyphoid show variable fusion among adults and can be considered a segmented or grouped element. These three sternal scores were converted into a single score in the same manner as the segmented elements but weights were not of equal proportions (see Table 4). Vertebral neural arch and body segments were scored separately and these were converted into a single score as described for the other segmented elements. For adults, vertebrae C1-2, C7, T-2, T10-12 and L1-5 were scored individually, while C3-7 and T1-9 were scored as grouped sets. For juveniles with unfused vertebrae C3-7, T1-12 and L1-5 were scored as grouped sets. All grouped vertebral scores were adjusted by taking the product of the score proportion and the number of elements present (as described above). The sum of the group values and individual proportions were then divided by the total number of true vertebral elements (24) and this proportion converted to a single score of 1, 2 or 3 for all true vertebrae. The ribs were also scored as a combination of individual (1st, 2nd, 11th and 12th) and grouped (3rd-10th) elements and these scores were adjusted as described for the vertebrae to result in a single score for all left, right and unsided ribs. For sided elements, left and right score proportions were halved and the new proportions summed to create a single score per element. In the case of grouped elements, where elements could be scored as left, right or unsided, the product of the average score proportion and number of elements present for each category was summed and this value was divided by the total number of possible elements for left and right sides combined. Thus, all scores when condensed/collapsed were adjusted to account for missing segments, sides and elements.

Table 4: **Proportional weights used to adjust preservation scores for grouped or segmented elements**

Element/Grouped Elements	Segments/Elements	Weights*
Humerus, radius, ulna, femur, tibia and fibula	Proximal epiphysis, Proximal 1/3 of shaft, Middle 1/3 of shaft, Distal 1/3 of shaft, Distal epiphysis	$^{1}/_{5}$
Ribs	1st, 2nd, 11th, 12th, 3rd-10th	$^{1}/_{12}$
	Centrum, Neural arch	$^{1}/_{2}$
Vertebrae	C1, C2, C3-6, C7, T1-9, T10, T11, T12, L1-5 (/juv: C1, C2, C3-7, T1-12, L1-5)	$^{1}/_{24}$
	Manubrium	0.45
	Body	0.45
Sternum	Xyphoid	0.1
Os coxae	Pubis, ilium, ischium	$^{1}/_{3}$
Tarsals		$^{1}/_{7}$
Carpals		$^{1}/_{8}$
Metacarpals, metatarsals		$^{1}/_{5}$
Hand phalanges, foot phalanges		$^{1}/_{14}$

*Each segment/element listed was weighted by multiplying the score proportion with the ”weight” proportion.

In order to present an average preservation score for each individual, all individual element scores were condensed into a total skeletal preservation score. This was calculated by summing the weighted preservation scores of each element/grouped element. Proportions/weights were taken from Silva et al. (2009). This publication reported the weights of skeletal elements on an early 20th century Portuguese population. While there are proportional differences in skeletal weights from different population groups (e.g. Lowrance and Latimer, 1964), this study was selected as a reference because it was conducted on a cemetery derived, rather than a cadaver/prepared, skeletal population and the entire skeleton was weighed (as opposed to only a few selected elements such as long bones which is often the case in bone density measurement studies) (Galloway et al., 1997; Willey et al., 1997). Additional grouping of elements was required to match the data reported in Silva et al. (2009). This was performed as described above for grouped elements and the weights used are provided in Table 5. The proportions used to weight the skeletal elements were the mean percentage skeletal weights taken from Table 6 in Silva et al. (2009) and these are reproduced in Table 6.

Table 5:**Proportions used to calculate grouped element scores to match** Silva et al. (2009)

Element/Grouped Elements	Segments/Elements	Weights*
Cranium	Frontal, parietal, temporal, occipital, sphenoid, maxilla, zygomatic	$^{1}/_{7}$ each
Vertebral column	Vertebrae	$^{24}/_{30}$
	Sacrum	$^{5}/_{30}$
	Coccyx	$^{1}/_{30}$
Hand	Carpals	$^{1}/_{5}$
	Metacarpals	$^{2}/_{5}$
	Hand phalanges	$^{2}/_{5}$
Foot	Tarsals	$^{4}/_{7}$
	Metatarsals	$^{1}/_{7}$
	Foot phalanges	$^{2}/_{7}$

* For sided elements, weights apply to side-condensed/adjusted scores.

Table 6:**Mean percentage skeletal weights reproduced from table 6**, Silva et al. (2009)

Element/Grouped Elements	Mean percentage skeletal weights
Skull	17.75
Mandible	1.79
Humerus	6.39
Radius	1.97
Ulna	2.5
Femur	18.45
Tibia	10.51
Fibula	2.30
Hand	2.62
Foot	5.69
Clavicle	0.98
Scapula	2.82
Coxae	8.4
Patella	0.59
Ribs	5.85
Sternum	0.41
Column	10.31

Relative frequencies were obtained by dividing the relevant frequency by the total sample size and multiplying the resulting proportion by 100. Pearson Chi-square tests for goodness of fit and independence were performed on 1- or 2-way tables, respectively. P-values smaller than 0.05 were considered sufficient to reject the null hypothesis. Unless otherwise indicated, expected cell values are greater than five. For contingency tables with >1 degrees of freedom (*df*), expected values of 1 can be tolerated provided no more 20% of the expected values are less than 5 (Cochran, 1954). Chi-square tests on I x J contingency tables are considered omnibus tests that do not reveal the nature or strength of association among variables, furthermore, Chi-square tests are also sensitive to sample size. Consequently, an evaluation of Pearson residuals and odds ratios was performed to identify the nature and strength of association among variables when the null hypothesis was rejected. Large residuals, those that exceed the absolute value of 2 or 3, indicate a lack of fit to the null hypothesis for individual cells (Agresti, 2013). Residuals were visually depicted on shaded mosaic plots. Odds ratios and corresponding confidence intervals were used to support Chi-square results and provide a measure of association. All confidence levels were set at 95% and the null hypothesis of independence (odds ratio, Θ=1) was rejected if the confidence interval excluded the null hypothesis. When observed frequencies of zero were present, a small value of 0.5 was added to all observed frequencies (Agresti, 2013). For small sample sizes, Fisher's exact test was used and two-sided p-values are reported.

Log-linear analyses were used to test association for 3-way tables when more than one variable could be considered dependent. Algorithmic stepwise procedures of backward elimination were used to identify the best fitting model; however, nested models were also assessed manually. Model fit was evaluated by examining and comparing p-values (rejected at the 5% significance level) for the residual deviance statistic (G^2) and change in deviance (ΔG^2) between hierarchical models. Models for which the deviance statistic was not significant, i.e. the null hypothesis was not rejected, were considered for selection. In this case the null hypothesis states that the fitted model frequencies and observed frequencies are not significantly different. Similarly, where there was no significant difference between nested models, the more parsimonious model was selected. Akaike information criteria (AIC) and Baysian information criteria (BIC) were also compared to identify the model with the best fit, i.e. the models with the lowest AIC and BIC values were considered for selection. An evaluation of standardized and studentized deviance residuals was conducted and residuals with an absolute value greater than 2 were considered a poor fit. Observed and fitted values were also evaluated to assess model fit. If 2-way associations are the highest order associations in the selected model, exponentiated model coefficients are equivalent to the odds ratios. Stepwise backwards elimination sometimes indicated that a non-comprehensive model had the best fit. These include models that do not contain all main effects. This indicates that the dimensionality of the data can be reduced e.g. a 3-way table can be redrawn as a 2-way table by summing over the nonsignificant variable (Bishop et al., 1975).

The stringr (Wickham, 2017) package, in addition to a number of R base functions, was used to aid in data cleaning and manipulation. Chi Square (chisq.test) statistics and p-values (pchisq) were calculated in R using the stats package (R Core Team, 2017), while odds ratios (loddsratio(log=F)) and confidence intervals (confint) were calculated using the vcd (D. Meyer et al., 2016) package. Log-linear models were generated using the *glm()* function (stats package) with family=poisson, link=log and default settings. Stepwise model selection was conducted using stepAIC of the MASS package (Venables and Ripley, 2002). Bar graphs and mosaic plots were created using ggplot2 (Wickham, 2009) and vcd (D. Meyer et al., 2016; Meyer et al., 2006; Zeileis et al., 2007), respectively. Mosaic plots were saved as enhanced metafiles using the devEMF package (Johnson, 2017). Other packages, namely gridExtra (Auguie, 2017) and ggpubr (Kassambara, 2017), were used to aid graphical presentation. Additional layout editing was performed in Inkscape.

GENETIC MATERIALS AND METHODS

All genetic analyses were performed by the author, in the laboratories of Professor Anne C. Stone at the Arizona State University.

ANTI-CONTAMINATION METHODS

DNA extraction and PCR assay preparations were performed in a facility dedicated to the study of ancient DNA. The laboratory consists of an outer chamber, in which bone samples were processed, and an inner chamber, in which DNA extractions and PCR assay preparations were performed. The inner chamber is a class 10 000, HEPA filtered clean room with laminar air flow. Overhead UV lights are active when the clean room is not in use.

No amplified DNA is allowed in the clean room and surfaces and equipment are routinely treated with a 10 or 20% bleach solution, sterile water and ethanol. Sterile and UV-irradiated disposable equipment and reagents were used where possible. Protective clothing was worn when working with aDNA and included a full forensic body suit with hood, shoe covers, double gloves, face mask and face shield. PCR amplification and post-amplification assays were performed in another laboratory, in a separate building. The daily movement of laboratory personnel was restricted to unidirectional travel from the aDNA to the modern DNA laboratory. The use of positive controls was limited as much as possible and all modern DNA templates used as positive controls were added in the modern DNA laboratory.

SAMPLE PROCESSING

Destructive analysis and export permits were obtained from the South African Heritage Resource Agency (SAHRA) and Heritage Western Cape (HWC), subject to the approval of the curating institution. Where possible, suitably sized fragments were selected from already damaged elements; however, this was only possible for some of the ribs. Approximately 2-3 cm^3 of bone was sampled from pathological lesions using a Dremel rotary tool with disposable cutting wheels. Smaller samples were cut from the original sample again using the rotary tool or small saw blades. The rotary tool, cutting wheels, grinding bits and saw blades were irradiated in a UV-crosslinker prior to use. All non-pathological surfaces were cleaned with a blade or the grinding attachment of the drill. All non-disposable drill parts and surfaces were cleaned with a 20% bleach solution and 70% ethanol solution between each sample. Disposable cutting wheels were used on a single sample and then discarded. Samples were powdered using one of two methods. Smaller fragments with greater trabecular bone were placed in UV-irradiated foil and pulverised into a powder with a hammer. Larger denser samples were powdered using a bone mill.

DNA EXTRACTION

DNA extractions were performed using the method of Rohland and Hofreiter (2007) and Rohland et al. (2010). This is a silica-based extraction method with an EDTA-proteinase K digestion. In summary, 100mg of powdered bone was incubated in a rotary incubator at 37°C overnight in a EDTA-Proteinase K buffer (0.45M EDTA, 0.25mg/ml proteinase K, total reaction volume = 1.5ml). The DNA sample was then incubated for a further 3 hrs at 37°C to facilitate binding to size-fractionated silica in the presence of a binding buffer (5M GuSCN, 0.3M Sodium Acetate). The silica pellet was then washed once with binding buffer and twice with washing buffer (50% v/v EtOH, 0.125M NaCl, 10mM Tris, 1mM EDTA). Finally, the sample was eluted in 50µl TE buffer (10mM Tris, 1mM EDTA), this process was repeated and eluates combined to a final volume of 100µl. One or two no-template controls were included with each extraction.

PCR

Following the extraction, a PCR was conducted to test for contamination and the presence of human mtDNA. The 9-bp deletion primers (L8215, H8297) of Wrischnik et al. (1987) were used and the sequences are reproduced in Table 7. If contamination was detected in any of the no-template extraction controls, the extraction was repeated. PCR assays included 5µl of sample DNA, 1x Platinum buffer, 0.2mM each dNTP mix, 0.8mg/ml BSA, 1.5mM $MgCl_2$, 0.5µM of each forward and reverse primer, and 0.02U/µl of Platinum® Taq DNA Polymerase (Invitrogen, Carlsbad, CA, USA) to a final reaction volume of 25µl. Cycling conditions include an initial denaturing step at 95°C for 4 mins, followed by 40 cycles of denaturing at 94°C for 30secs, annealing at 54°C for 30secs and extension at 72°C for 30secs. The final extension step was 6 min long at 72°C. PCR results were visualized by standard Gel Electrophoresis (2.5% Agarose) and the product size verified by comparison to a 1KB DNA ladder. Two no-template controls were included for each assay.

Three assays, amplifying portions of the IS*6110* and IS*1081* insertion elements, as well as the *rpoB* gene, were conducted to test for the presence of MTBC DNA. Due to greater specificity, Taqman probes were used and the primer and probe sequences are reported in Table 7. For each reaction, 2µl of DNA template, the Taqman probe and the forward and reverse primers were added to a 1x Taqman® Universal Master Mix II, no UNG (Applied Biosystems, Forster City, CA, USA) to a final reaction volume of 20µl. The primer and probe concentrations differed for each assay and are reported in Table 7. The qPCRs were conducted on an ABI 7900HT and cycling conditions include an initial step of 95°C for 10mins, followed by 50 cycles of 95°C for 15secs and 60°C for 1min. A five series 10 fold standard dilution was prepared from the *M. tuberculosis* H37Rv strain representing 10-100 000 copies. Three replicates for each standard, sample and no-template control were included in the qPCR assay.

Table 7: **PCR and qPCR primers and probes used in this study.**

Target/ Assay	Amplicon size (bp)	Reference	Primer/ Probe	Sequence	Final concentration (nM)
Human mtDNA (9bp deletion)	112 (with deletion) 121 (without deletion)	Wrischnik et al. (1987)	F primer	ACAGTTTCATGCCCATCGTC	500
			R primer	ATGCTAAGTTAGCTTTACAG	500
IS*6110*	63	Klaus et al. (2010)	F primer	GGGTAGCAGACCTCACCTATGTG	50
			R primer	CGGTGACAAAGGCCACGTA	300
			Probe	ACCTGGGCAGGGTT	60
IS*1081*	84	Harkins et al. (2014)	F primer	GCACTCCATCTACGACCAG	300
			R primer	GGGAGTTTGTCGGTCAGAG	300
			Probe	ATTGGGCAACAACTGATTCGGCGTCG	300
*RpoB*2	70	Harkins et al. (2014)	F primer	CAACGTCGAGGTGCTATCG	300
			R primer	CTCCAGGTCCTCGTCCTCA	300
			Probe	TCGCCGCACCGTCACT	150

CHAPTER 5: RESULTS

This chapter is divided into two parts. The first consists of a general description of all data collected for this project. This includes an analysis of overall preservation, preservation differences among institutions, as well as preservation differences among skeletal elements, sides and sub-elements. Sex and age distributions are reported and the relationship between these variables and preservation is explored. Distributions of the chronological age of the individuals examined are presented, as are the geographical locations of burials, where these could be identified. Finally, a summary of the observed pathology, excluding tuberculosis, is presented. The second part presents a detailed description of all possible cases of tuberculosis identified in this study.

COMPLETE ASSEMBLAGE

TOTAL SKELETAL PRESERVATION

Figure 3 presents the relative frequencies of total skeletal preservation scores. In general, skeletal preservation is poor among the South African skeletons examined. More than half of the sample (53%) is poorly preserved (<25% of the skeleton is present), 27% of the individuals present show intermediate preservation (25-75% of the skeleton is present) and 20% are well preserved (>75% of the skeleton is present). The chi-square goodness-of-fit statistic is significant ($\chi^2 = 438.94$, $df = 2$, $n = 2321$, $p < .0001$) and the null hypothesis of equal proportions can be rejected.

This pattern of total skeletal preservation appears to be consistent among institutions, with the exception of MMK, NMB and UP (Figure 4a). A chi-square test of independence is significant ($\chi^2 = 170.24$, $df = 10$, $n = 2321$, $p < .0001$) indicating the null hypothesis of independence between preservation score and institution can be rejected. Examination of the residuals show that MMK, NMB (|Pearson's residuals| > 4) and to a lesser extent UP and

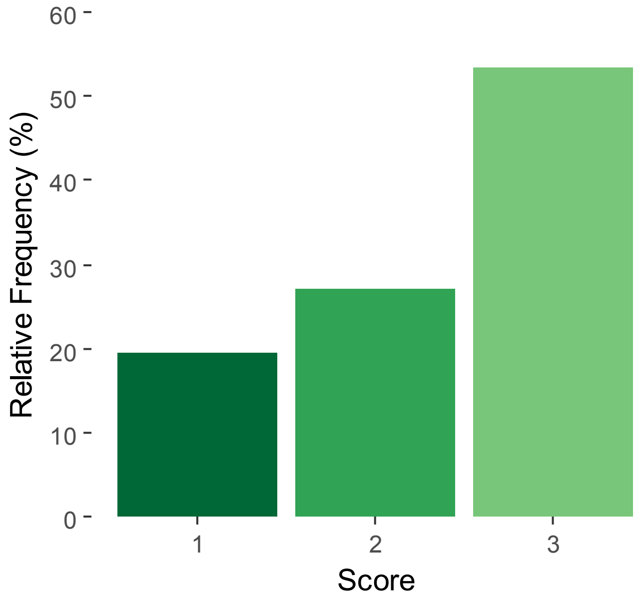
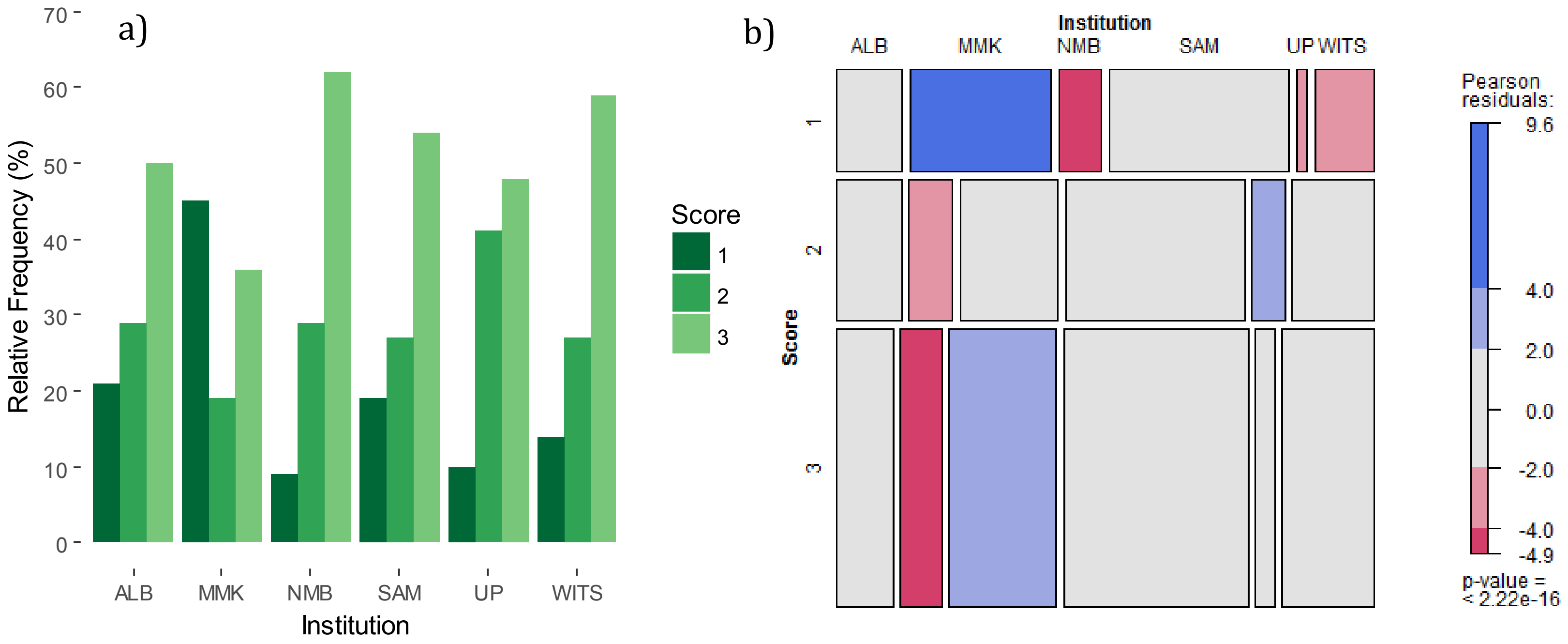


Figure 3: Relative frequency of total skeletal preservation by score (n=2307). Preservation scores reflect the percentage of the total skeleton present: 1=75-100%, 2=25-50% and 3=0-25%. Grouped, sided and subdivided elements have been condensed into a single element category and scores are adjusted accordingly. These element scores were grouped to match Silva et al. (2009) and weighted according to the proportions outlined in the publication to provide a single preservation score for each individual.

WITS (|Pearson's residuals| > 2) are driving the departure from independence (Figure 4b). Oddsratios (using a score of 2 and the institution ALB as reference levels) and their corresponding confidence intervals (95%) show that only MMK, NMB and UP have confidence intervals that do not include the null (Figure 4c). The skeletal collections at NMB and UP show poorer preservation than the other institutions and, specifically, have fewer well preserved (score 1) individuals. The odds of encountering well preserved skeletons are approximately 2 times greater for ALB than NMB and approximately 3 times greater for ALB comparted to UP (Figure 4c). The converse is true for MMK which has the greatest relative proportion of well-preserved individuals. Just over a third of the MMK sample (n=106/285) consists of individuals unearthed from the Gladstone cemetery (GLD). These burials occurred relatively recently (AD 1879-1900, van der Merwe et al., 2010) and the remains are generally well preserved (Figure 5a). A chi-square test of independence reveals a significant difference between group (GLD or other) and preservation score ($\chi^2 = 80.66$, $df = 2$, n = 285, $p < .0001$). Residuals indicate this difference is driven by the large number of well preserved (score 1) individuals in the GLD group and the small number of well preserved individuals in



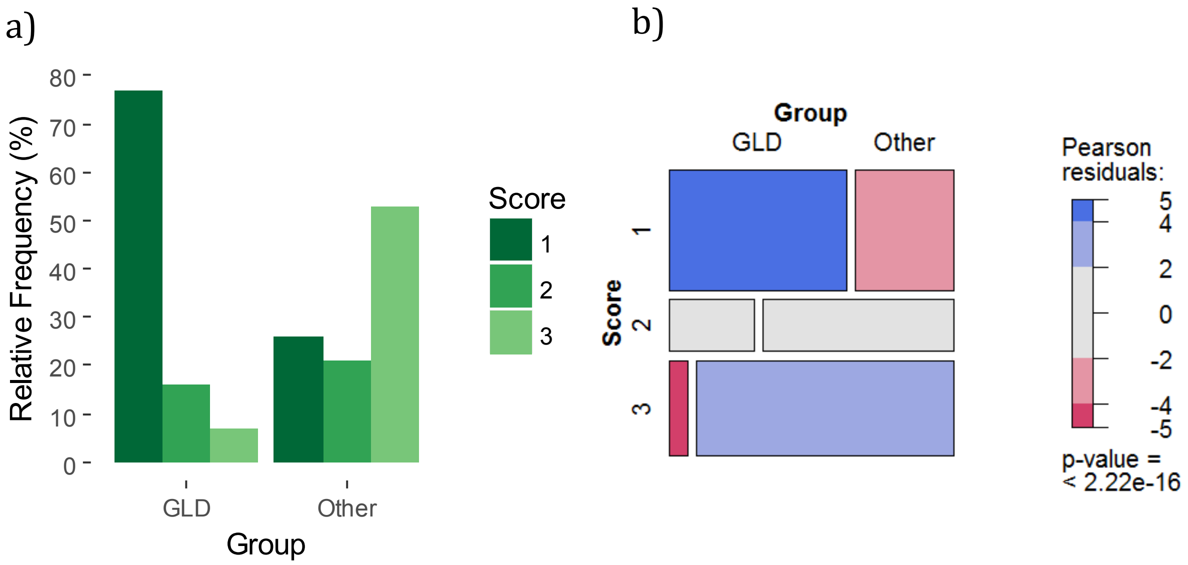
c) **Oddsratios (95% CI)**

Score	ALB:MMK	ALB:NMB	ALB:SAM	ALB:UP	ALB:WITS
1:2	**0.32 (0.2-0.5)**	**2.32 (1.42-3.79)**	1.01 (0.69-1.5)	**3.11 (1.45-6.69)**	1.43 (0.89-2.28)
2:3	1.08 (0.7-1.66)	1.26 (0.89-1.78)	1.17 (0.85-1.61)	0.71 (0.43-1.16)	1.32 (0.92-1.88)

Figure 4 Relative frequency plot, mosaic plot (with shaded residuals) and oddsratio table for institution and preservation scores. a) Relative frequency of total skeletal preservation by institution stratified by score (n=2321). Relative frequencies were obtained by dividing each score by the total number of individuals observed in each institution. These institutional totals are provided with the institutional code descriptions below. b) Mosaic plot showing total skeletal preservation by score and institution. c) Table showing oddsratios with 95% confidence intervals. Confidence intervals that do not contain the null hypothesis are shown in bold. Preservation scores reflect the

percentage of the total skeleton present: 1=75-100%, 2=25-50% and 3=0-25%. Institutional codes: ALB=Albany Museum (n=280), MMK=McGregor Museum (n=285), NMB=National Museum Bloemfontein (n=424), SAM=IZIKO South African Museum (n=836), UP=University of Pretoria (n=99), WITS=University of the Witwatersrand (n=382). The single individual from the University of Cape Town (UCT) has been removed.

the 'other' group (Figure 5b). Oddsratios show that this effect is relatively large and 95% confidence intervals do not contain the null (Figure 5c). If the GLD individuals are removed from MMK, the chi-square test of independence between preservation score and institution is still significant ($\chi^2 = 53.05$, $df = 10$, $n = 2215$, $p < .0001$). This reduces all of the residuals but well preserved individuals for MMK, NMB and UP still have absolute Pearson's residuals greater than two (Figure 6a). However, only NMB and UP have 95% confidence intervals for the oddsratios that do not contain the null (Figure 6b).

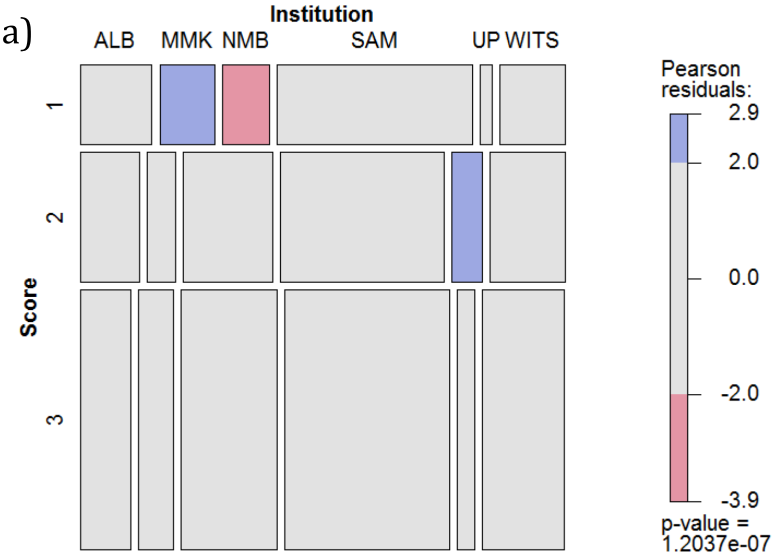


c) **Oddsratios (95% CI)**

Score	GLD:Other
1:2	**3.98 (2.03-7.84)**
2:3	**6.07 (2.33-15.81)**

Figure 5: Relative frequency plot, mosaic plot and odds ratio table for McGreggor Museum (MMK) individuals. a) Relative frequency of total skeletal preservation of MMK individuals (n=285) showing the difference among preservation scores between individuals unearthed at the Gladstone cemetery (GLD) and those unearthed/donated from other locations. b) Mosaic plot with shaded Pearson's residuals. c) Table showing oddsratios and 95% confidence intervals. Confidence intervals that do not contain the null hypothesis are shown in bold.

a)



b) **Oddsratios (95% CI)**

Score	ALB:MMK	ALB:NMB	ALB:SAM	ALB:UP	ALB:WITS
1:2	0.61 (0.36-1.05)	**2.32 (1.42-3.79)**	1.01 (0.69-1.5)	**3.04 (1.41-6.54)**	1.4 (0.88-2.24)
2:3	1.46 (0.91-2.32)	1.26 (0.89-1.78)	1.18 (0.86-1.62)	0.68 (0.41-1.12)	1.29 (0.9-1.85)

Figure 6: Mosaic plot and odds ratios comparing institution and preservation score after the removal of all Gladstone individuals from the McGregor Museum (MMK). a) Mosaic plot showing total skeletal preservation by score and institution with GLD individuals removed from MMK (n=2200). Preservation scores reflect the percentage of the total skeleton present: 1=75-100%, 2=25-50% and 3=0-25%. Institutional codes: ALB=Albany Museum, MMK=McGregor Museum, NMB=National Museum Bloemfontein, SAM=IZIKO South African Museum, UP=University of Pretoria, WITS=University of the Witwatersrand. The single individual from the University of Cape Town (UCT) has been removed. c) Table showing oddsratios with 95% confidence intervals. Confidence intervals that do not contain the null hypothesis are shown in bold.

SKELETAL PRESERVATION BY ELEMENT

Figure 7 presents the relative frequencies of skeletal elements, stratified by preservation score, ordered from highest to lowest frequency. All elements of the skull are present in higher frequencies than the postcranial elements. With the exception of the palatines, elements of the cranium show greater frequencies than the mandible. The bones of the neurocranium (frontal, parietals, occipital and temporals) are present in higher frequencies than those of the face (zygomatics, maxillae and palatines) with the exception of the sphenoids which are encountered more infrequently than the maxillae. Of the postcranial,

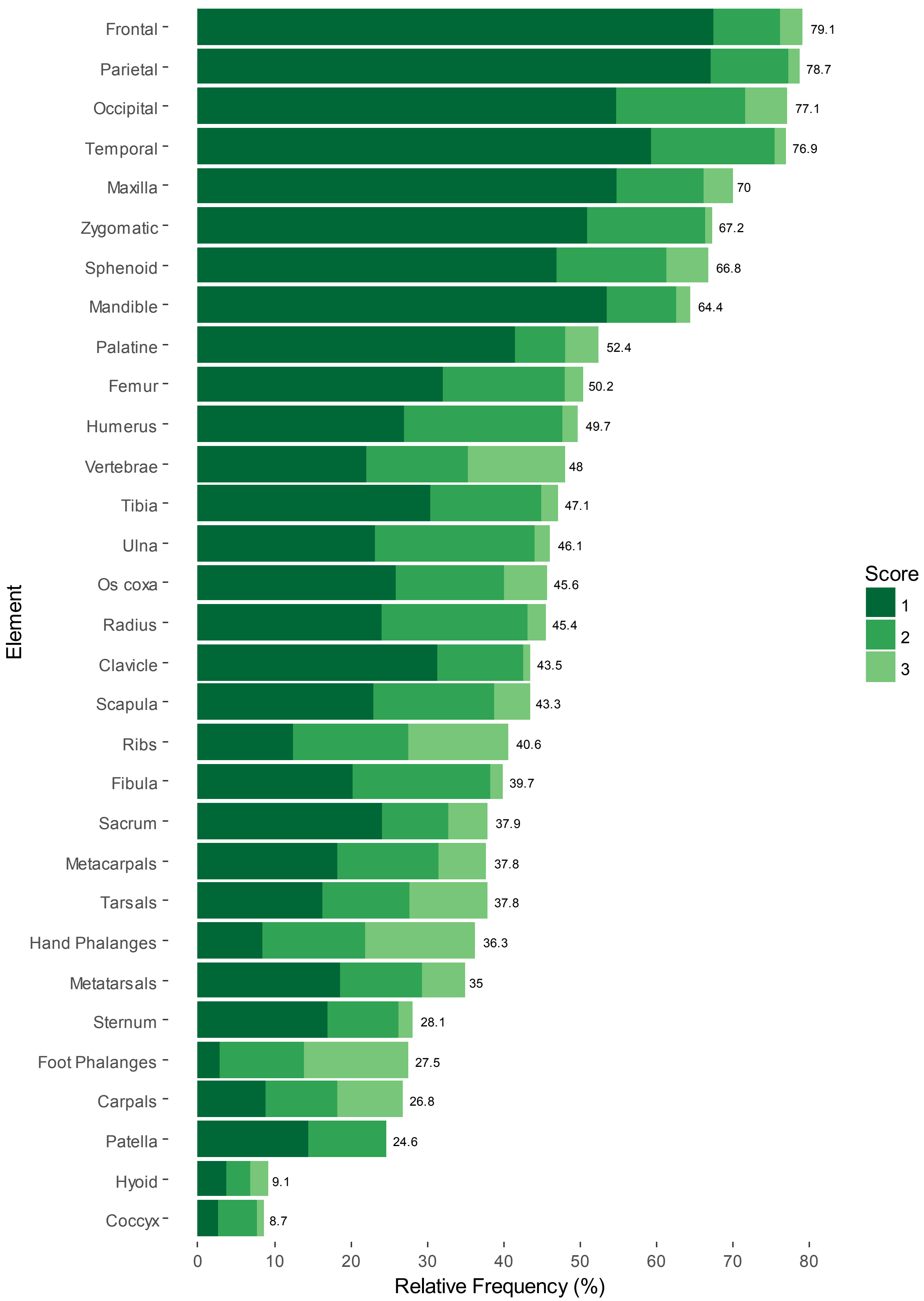


Figure 7: Relative frequency plots for all skeletal elements stratified by preservation score (n=2321). Preservation scores reflect the percentage of skeletal element or grouped elements present: 1=75-100%; 2= 25-50% and 3=0-25%. Grouped, sided and subdivided elements have been condensed into a single element category and scores are adjusted accordingly.

axial elements, the vertebrae have the highest recorded frequency, followed in decreasing order, by the ribs, sacrum, sternum and coccyx. For the appendicular skeleton the proximal elements of the limbs were better represented than the distal elements. Furthermore, the elements of the lower limbs are generally present at higher frequencies than their upper limb counterparts. The fibulae and the bones of the hands and feet are the exception to this pattern. The fibulae show the lowest frequency of the large appendicular long bones. For the hands and feet, while the tarsals and carpals follow the pattern described above, this pattern is reversed for the remaining element groups. The metacarpals are present at higher frequencies than the metatarsals. Similarly, the hand phalanges are present at a higher frequency than the foot phalanges. The skeletal elements with the lowest frequencies are the hyoid and coccyx.

The cranial elements, in addition to being present at the highest frequencies, are also generally better preserved with a greater frequency of well-preserved relative to moderate and poorly preserved elements. For the postcranial elements, as the total frequency decreases, in general, the quality of preservation also decreases with relatively fewer well-preserved and more moderate to poorly preserved elements recorded. The relationship between preservation score and element is statistically significant (χ^2 =6797.9, *df* = 60, n = 2321, *p* < .0001) and Pearson's residuals show the pattern of preservation described above (Figure 8). No poorly preserved elements (score 3) were recorded for the right patella but the expected cell count is greater than 5. Grouped elements (e.g. vertebrae, ribs, phalanges, etc.) also show poor preservation relative to ungrouped elements.

SKELETAL PRESERVATION BY SIDE AND SCORE

Figure 9 presents a barplot of skeletal elements stratified by side and score. Among sided elements, no clear pattern of left or right dominance emerges and differences between sides are typically small. Where elements have been classified as left, right or unsided, unsided frequencies are much lower than either of the sided frequencies. The hand and foot phalanges are the exception; here elements were typically not sided so the unsided

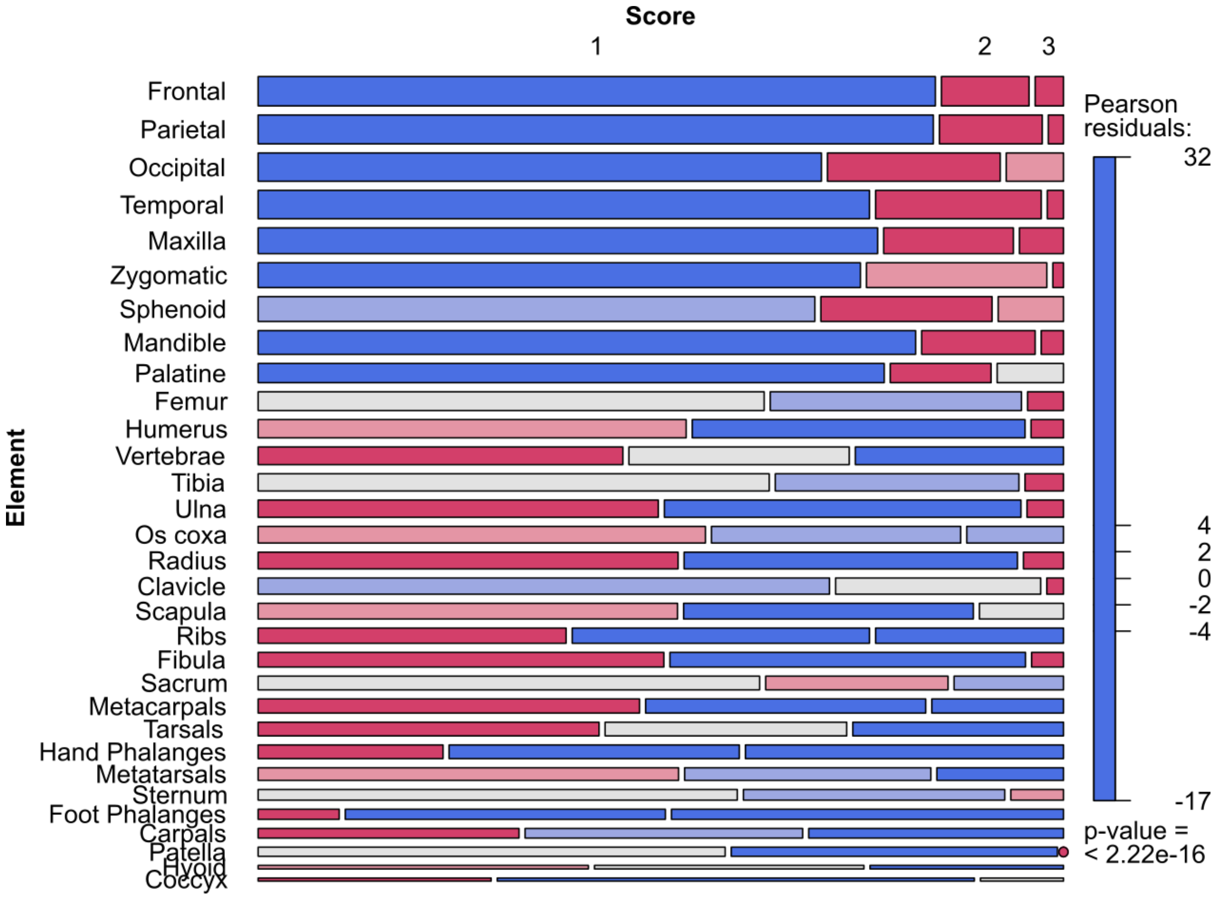


Figure 8: Mosiac plot of element by preservation score.

category has a much higher frequency than either of the sided categories. Log-linear modeling was used to investigate the association among the variables element (E), preservation score (P) and side (S). All models include only sided elements. Midline and unsided elements have been excluded as these create a large number of structural zeros. The best fitting model has main effects for element and preservation score and an interaction term for element and score (Model = EP, $G^2 = 40.99$, $df = 81$, $p = {\sim}1$) (Table 8). None of the residuals exceed the absolute value of 2 and the fitted values are close to the observed values (see Appendix, Table A 1). This indicates that there is no significant difference between left and right sides, that side is not significantly associated with element or score, but that these are related to each other (model parameter are presented in Appendix A (Table A 2).

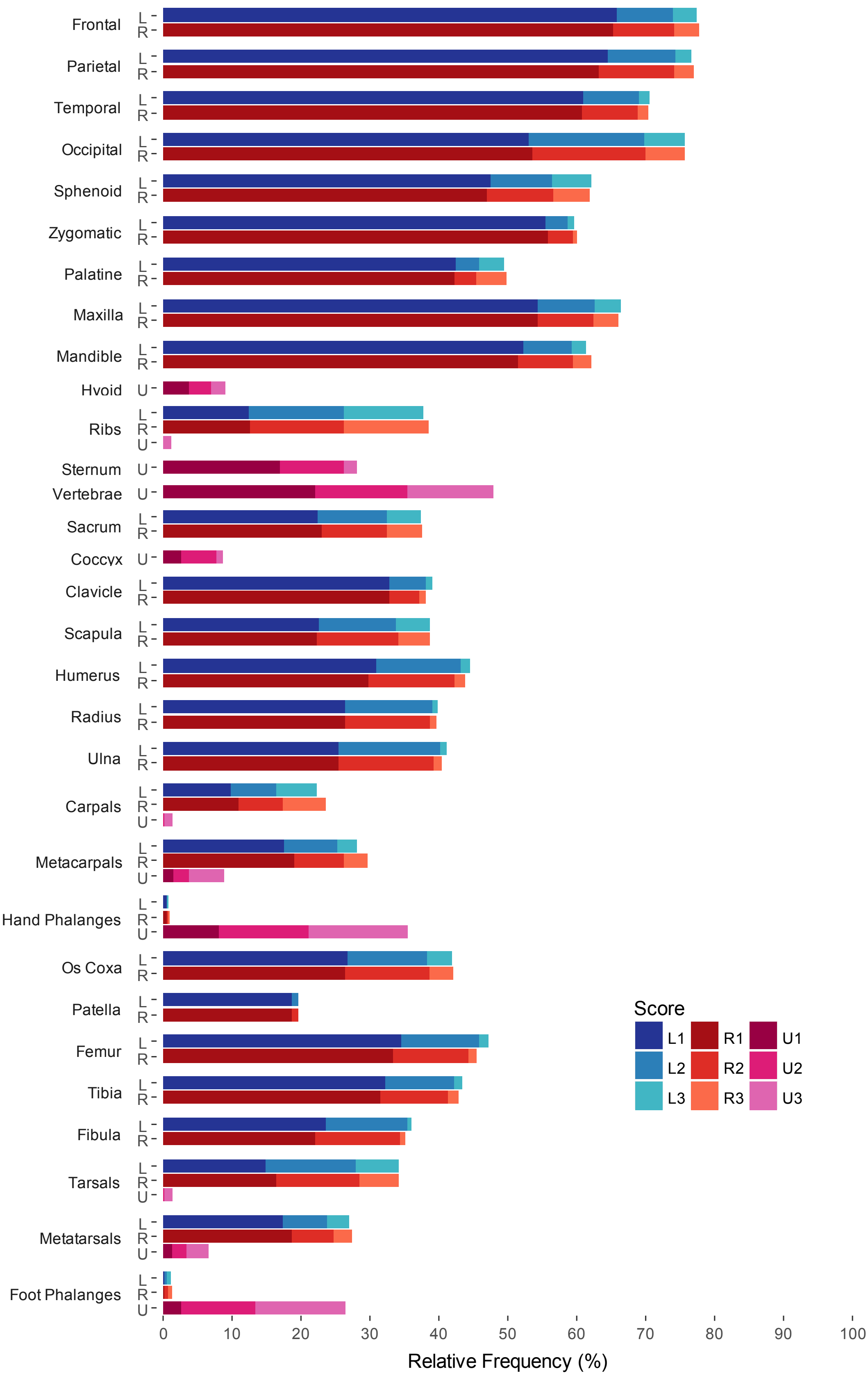


Figure 9: Relative frequency plot for skeletal elements stratified by preservation score and side (n=2321). Preservation scores reflect the percentage of skeletal element present: 1=75-100%; 2= 25-50% and 3=0-25%. Score legend prefixes and discrete axis labels L, R and U refer to left, right and unsided/midline elements, respectively.

Table 8: Log-linear modal fit statistics for variables element, preservation score and side

Model*	G^2	*df*	*p*-value	H_0	ΔG^2	Δdf	*p*-value	AIC	BIC
EPS	0	0	1.00000	NA	NA	NA	NA	1427.83	1928.02
EP,PS,ES	35.8	52	0.95771	$\lambda_{ijk}^{EPS}=0$	35.8	52	0.957711	1359.63	1699.27
EP,PS	40.44	78	0.99986	$\lambda_{ik}^{ES}=0$	4.64	26	0.999999	1312.27	1571.63
PS,ES	6119.39	104	0.00000	$\lambda_{ij}^{EP}=0$	6083.59	52	0.000000	7339.22	7518.3
EP,ES	36.01	54	0.97168	$\lambda_{jk}^{PS}=0$	0.21	2	0.900325	1355.84	1689.3
EP,S	40.99	80	0.99991	$\lambda_{jk}^{PS}=0$	0.55	2	0.759572	1308.82	1562
PS,E	6124.37	130	0.00000	$\lambda_{ik}^{ES}=0$	4.98	26	0.999998	7292.2	7391
ES,P	6119.94	106	0.00000	$\lambda_{ij}^{EP}=0$	6083.93	52	0.000000	7335.77	7508.67
EP	**40.99**	**81**	**0.99994**	$\boldsymbol{\lambda_{k}^{S}=0}$	**0**	**1**	**1.000000**	**1306.82**	**1556.91**
PS	20355.25	156	0.00000	$\lambda_{i}^{E}=0$	14230.88	26	0.000000	21471.07	21489.6
ES	46906.2	108	0.00000	$\lambda_{j}^{P}=0$	40786.26	2	0.000000	48118.02	48284.75
E,P,S	6124.92	132	0.00000	$\lambda_{ij}^{EP}=0$	6083.93	52	0.000000	7288.75	7381.37

*Variables: element (E), preservation score (P) and side (S).
Best fitting model is shown in bold font.

SUB-DIVIDED/GROUPED ELEMENTS

Relative frequency barplots for sub-divided/grouped temporal, scapular, sternal, rib, os coxal and vertebral elements are show in Figure 10 and frequency tables are presented in Appendix A (Table A 3). For sided elements there is no obvious pattern of left or right dominance and differences between left and right frequencies are minor. For the scapula, ribs and os coxa, in all cases the best fitting log-linear model was a non-comprehensive model with main effects for segment and preservation score and an interaction between preservation and segment only (EP) (Appendix, Table A 4). This indicates that there is no significant difference between left and right sides, no significant association between side and either element or preservation score and that the dimensionality of the data can be reduced to two variables, element and preservation score. All model fitted values and residuals are presented in Appendix A (Table A 5) and model coefficients are presented in Appendix A (Table A 6).

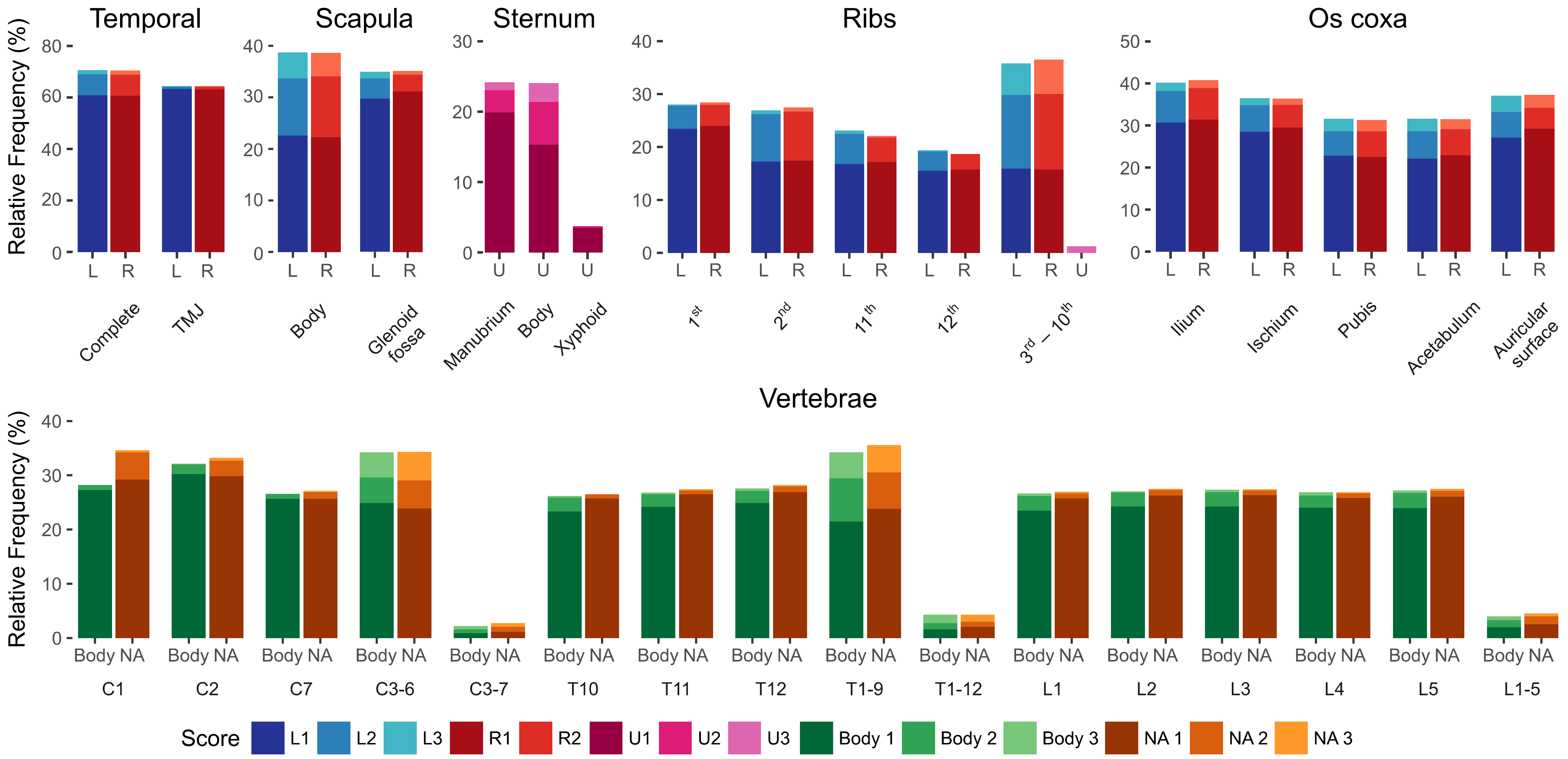


Figure 10: Relative frequency plots for the temporal bones, scapulae, sternum, ribs, os coxae and vertebrae stratified by preservation score and anatomical side, number and/or segment (n=2321). Scores represent the approximate percentage of skeletal element/segment present as follows: 1 = 75%-100%, 2 = 25%-75%, 3 = 0%-25%. Score legend prefixes and discrete axes labels: L=left; R=right; U=unsided/midline; body= vertebral body; and NA=neural arch. Vertebral discrete axis group category prefixes C, T and L refer to cervical, thoracic and lumbar vertebrae, respectively.

The temporo-mandibular joint (TMJ) shows relatively good preservation. Chi-square and log-linear statistics are not reported for the temporal because the assumption of independence is not met i.e. all individuals for which a TMJ score was recorded are also included in the complete category. It can, however, be noted that in most cases (91%) if the temporal bone is present the TMJ is likely to be preserved.

The log-linear model (EP) for the scapula fits relatively well ($G^2 = 8.65$, $df = 6$, $p = .19$) and the only standardized deviance residuals exceeding the absolute value of 2 are those of the glenoid fossa, score 3 (max = -2.4). The glenoid fossa shows better preservation than the scapular body but the body is observed in greater frequency. The odds of observing a moderate (score 2) compared to well (score 1) preserved body segment is 4.44 times that of the glenoid fossa and this is significant ($SE = 0.1$, $z = 15.11$, $p = <.0001$). The odds of observing a moderate compared to poorly preserved (score 3) scapular segment is 1.47 times greater for glenoid fossa than the scapular body and although the effect is not as great it is still significant ($SE = 0.19$, $z = -2.1$, $p = 0.04$).

Of the sternal elements, the manubrium is observed at the highest frequency (19.9%), followed in decreasing order by the sternal body (15.26%) and xyphoid (3.47%). A Chi-square goodness-of-fit test indicates these frequency differences are significant ($\chi^2 = 371.14$, $df = 2$, n = 2321, $p < .0001$), however, this difference is largely driven by the low xyphoid frequencies. The frequency difference between the manubrium and body are not significant when the xyphoid frequency is removed ($\chi^2 = 0.0009$, $df = 1$, n = 2321, $p < .98$). The association between sternal element and preservation score is significant ($\chi^2 = 73.44$, $df = 4$, n = 2321, $p < .0001$) and all residuals are greater than |2| but less than |4|. In general, the manubrium and xyphoid are well preserved, when present, relative to the sternal body. The odds of observing a well compared to moderately preserved manubrium is 2.51 times greater than the sternal body (95% CI [1.83, 3.44]), while that of the xyphoid is 5.88 times greater than the sternal body (95% CI [2.42, 14.24]). The odds of observing a moderate compared to poorly preserved manubrium are close to one and the confidence interval contains the null (θ=1.27, 95% CI [0.74, 2.18]), the effect between the body and xyphoid is much larger but this too is not significant (θ=0.21, 95% CI [0.01, 3.78]).

The highest rib frequencies are observed for the grouped 3rd-10th ribs (36.13%). The 1st ribs are the most frequently observed of the individual ribs (28.22%), followed in decreasing order by the 2nd (27.16%), 11th (22.58%) and 12th (19.07%) ribs. The relative frequencies reported above were determined by averaging left and right sides. Unsided ribs were not included in the analyses because the frequency is low (1.17%) and their inclusion results in a large number of structural zeros. The best fitting model (EP) for the ribs was one including the main effects and an interaction term for element and preservation score ($G^2 = 12.72$, $df = 15$, $p = .62$). The residuals are all less than |2|. The 1st and 12th ribs have the best relative preservation. The odds of observing a well preserved 1st rib is 1.18 times that of the 12th rib but this difference is not significant (θ = 1/0.84 = 1.18, $SE = 0.12$, $z = -1.43$, $p = .15$). The 11th (θ = 0.59, $SE = 0.11$, $z = -4.97$, $p = <.0001$) and 2nd (θ = 0.34, $SE = 0.1$, $z = -11.03$, $p = <.0001$) ribs have significantly fewer well relative to moderately preserved individuals when compared to the 1st rib and the 3rd – 10th ribs (θ = 0.2, $SE = 0.1$, $z = -17.06$, $p = <.0001$) have the worst relative preservation. The differences between frequencies of score 2 and 3 (moderate and poor preservation) are not significant (see Appendix, Table A 6) with the exception of the 3rd to 10th ribs. The odds of observing a poor relative to moderately preserved 3rd-10th rib set are 6.01 times greater than that of the 1st rib ($SE = 0.29$, $z = 6.28$, $p = <.0001$).

The ilium is the most frequently observed os coxal segment (40.46%), followed, in decreasing order by the ischium (36.32%) and pubis (31.45%). Of the joint surfaces the auricular surface (37.21%) is more frequently observed than the acetabulum (31.64%). The auricular surface and acetabulum were not included in the log-linear modelling since doing so would violate the assumption of independence. The best fitting model in this case, as with all the others, is the one that does not include a main effect or any interaction term for the variable side. This model (EP) fits well ($G^2 = 3.02$, $df = 9$, $p = .96$) and residuals are all below |2| (Appendix, Table A 5). The pubis, in addition to being the most infrequently observed segment is also the least well preserved. The odds of observing a well relative to moderately preserved ischium is 1.34 times that of the pubis and 1.1 times greater for the ilium. While the difference between the pubis and ischium is significant (*SE* = 0.1, *z* = 3.11, *p* = .002), the difference with the ilium is not (*SE* = 0.09, *z* = 1.01, *p* = .31). Similarly, the odds of observing a poor relative to moderately preserved pubis is 1.75 times that of the ischium (*SE* = 0.17, *z* = -3.23, *p* = .001) and 1.78 times that of the ilium (*SE* = 0.16, *z* = -3.62, *p* = .0003) and in both instances the differences are significant.

The highest frequencies of vertebral elements were observed for the grouped T1-9, C3-6 and individual elements C1 and C2. Conversely, the lowest frequencies were observed for the grouped juvenile vertebrae C1-7, T1-12 and L1-5 (Figure 10, Appendix, Table A 3). Neural arches are present in greater frequencies than their vertebral body counterparts for all elements except L4 (body: n = 621, NA: n = 620). For the individual thoracic and lumbar vertebrae the neural arches are also better preserved with greater frequencies of well relative to moderate and poorly preserved individuals. This trend does not extend to the cervical vertebra for which body elements are typically better preserved and the grouped vertebrae which have much higher frequencies of moderate and poorly preserved elements. Log-linear analysis supports the significance of these patterns. The saturated log-linear model (EPS) had the best fit indicating a significant 3-way interaction between element, preservation score and segment (Appendix, Table A 4). This indicates that the pattern of interaction between element and segment varies for different levels of preservation score. When using L5, score 2, and vertebral body as reference baselines, significant 3-way interactions are observed for all cervical and all grouped vertebral elements with the exception of T1-12 (Appendix, Table A 7). Unsurprisingly significant 2-way interactions with score and segment are also observed for these elements. The association between segment and score is also significant. Observed frequencies for score 3 are low and sparseness may result in spurious significance for 3-way interactions (Zelterman, 2002), furthermore, there are no significant 2-way interactions between score 3 and segment and no significant 3-way interactions involving score 3. To determine if this might be the reason for significant 3-way associations, the model was refitted collapsing scores 2 and 3 into a single category; however, significant 3-way interactions for cervical and grouped vertebrae were still observed.

The relative frequencies of the humerus, radius, ulna, femur, tibia and fibula, stratified by side, score and segment are presented in Figure 11 (Appendix, Table A 8). All six elements show similar frequency patterns. The proximal, middle and distal diaphyseal segments are all observed at greater total frequencies than the proximal and distal epiphyses. In addition to greater total frequencies, the diaphyseal segments are also generally better preserved with greater frequencies of well, relative to moderate and poorly, preserved segments. Observed total frequencies for the proximal and distal epiphyses are similar for most elements with the exception of the ulna and fibula. The proximal ulna epiphysis is observed in greater frequency than the distal epiphysis, while the converse is true for the fibula. With the exception of the femur, all distal epiphyses are better preserved than proximal epiphyses with greater frequencies of well, relative to moderate and poorly, preserved segments. To investigate the association among variables, log-linear models were explored for each element. In all cases, the non-comprehensive model, with main effects and an interaction between element and preservation score, was identified as the best fitting model (Appendix, Table A 9). This implies that there are no significant differences between left and right sides and no significant interactions between side and preservation score or element as was the case with the other sided elements. Thus, contingency tables were collapsed over side. Most of the log-linear models indicate there is no significant difference between element and preservation scores 2 and 3. The femur and tibia are the exceptions, where both epiphyses show significant interactions when comparing scores 2 and 3. Given the low frequencies of score 3 for all segments and the lack of significant association for most, score 2 and 3 were combined into a single category and the results of these models are presented below. Table 9 presents the loglinear model parameters and p-values. The odds ratios for segment and score comparisons were obtained by exponentiating the parameter estimates (the middle epiphyseal segment and score 1 are used as references). The odds ratios for each element show a similar ordering of preservation. Middle epiphyseal elements show the best relative preservation (i.e. have a greater frequency of well, relative to moderate and poorly, preserved segments), this is followed by the proximal or distal epiphyseal segments, while the epiphyses show the worst relative preservation. The odds ratios indicate effects are relative small among the diaphyseal segments; however, these are

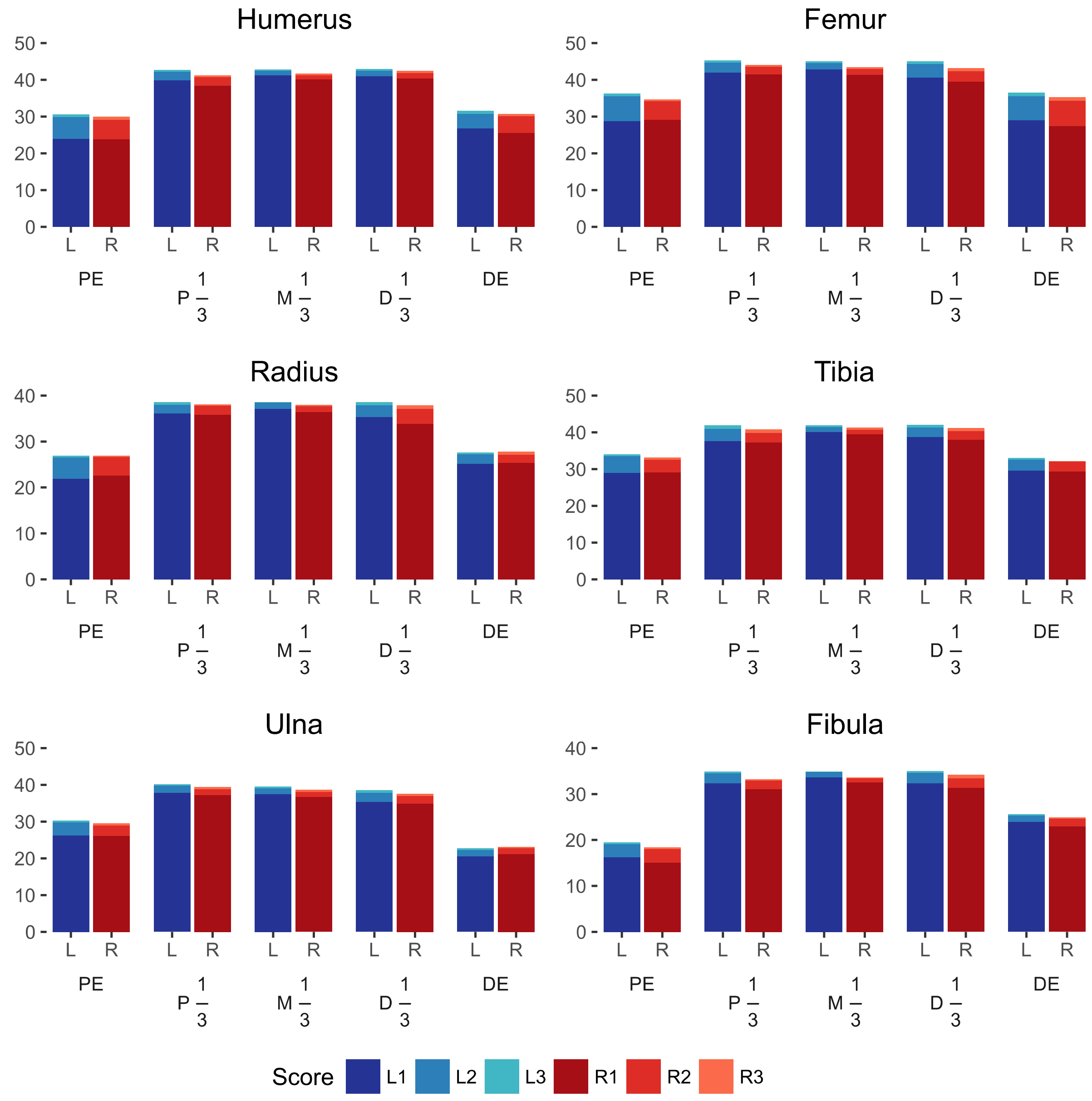


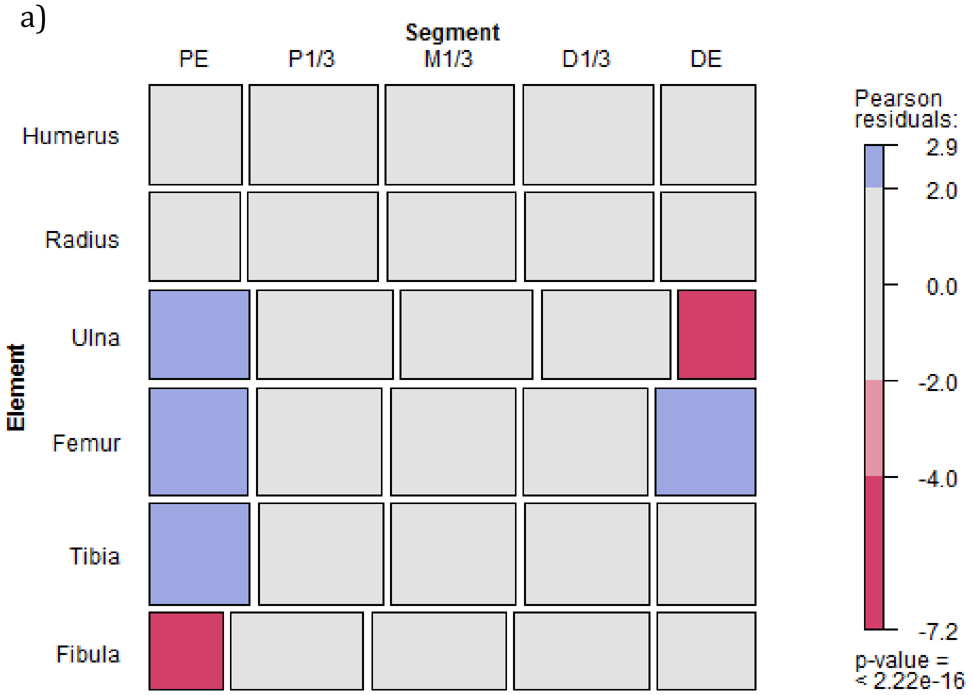
Figure 11: Relative frequency plots of the humerus, radius, ulna, femur, tibia and fibula stratified by side and anatomical segment (n=2321). The discrete axes labels and score prefixes, L and R, refer to the left and right skeletal elements respectively. Relative frequencies for each element are divided into the following five segments: proximal epiphysis (PE), proximal 1/3rd of the diaphysis (P1/3), middle 1/3rd of the diaphysis (M1/3), distal 1/3rd of the diaphysis (D1/3) and distal epiphysis (DE). Preservation scores reflect the percentage of the skeletal segment present: 1=75-100%; 2= 25-50% and 3=0-25%.

significant in most cases (the exceptions are the humerus D⅓ and ulna P⅓). The effects are much larger for the epiphyses and, with the exception of the femur, distal epiphyses are better preserved than proximal epiphyses. While the pattern of total preservation appears similar among all six elements, a Chi-square test of homogeneity indicates that differences are significant ($\chi^2 = 150.49$, *df* = 20, n = 2321, *p* < .0001). However, an examination of the residuals (Figure 12a) and odds ratios (Figure 12b) reveal this difference is driven by differences among the epiphyses only and effects are relatively small. Residuals for the proximal epiphyses of the ulna, femur and tibia, as well as the distal epiphysis of the femur are all greater than expected under the model of independence (i.e. > 2), while frequencies for the proximal epiphysis of the fibula and distal epiphysis of the ulna are both less than expected (i.e. < 4). Odds ratios are all relatively close to one. The only comparisons for

Table 9: Loglinear (EP) parameter estimates, standard error, Wald z-statistic and p-values for humerus, radius, ulna, femur, tibia and fibula. Parameter estimates were obtained from six separate log-linear models, one for each element. These have been combined into a single table for convenience. Reference/baselines: Segment = M⅓, Score = 1. The Odds ratios are obtained by exponentiating the parameter estimates. Score 2 and 3 have been combined into a single category.

Element	Interactions	Estimate	Odds ratio	*SE*	*z*-value	*p*-value
Humerus	Element-PE:Score-2+3	1.92	6.81	0.136	14.09	**<0.0001**
	Segment-P⅓:Score-2+3	0.6	1.82	0.15	3.98	**<0.0001**
	Segment-D⅓:Score-2+3	0.22	1.24	0.16	1.36	0.17379
	Segment-DE:Score-2+3	1.57	4.83	0.14	11.29	**<0.0001**
Radius	Segment-PE:Score-2+3	1.6	4.98	0.142	11.33	**<0.0001**
	Segment-P⅓:Score-2+3	0.39	1.47	0.157	2.47	**0.01355**
	Segment-D⅓:Score-2+3	0.86	2.37	0.146	5.92	**<0.0001**
	Segment-DE:Score-2+3	0.81	2.26	0.156	5.23	**<0.0001**
Ulna	Segment-PE:Score-2+3	0.96	2.61	0.136	7.07	**<0.0001**
	Segment-P⅓:Score-2+3	0.09	1.1	0.148	0.63	0.53005
	Segment-D⅓:Score-2+3	0.44	1.56	0.14	3.15	**0.00161**
	Segment-DE:Score-2+3	0.6	1.81	0.153	3.9	**0.0001**
Femur	Segment-PE:Score-2+3	1.51	4.53	0.123	12.33	**<0.0001**
	Segment-P⅓:Score-2+3	0.34	1.41	0.138	2.49	**0.01282**
	Segment-D⅓:Score-2+3	0.69	2	0.13	5.33	**<0.0001**
	Segment-DE:Score-2+3	1.71	5.52	0.12	14.18	**<0.0001**
Tibia	Segment-PE:Score-2+3	1.23	3.41	0.134	9.14	**<0.0001**
	Segment-P⅓:Score-2+3	0.82	2.28	0.136	6.04	**<0.0001**
	Segment-D⅓:Score-2+3	0.58	1.79	0.141	4.12	**<0.0001**
	Segment-DE:Score-2+3	0.81	2.25	0.143	5.68	**<0.0001**
Fibula	Segment-PE:Score-2+3	1.79	5.97	0.168	10.66	**<0.0001**
	Segment-P⅓:Score-2+3	0.75	2.11	0.173	4.31	**<0.0001**
	Segment-D⅓:Score-2+3	0.9	2.46	0.169	5.31	**<0.0001**
	Segment-DE:Score-2+3	0.8	2.23	0.182	4.41	**<0.0001**

a)



b) **Odds ratios (95%**

	Element				
Segment	Humerus:Radius	Humerus:Ulna	Humerus:Femur	Humerus:Tibia	Humerus:Fibula
PE:M⅓	1.019	0.943	**0.895**	**0.889**	**1.302**
	(0.923-1.127)	(0.855-1.04)	**(0.814-0.983)**	**(0.807-0.978)**	**(1.169-1.45)**
P⅓:M⅓	0.994	0.978	0.983	1	1.002
	(0.908-1.089)	(0.893-1.07)	(0.901-1.073)	(0.915-1.093)	(0.912-1.101)
M⅓:D⅓	0.984	0.962	0.987	0.987	0.996
	(0.898-1.078)	(0.879-1.053)	(0.904-1.077)	(0.903-1.079)	(0.907-1.094)
M⅓:DE	0.983	**0.796**	**1.103**	1.062	1
	(0.891-1.086)	**(0.719-0.882)**	**(1.004-1.212)**	(0.965-1.169)	(0.904-1.108)

Figure 12: Mosaic plot and odds ratios comparing segments of the humerus, radius, ulna, femur, tibia and fibula. a) Mosaic plot of long bone elements stratified by segment with shaded Pearson residuals. b) Odds ratios for long bone element and segment. Odds ratios with 95% confidence intervals that do not contain the null hypothesis are shown in bold font. PE = proximal epiphysis, P⅓ = diaphyseal proximal ⅓, M⅓ = diaphyseal middle ⅓, D⅓ = diaphyseal distal ⅓, DE = distal epiphysis.

which the 95% confidence intervals do not contain the null hypothesis are the proximal epiphysis of the tibia and fibula, the distal epiphysis of the ulna and both femoral epiphyses, when the humerus and diaphyseal middle ⅓ are used as references. Even in these cases confidence intervals are small and the lower/upper limit is very close to one, with a possible exception of the ulna and fibula.

Figure 13 shows the relative frequencies of adult individuals stratified by sex. For 13.2% of the individuals present, sex could not be determined and a further 14.1% were classified as ambiguous sex. A greater number of males (total = 40.9%, male = 20.11%, probably male = 20.79%) are reported than females (total = 31.8%, female = 15.11%, probably female = 16.69%) (Appendix, Table A 10).

Figure 14a presents the relative frequencies of preservation stratified by sex and preservation score. In this case, the score represents the combined percentage preservation for the skull and os coxae only. A score of zero indicates the skull and os coxae were not preserved. In general, and not surprisingly, better relative preservation (i.e. greater frequencies of score 1 relative to 2 and score 2 relative to 3) coincides with greater confidence in classification of sex. This pattern is statistically significant ($\chi^2 = 621.48$, $df = 10$, n = 1707, $p < .0001$) and is supported by an examination of the residuals (Figure 14b). For

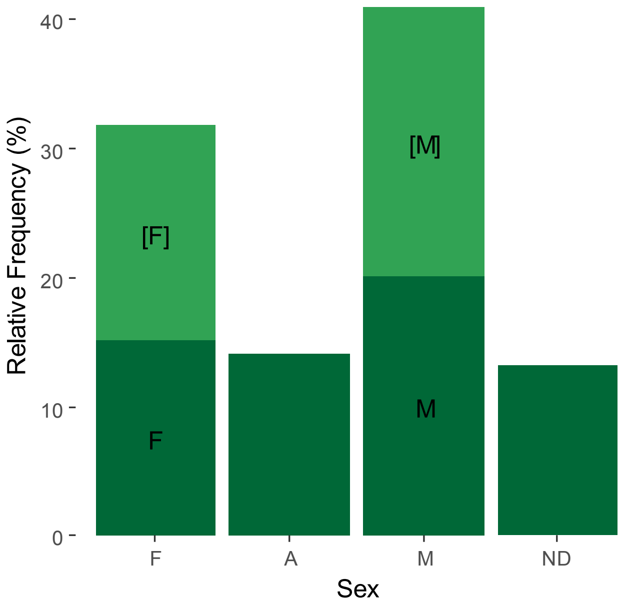
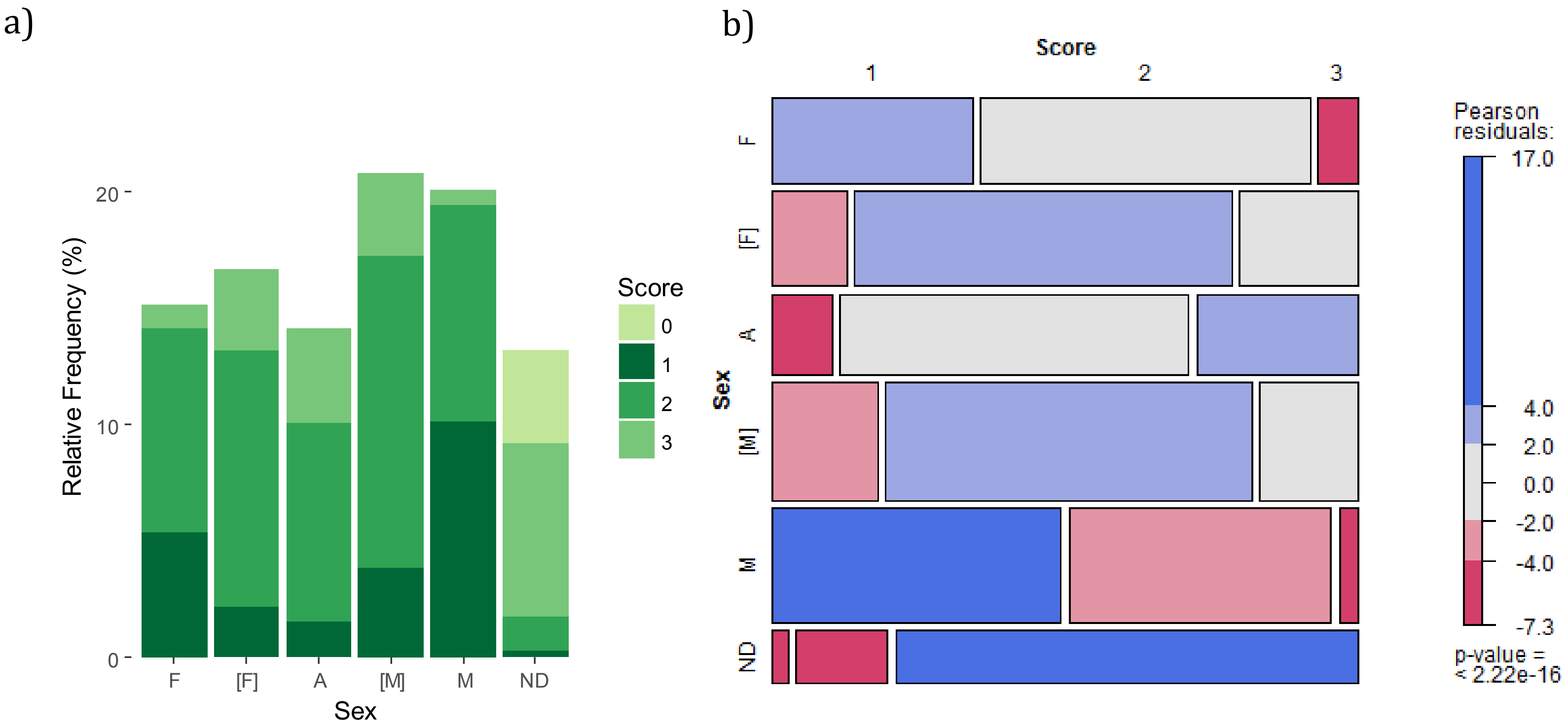


Figure 13: Relative frequency for sex with combined male and female sub-categories (n=1780). Sex: F = female, [F] = probably female, A = ambiguous, [M] = probably male, M = male, ND = not determined.



c)

	Score	
Sex	1:2	2:3
F:[F]	**3.11 (2.03-4.77)**	**2.68 (1.52-4.72)**
F:A	**3.49 (2.15-5.65)**	**4.08 (2.32-7.16)**
F:[M]	**2.14 (1.48-3.09)**	**2.28 (1.3-4)**
F:M	**0.56 (0.4-0.78)**	0.63 (0.29-1.35)
F:ND	**3.22 (1.2-8.67)**	**44.05 (23.13-83.88)**

Figure 14: Relative frequency plot, mosaic plot and odds ratios comparing associations between sex and score. a) Relative frequency of sex stratified by score (n = 1779). Here score represents the combined preservation scores for the skull and pelvis only. b) Mosaic plot of sex and preservation score with shaded Pearson residuals. All individuals with scores = 0 were removed for this analysis (n = 1707). c) Odds

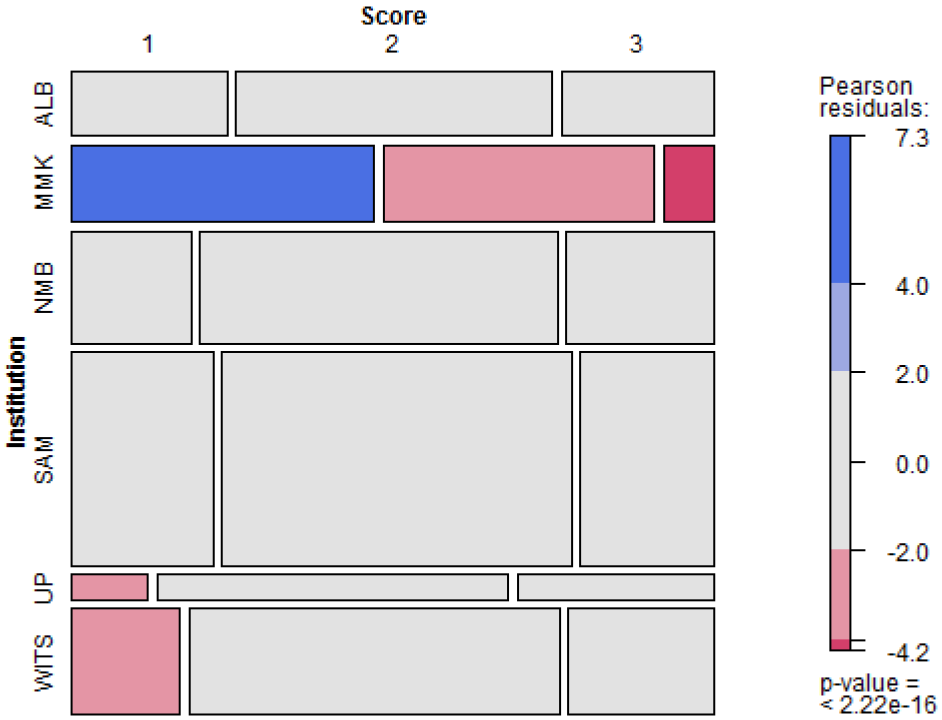
ratios examining associations between sex and score using female and score as references (n = 1707). Confidence intervals (95%) that do not contain the null hypothesis are shown in bold.

the male (M) and female (F) categories, large residuals (>2) are present for score 1, indicating a greater than expected frequency of well preserved elements and small residuals (<-2) are present for scores 3 indicating fewer poorly preserved elements than expected. The inverse of this pattern is observed for the remaining categories of sex. The odds ratios show moderate to large effects and suggest a ordering of preservation from best to worst as follows: male, female, probably male, probably female, ambiguous and not determined (Figure 14c).

Unsurprisingly, the association between preservation (skull and os coxae) and institution is similar to the pattern observed and discussed above and, as such, will not be discussed in detail here. A residual shaded mosaic plot and table of odds ratios are presented in Figure 15 and show that effects are moderate, there no significant differences between scores 2 and 3 and that MMK has the best relative preservation, while WITS has a lower frequency of well-preserved individuals than expected.

While for the sample as a whole there is a greater frequency of males, this pattern is not consistent across all institutions (Figure 16a). For ALB, MMK, NMB and SAM, a greater number of individuals were classified as male; however, for both UP and WITS, a greater frequency of females is reported. The McGregor museum has the greatest frequency of males relative to females. This extreme pattern is largely due to the Gladstone Cemetery individuals (Figure 16b). When the Gladstone individuals are excluded, the pattern of sex is more similar to that of the other institutions. A Chi-square test of independence shows that there is a significant relationship between sex and institution (χ^2 = 156.23, *df* = 25, n = 1707, *p* < .0001). Shaded residual mosaic plots and odds ratios are presented in Figure 17 a and b, respectively. An analysis of the residuals indicates there are fewer males at ALB relative to other institutions. MMK has a greater relative frequency of males, with fewer unclassified/not determined individuals and fewer individuals classified as probably female. The odds of observing a male relative to female at MMK is 5.12 times (95% CI [2.66,9.85]) that at ALB. NMB also has significantly more males relative to females then ALB (θ=2.68,

a)



b) **Odds ratios**

	Score	
Institution	1:2	2:3
ALB:MMK	**0.44 (0.28-0.69)**	**0.39 (0.21-0.72)**
ALB:NMB	1.48 (0.94-2.32)	0.86 (0.55-1.34)
ALB:SAM	1.22 (0.81-1.82)	0.79 (0.53-1.19)
ALB:UP	2.2 (0.98-4.91)	1.03 (0.54-1.96)
ALB:WITS	**1.61 (1.01-2.57)**	0.8 (0.51-1.26)

Figure 15: Mosaic plot and odds ratios comparing associations between institution and score (n = 1707). a) Mosaic plot of institution and preservation score with shaded Pearson residuals. b) Odds ratios examining associations between institution and score using ALB and score as references. Confidence intervals (95%) that do not contain the null hypothesis are shown in bold. Score refers to preservation of the skull and pelvis only. All individuals with score of zero (indicating the skull and pelvis were not preserved) were removed for these analyses. Institution: ALB = Albany Museum, MMK = McGregor Museum, NMB = National Museum, Bloemfontein, SAM = IZIKO South African Museum, UP = University of Pretoria and WITS = University of the Witwatersrand.

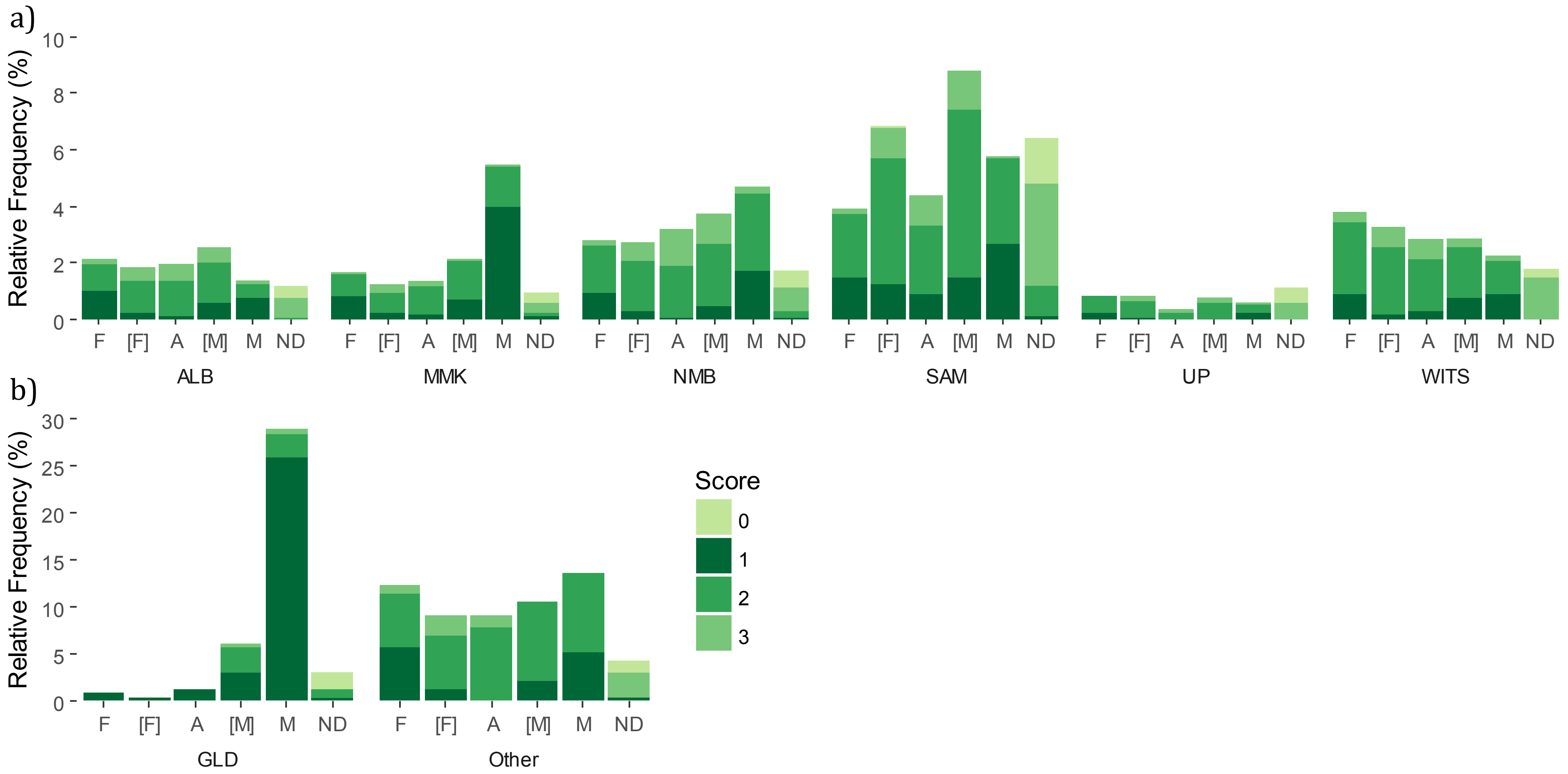
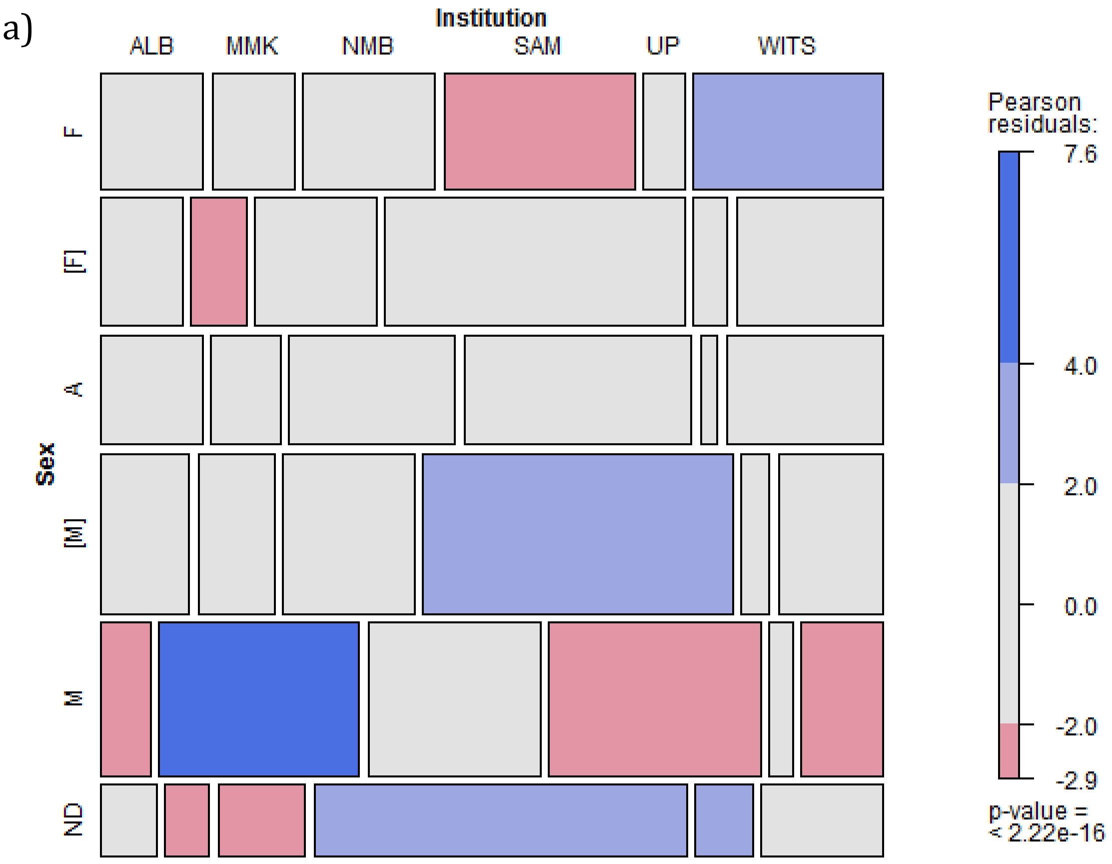


Figure 16 Relative frequency plots for sex. a) Relative frequency plot for sex stratified by score and institution (n = 1779). b) Relative frequency of sex stratified by score and group for the McGregor Museum (MMK) (n = 228). The plot compares frequencies from all individuals unearthed from the Gladstone Cemetery (GLD) to all other MMK individuals. Sex: F = female, [F] = probably female, A = ambiguous, [M] = probably male, M = male, ND = not determined. Score (refers to preservation of the skull and os coxae only): 0 = Elements not present, 1 = 75%-100% of elements present, 2 = 25%-50% of elements present, 3 = <25% elements present. Institution: ALB = Albany Museum, MMK = McGregor Museum, NMB = National Museum, Bloemfontein, SAM = IZIKO South African Museum, UP = University of Pretoria and WITS = University of the Witwatersrand.

a)



b) **Odds ratios (95% CI)**

	Institution				
Sex	ALB:MMK	ALB:NMB	ALB:SAM	ALB:UP	ALB:WITS
F:[F]	0.84 (0.41-1.74)	1.15 (0.62-2.12)	**1.97 (1.14-3.43)**	1.07 (0.45-2.55)	1 (0.56-1.79)
F:A	0.87 (0.43-1.76)	1.26 (0.7-2.29)	1.21 (0.69-2.12)	0.43 (0.15-1.24)	0.83 (0.46-1.49)
F:[M]	1.07 (0.56-2.04)	1.14 (0.64-2.01)	**1.88 (1.12-3.15)**	0.79 (0.34-1.84)	0.64 (0.37-1.13)
F:M	**5.12 (2.66-9.85)**	**2.68 (1.44-4.99)**	**2.33 (1.29-4.22)**	1.06 (0.41-2.73)	0.95 (0.5-1.8)
F:ND	0.97 (0.38-2.53)	1.19 (0.53-2.7)	**3.55 (1.75-7.18)**	1.95 (0.7-5.39)	1.13 (0.52-2.46)

Figure 17 Mosaic plot and odds ratios comparing associations between sex and institution (n = 1707). a) Mosaic plot of sex and institution with shaded Pearson residuals. b) Odds ratios examining associations between sex and institution using female and ALB as references. Confidence intervals (95%) that do not contain the null hypothesis are shown in bold. Sex: F = female, [F] = probably female, A = ambiguous, [M] = probably male, M = male, ND = not determined. Institution: ALB = Albany Museum, MMK = McGregor Museum, NMB = National Museum, Bloemfontein, SAM = IZIKO South African Museum, UP = University of Pretoria and WITS = University of the Witwatersrand.

95% CI [1.44, 4.99]). SAM has fewer females and males than expected and a greater relative frequency of unclassified individuals and those classified as probably male. All 95% confidence intervals for SAM-ALB odds ratios do not contain one, with the exception of the female and ambiguous comparison. While UP and WITS have greater frequencies of females compared to males (when “certain” and “probable” categories are combined), this does not appear to be statistically significant.

AGE-AT-DEATH ESTIMATION

All but two individuals could be placed into the binary age categories: mature and immature (n=2319). In total, 29.8% (n=691) were categorized as immature and 70.2% (n=1628) as mature. All individuals for which epiphyses were not completely fused, including the medial clavicle, were classified as immature. In this regard, 40 individuals were recorded as immature with partially fused or unfused medial clavicular epiphyses when all other epiphyses were fused. Table 10 shows the frequency of individuals placed into different broad age categories. For approximately half (49.46%) of the individuals, age estimates could not be refined further than the broad category of adult (>20 yrs). Of the adult categories, the 2nd largest frequency is observed for the middle adult (35-49 yrs) category (12.85%). This is followed in decreasing order by the young adult (20-34 yrs) category (4.27%), and the old adult (>50 yrs) category (2.33%). Among immature individuals, the categories with the greatest frequencies are the 10-34 yrs (5.43%) and 10-19 yrs (5%). All other juvenile categories have frequencies below 5%.

Figure 18 presents the relative frequency of age stratified by score. As with the sample as a whole, preservation of the age stratified sample is generally poor. The adolescent and adult age categories show better relative preservation with greater frequencies of well preserved, relative to moderate and poorly preserved, individuals. Conversely, the juvenile categories show relatively poor preservation. Due to low observed frequencies, the number of age categories was reduced by pooling the birth-9 yrs, 2-9 yrs and birth-19 yrs into a single category. The 6-9 yrs and 6-19 yrs were also combined into a single category. This selection aimed to reduce the number of categories with low observed frequencies, while maximizing

Table 10: Frequencies (relative frequencies %) by broad age category (n=2319).

	Category Max						
Category Min	1 yr	5 yrs	9 yrs	19 yrs	34 yrs	49 yrs	>50 yrs
Prenatal	49 (2.11)	9 (0.39)	0	0	0	0	0
Birth	47 (2.03)	50 (2.16)	12 (0.52)	28 (1.21)	0	0	0
2 yrs	0	56 (2.41)	65 (2.8)	28 (1.21)	0	0	0
6 yrs	0	0	10 (0.43)	69 (2.98)	0	0	0
10 yrs	0	0	0	116 (5)	126 (5.43)	0	0
20 yrs	0	0	0	0	99 (4.27)	35 (1.51)	1147 (49.46)
35 yrs	0	0	0	0	0	298 (12.85)	21 (0.91)
50 yrs	0	0	0	0	0	0	54 (2.33)

the number of remaining categories. A Chi-square test of independence shows a significant relationship between preservation score and age (χ^2 = 1174.9, *df* = 28, n = 2319, *p* < .0001). In one instances, the expected cell frequency was <5 but this is less than 20%. An examination of the Pearson residuals supports the pattern described above (Figure 19a), i.e. all age categories greater than 10-19 yrs/10-34 yrs have residuals exceeding 4, with the exception of the >20 yrs category which has lower frequencies of scores 1 and 2 than expected (residuals <-4). The juvenile categories of prenatal-1 yr, birth-1 yr and 2-5 yrs have greater frequencies of moderate preservation. Among juveniles, the odds ratios show differences between adjacent age categories are not significant when comparing preservation scores 1 and 2, with the exception of the prenatal-1 yr and birth-1 yr categories (Figure 19b). However, there are significant differences when comparing the odds between scores 2 and 3. The effects are moderate to large; however, large odds ratios are all associated with wide confidence intervals. The 10-34 yrs category shows poor preservation relative to the 10-19 yrs and 20-34 yrs categories. Finally, of the adult age categories the only significant difference occurs for comparisons involving the >20 yrs age group which shows poor relative preservation. These results should be interpreted with caution, since Chi-square approximations can be poor when both small and large observed frequencies are present for sparse tables (Haberman, 1988) as is the case in this instance.

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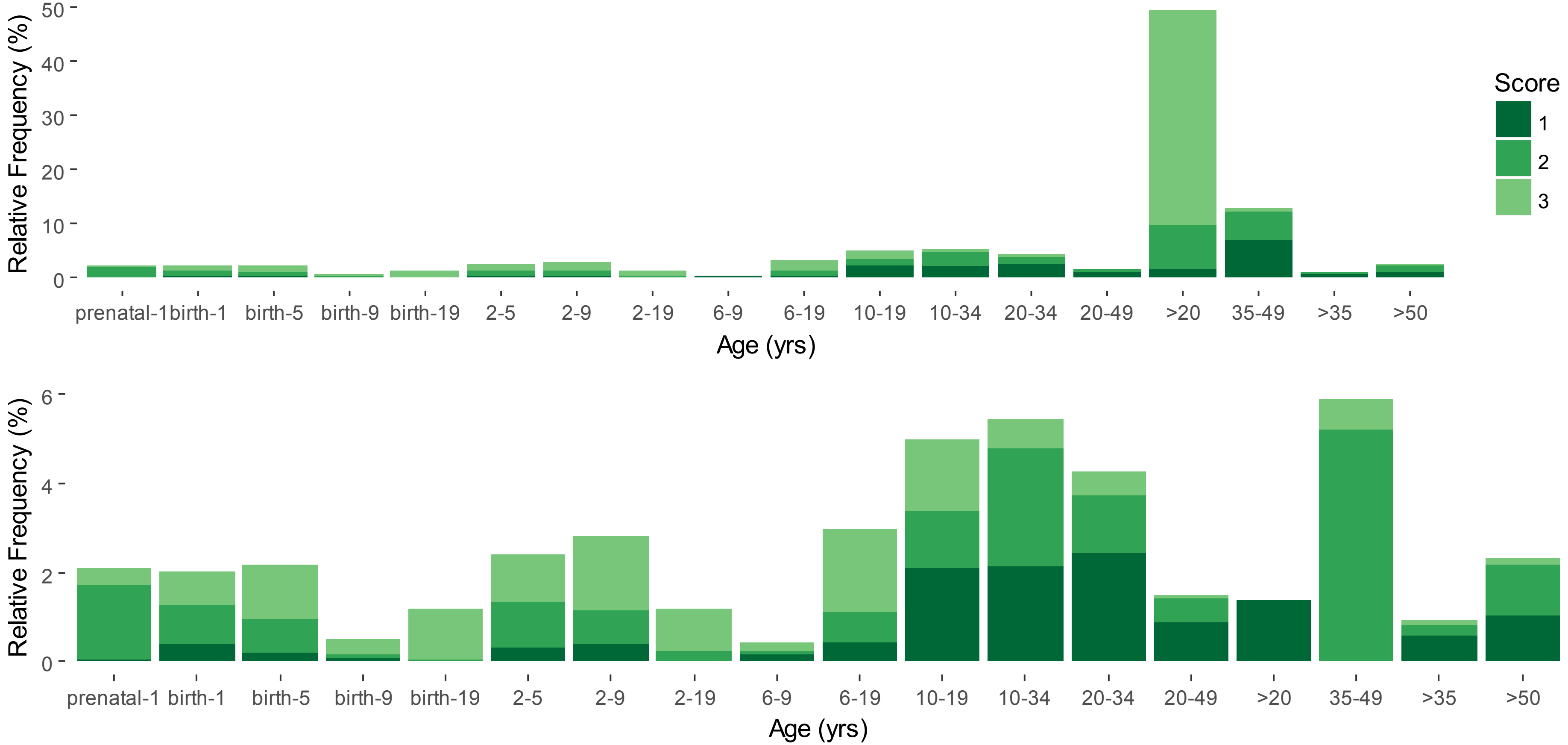
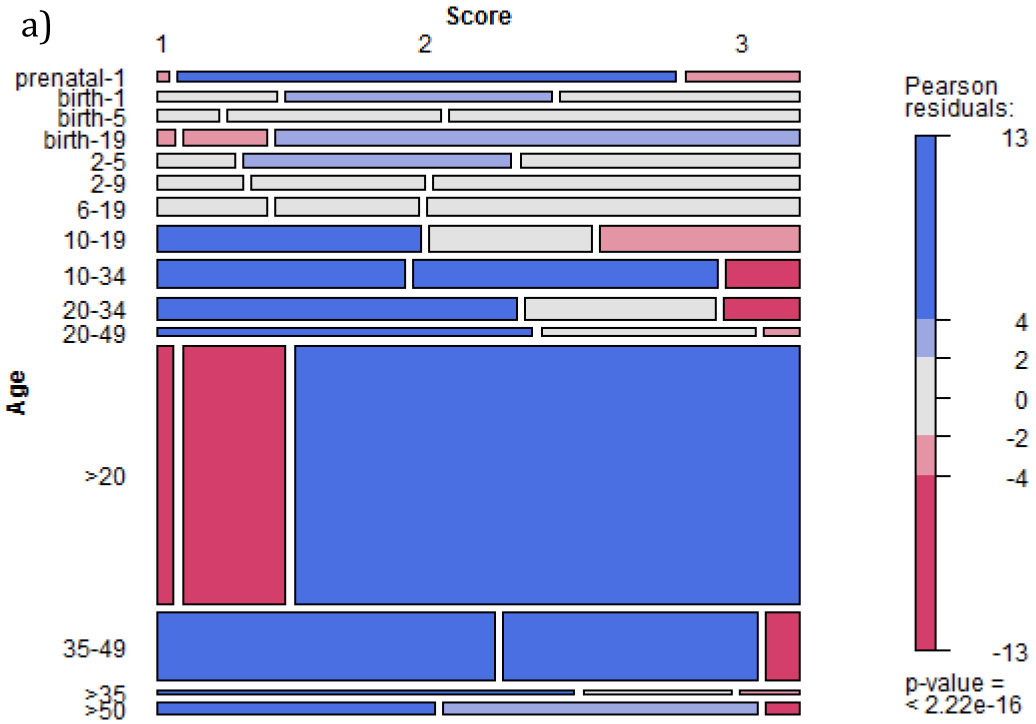


Figure 18: Relative frequency plots for age stratified by preservation score (n = 2319). The upper image shows all the data. Due to the disproportionately large values of the >20 yrs and 35-49 yrs categories, the remaining categories are difficult to evaluate. The lower image is identical to the upper image with an adjusted scale to aid visualization. Preservation scores reflect the total preservation (all elements are included): 1=75-100%; 2= 25-50% and 3=0-25%.

a)



b) **Odds ratios (95% CI)**

Age	Score 1:2	Score 2:3
prenatal-1:birth-1	**0.06 (0.01-0.48)**	**3.9 (1.49-10.23)**
birth-1:birth-5	1.53 (0.43-5.45)	1.83 (0.76-4.4)
birth-5:birth-19	1.32 (0.21-8.23)	**3.85 (1.52-9.71)**
birth-19:2-5	0.76 (0.13-4.38)	**0.16 (0.07-0.4)**
2-5:2-9	0.58 (0.18-1.86)	2.03 (0.92-4.48)
2-9:6-19	0.64 (0.22-1.86)	1.24 (0.57-2.7)
6-19:10-19	0.48 (0.21-1.1)	**0.47 (0.23-0.98)**
10-19:10-34	**1.99 (1.11-3.59)**	**0.2 (0.09-0.42)**
10-34:20-34	**0.43 (0.24-0.77)**	1.63 (0.68-3.91)
20-34:20-49	1.09 (0.47-2.5)	0.42 (0.08-2.15)
20-49:>20	**10.34 (4.63-23.05)**	**29.4 (6.53-132.42)**
>20:35-49	**0.13 (0.08-0.2)**	**0.03 (0.02-0.05)**
35-49:>35	0.48 (0.17-1.36)	3.03 (0.54-16.91)
>35:>50	3.15 (0.99-10.05)	0.28 (0.04-2.11)

Figure 19: Mosaic plot and odds ratio comparing age and score (n = 2319). a) Mosaic plot with shaded Pearson residuals. b) Table of odds ratios and 95% confidence intervals comparing adjacent categories. Odds ratios and confidence intervals that do not contain the null hypothesis are shown in bold. Preservation scores represent total skeletal preservation (all elements included): 1 = 75-100%, 2 = 25-75% and 3 = 0-25%.

This problem extends to the odds ratios as well, as is indicated by the very wide confidence intervals for some of the comparisons

The pattern of age category frequencies is relatively similar among institutions (Figure 20). The pattern of juvenile frequencies varies by institution. The pattern of adult frequencies among institutions is similar to the overall pattern described above, i.e. >20 yrs has the highest frequencies followed in descending order by the 35-49 yrs, 20-34 yrs and >50 yrs categories. Only two institutions, SAM and UP, differ. SAM has a greater frequency of individuals in the >50 yrs category, while UP has no individuals in the 20-34 yrs category. The relatively large number of categories resulted in a sparse contingency table, so data were pooled once again in order to combat this problem. All juvenile categories <19 yrs, with the exception of the 10-34 yrs category, were combined into a single category; thus variations among juvenile categories were not assessed. The adult age categories of 20-49 yrs, >35 yrs and >20 yrs were also combined into a single category. A Chi-square test of independence indicated there is a significant relationship between age and institution (χ^2 = 152.04, *df* = 25, n = 2319, *p* < .0001). Two expected values are below 5 but this is less than 20%. An examination of the residuals indicates ALB has a greater number of juveniles than expected (Figure 21a). MMK has a greater frequency of adolescents, young adults and middle adults than expected, with fewer old and unclassified adults. SAM has a greater frequency of old adults and lower frequency of individuals in the 10-34 yrs category than expected. Finally, WITS has fewer old adults than expected. Odds ratios indicate effects are minor to moderate (Figure 21b). The largest odds ratios also have the widest confidence intervals. When using the juvenile category, birth-19 yrs, and ALB as references, the only significant comparisons are with MMK (10-34 yrs, 20-34 yrs and 35-49 yrs), SAM (>50 yrs) and the >20 yrs category for NMB, SAM and WITS.

A comparison of age stratified by sex reveals a greater frequency of males for most age categories (Figure 22). The 10-34 yrs and >50 yrs age categories have a greater frequency of females. The frequency of ambiguous sex is relatively low for all categories except the 10-34 yrs, while not determined sex is low for all categories except the >20 yrs category. To

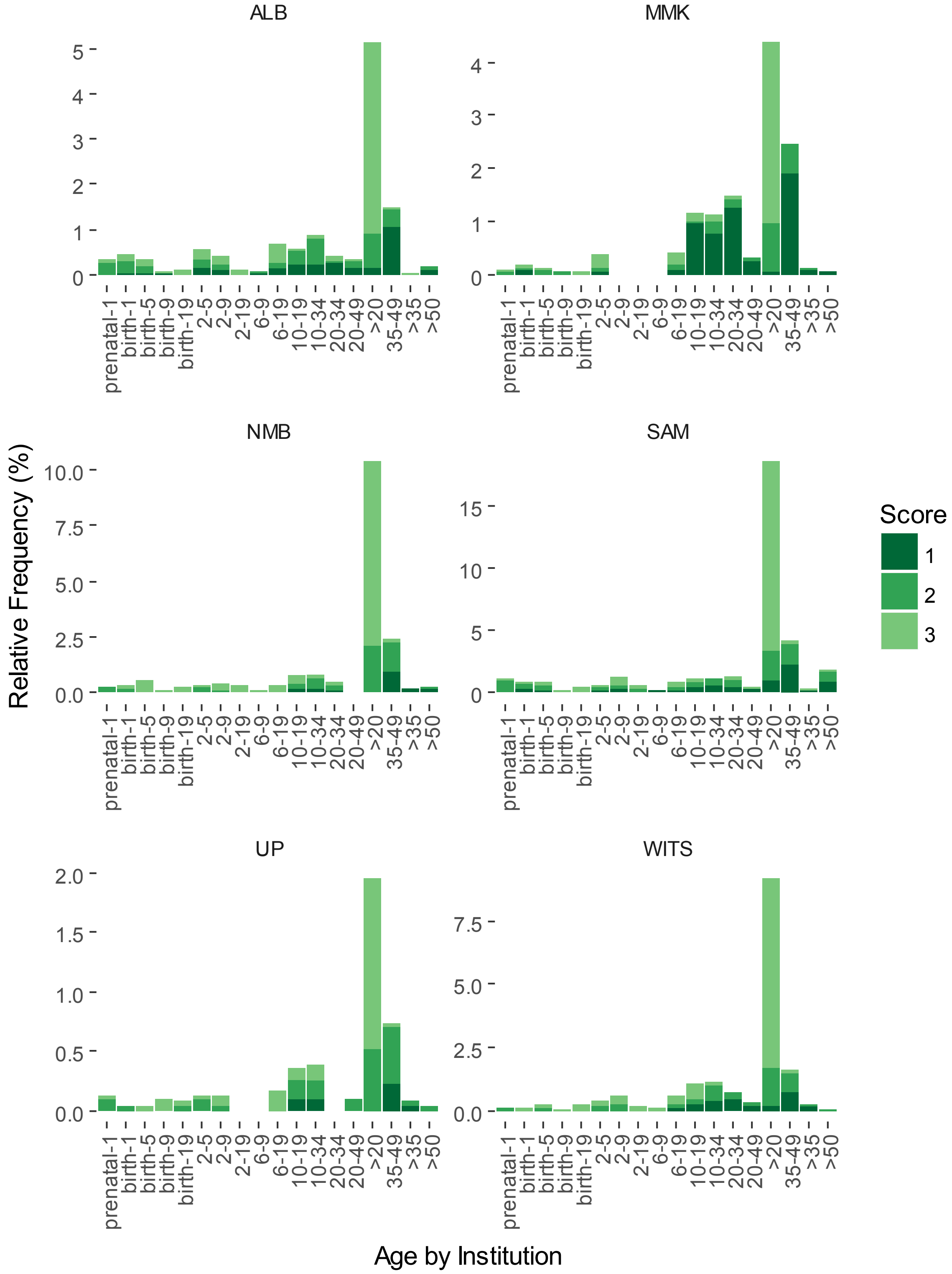
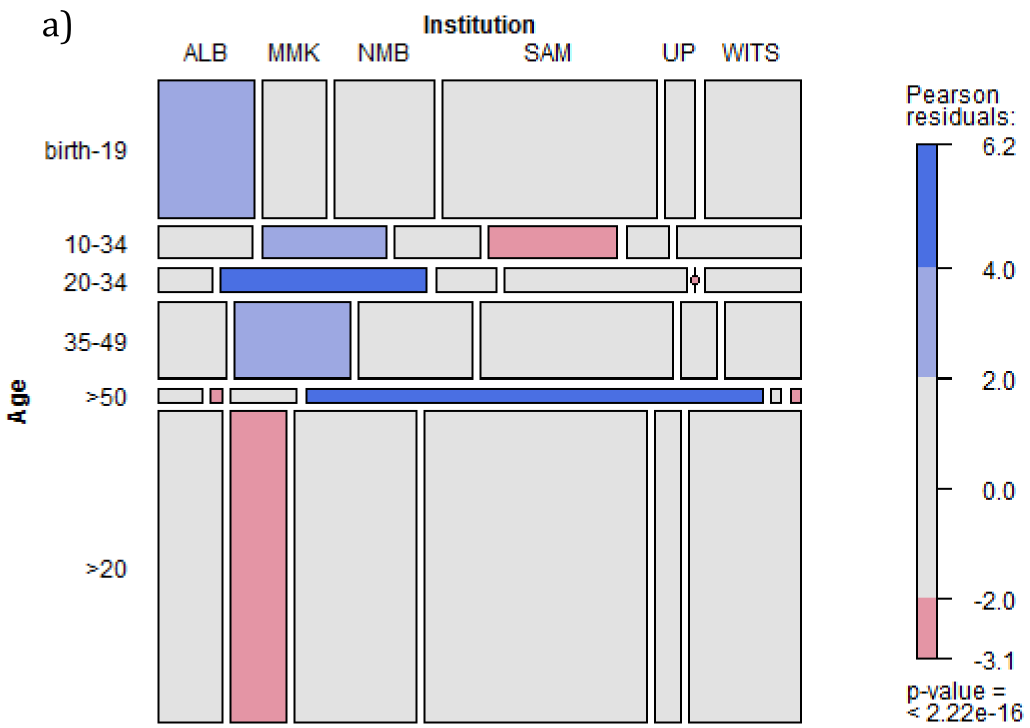


Figure 20: Relative frequency plot of age stratified by institution and score (n = 2319). Preservation score reflects the total skeletal preservation (all elements included): 1 = 75%-100% of elements present, 2 = 25%-50% of elements present, 3 = 0-25% elements present. Institution: ALB = Albany Museum, MMK = McGregor Museum, NMB = National Museum, Bloemfontein, SAM = IZIKO South African Museum, UP = University of Pretoria and WITS = University of the Witwatersrand. The single individual from UCT is not included.



b) **Odds ratios (95% CI)**

	Institution				
Age	ALB:MMK	ALB:NMB	ALB:SAM	ALB:UP	ALB:WITS
birth-19:10-34	**1.96** **(1-3.81)**	0.87 (0.44-1.75)	0.61 (0.33-1.14)	1.44 (0.6-3.48)	1.28 (0.67-2.45)
birth-19:20-34	**5.5** **(2.49-12.14)**	1.07 (0.42-2.7)	1.46 (0.67-3.15)	0.16 (0.01-2.9)	1.72 (0.73-4.03)
birth-19:35-49	**2.52** **(1.47-4.33)**	1.58 (0.94-2.65)	1.26 (0.79-2)	1.58 (0.77-3.23)	1.1 (0.64-1.91)
birth-19:>50	0.5 (0.08-3.3)	1.4 (0.4-4.81)	**4.18** **(1.53-11.44)**	1.04 (0.16-6.91)	0.33 (0.05-2.14)
birth-19:>20	1.31 (0.86-2)	**1.85** **(1.28-2.66)**	**1.57** **(1.14-2.17)**	1.2 (0.7-2.06)	**1.74** **(1.2-2.52)**

Figure 21: Mosaic plot and odds ratios for age stratified by institution. a) Mosaic plot with shaded Pearson residuals. b) Odds ratios and 95% confidence intervals. Confidence intervals that do not contain the null are shown in bold. Age categories have been pooled to reduce sparseness. Sex: F = female, [F] = probably female, A = ambiguous, [M] = probably male, M = male, ND = not determined. Institution: ALB = Albany Museum, MMK = McGregor Museum, NMB = National Museum, Bloemfontein, SAM = IZIKO South African Museum, UP = University of Pretoria and WITS = University of the Witwatersrand. The single individual from UCT is not included.

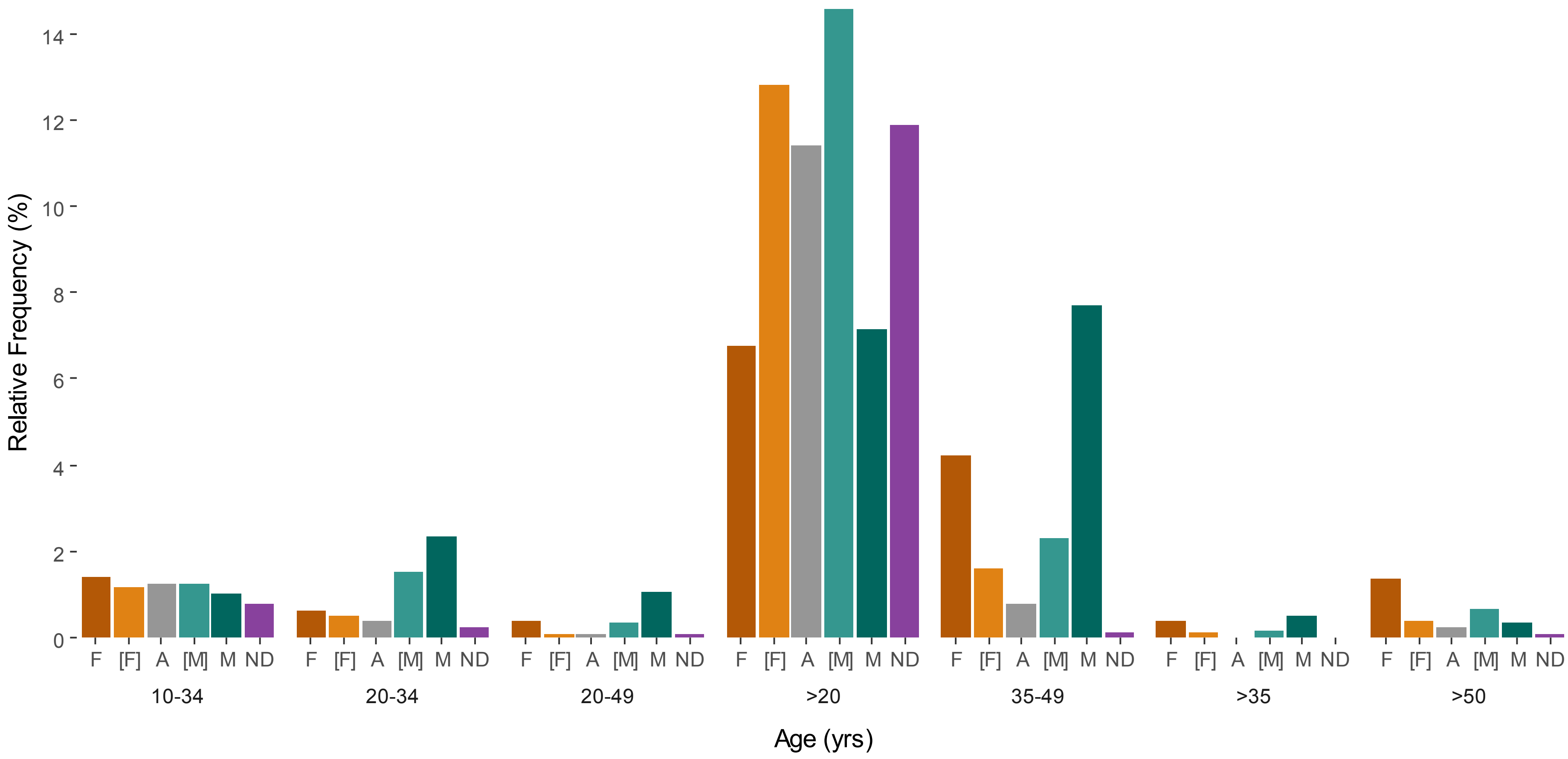
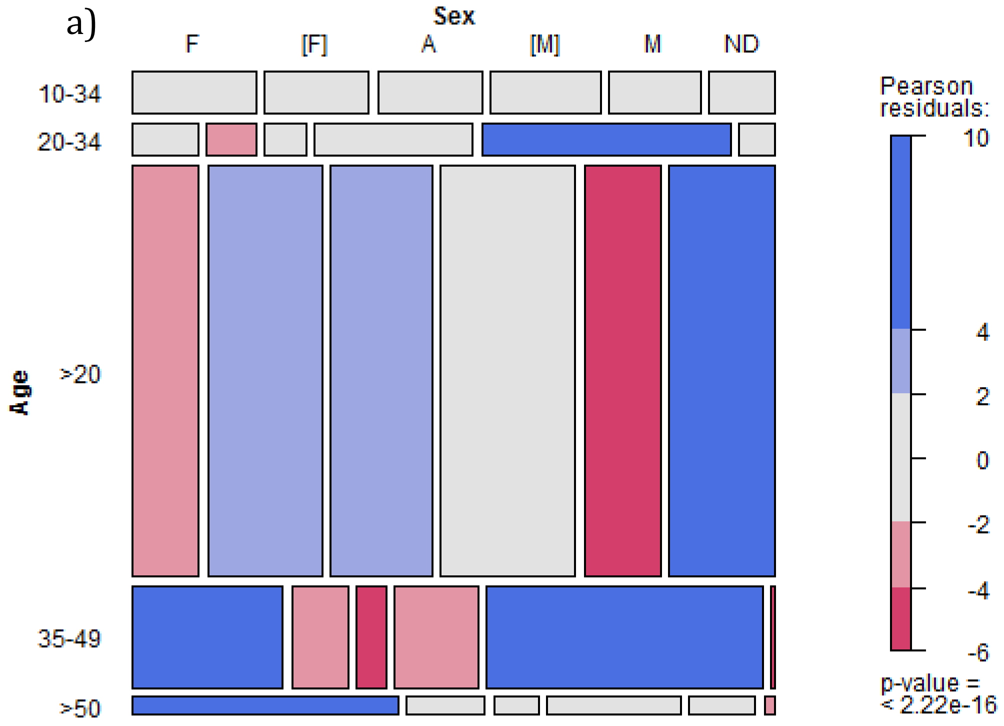


Figure 22: Relative frequency plot of age stratified by sex (n= 1778). Sex: F = female, [F] = probably female, A = ambiguous, [M] = probably male, M = male, ND = not determined

a)



b) **Odds ratios (95% CI)**

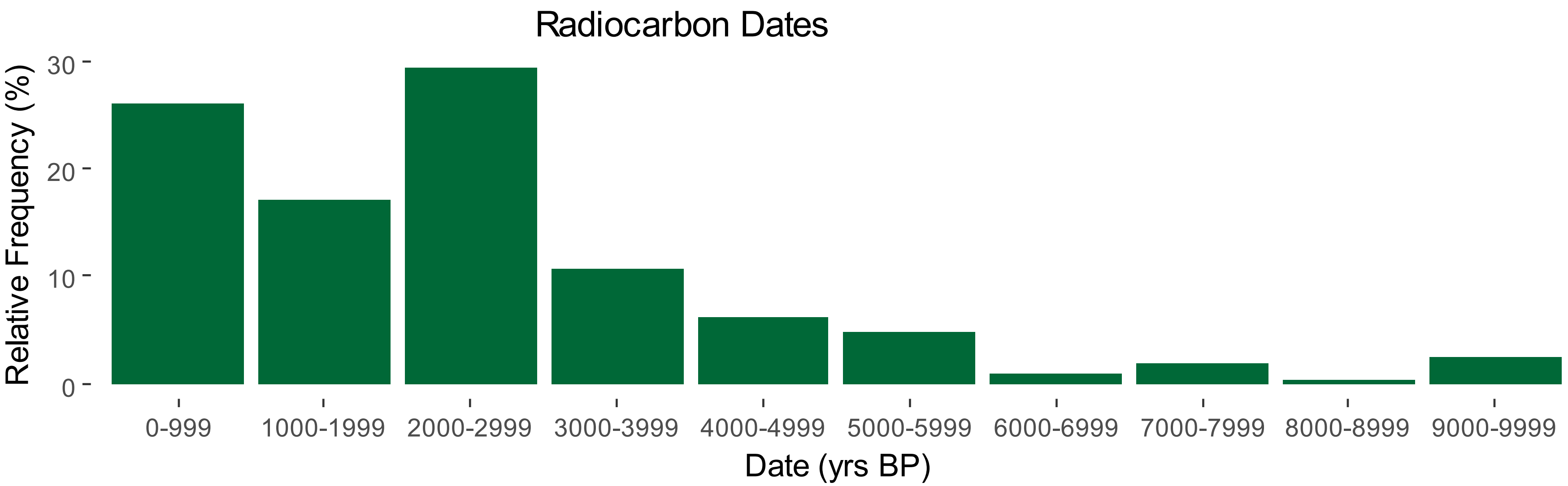
	Age			
Sex	10-34:20-34	10-34:>20	10-34:35-49	10-34:>50
F:[F]	0.97 (0.34-2.8)	**2.06 (1.11-3.82)**	**0.44 (0.22-0.92)**	**0.35 (0.12-0.97)**
F:A	0.72 (0.24-2.19)	1.73 (0.94-3.19)	**0.21 (0.09-0.48)**	**0.19 (0.06-0.63)**
F:[M]	**2.79 (1.13-6.9)**	**2.27 (1.24-4.18)**	0.62 (0.31-1.24)	0.57 (0.23-1.4)
F:M	**5.3 (2.16-13.03)**	1.61 (0.84-3.07)	**2.54 (1.3-4.95)**	0.35 (0.12-1.02)
F:ND	0.65 (0.17-2.43)	**2.83 (1.42-5.63)**	**0.05 (0.01-0.22)**	**0.07 (0.01-0.61)**

Figure 23: Mosaic plot and odds ratios for age stratified by sex. a) Mosaic plot with shaded Pearson residuals. b) Odds ratios and 95% confidence intervals. Confidence intervals that do not contain the null are shown in bold. Age categories have been pooled to reduce sparseness. Sex: F = female, [F] = probably female, A = ambiguous, [M] = probably male, M = male, ND = not determined.

resolve the issue of sparseness, the 20-49 yrs, >35 yrs and >20 yrs categories were pooled. A Chi-square test of independence reveals a significant association between sex and age (χ^2 = 352.02, *df* = 20, n = 1778, *p* < .0001). An examination of the Chi-square Pearson residuals reveals a greater frequency of males for 20-34 yrs age category than expected (Figure 23a). The >20 yrs category has fewer female and males than expected with a greater frequency of individuals in the probably female, ambiguous and not determined categories. The 35-49 yrs age category has a greater frequency of females and males with fewer individuals in the remaining categories than expected. Finally, the >50 yrs category has a greater number of females than expected. The odds ratios indicate that the greater frequency of females relative to males is significant for the 10-34 yrs age category (Figure 23b). While these results are not presented, if the >50 yrs category is used as a reference then there are significantly more females than males for this category too; however, there is no significant difference between the 10-34 yrs and >50 yrs categories.

CHRONOLOGICAL DATE

Chronological dates were available for 615 individuals (26.5%) based on relative or absolute methods. Thus, approximately ¼ of the archaeologically derived South African human remains have an associated date. Uncalibrated radiocarbon dates were available for 310 individuals (13.36%), while for the remaining 305 individuals (13.14%), dates were based on relative methods. The frequency of individuals for which relative dates can be obtained is probably underestimated here. An examination of historical records and associated cultural material may yield additional information; however, cultural materials are not typically stored with human remains and this was beyond the scope of this investigation. The relative frequency plots for absolute and relative dates are presented in Figure 24. Most of the radiocarbon dates fall between 0 and 3000 yrs BP. Thereafter, there is a steady decline in the frequency of individuals as the estimated age increases, with very low frequencies after 6000 yrs BP. The relative dates show the greatest frequency for categories between AD 1800 and AD 2000, while frequencies in all categories prior to AD 1800 are very low. The high frequencies post-1800 are large due to excavations from historic informal cemeteries.



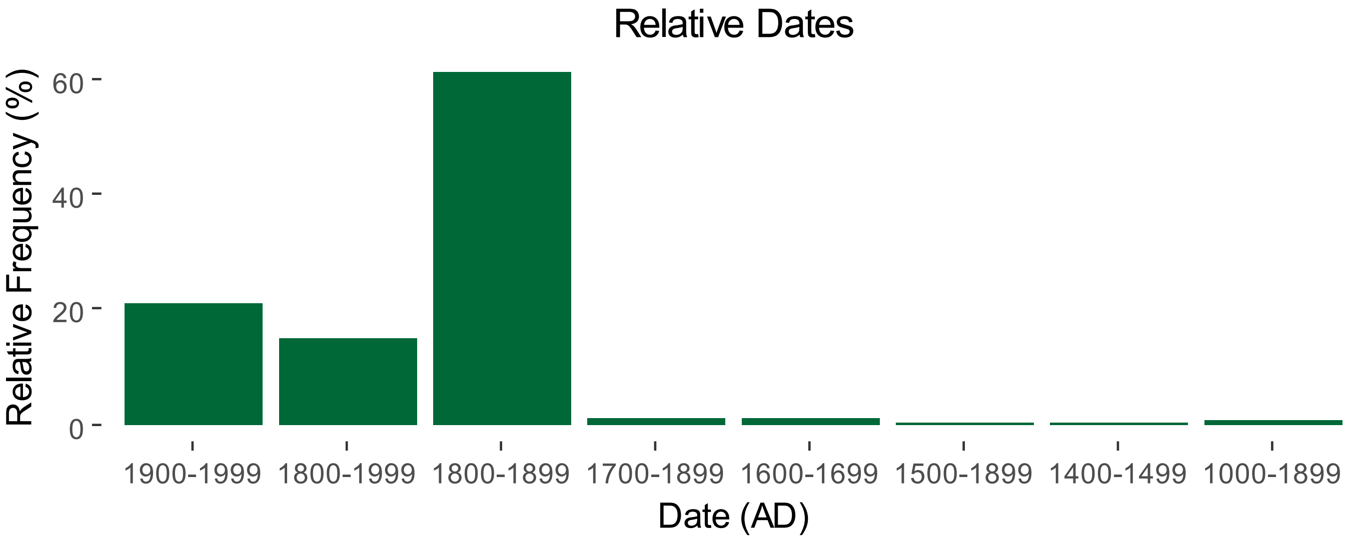


Figure 24: Relative Frequency plots for dated individuals using absolute methods (upper, n = 310) and relative (lower, n = 305) methods.

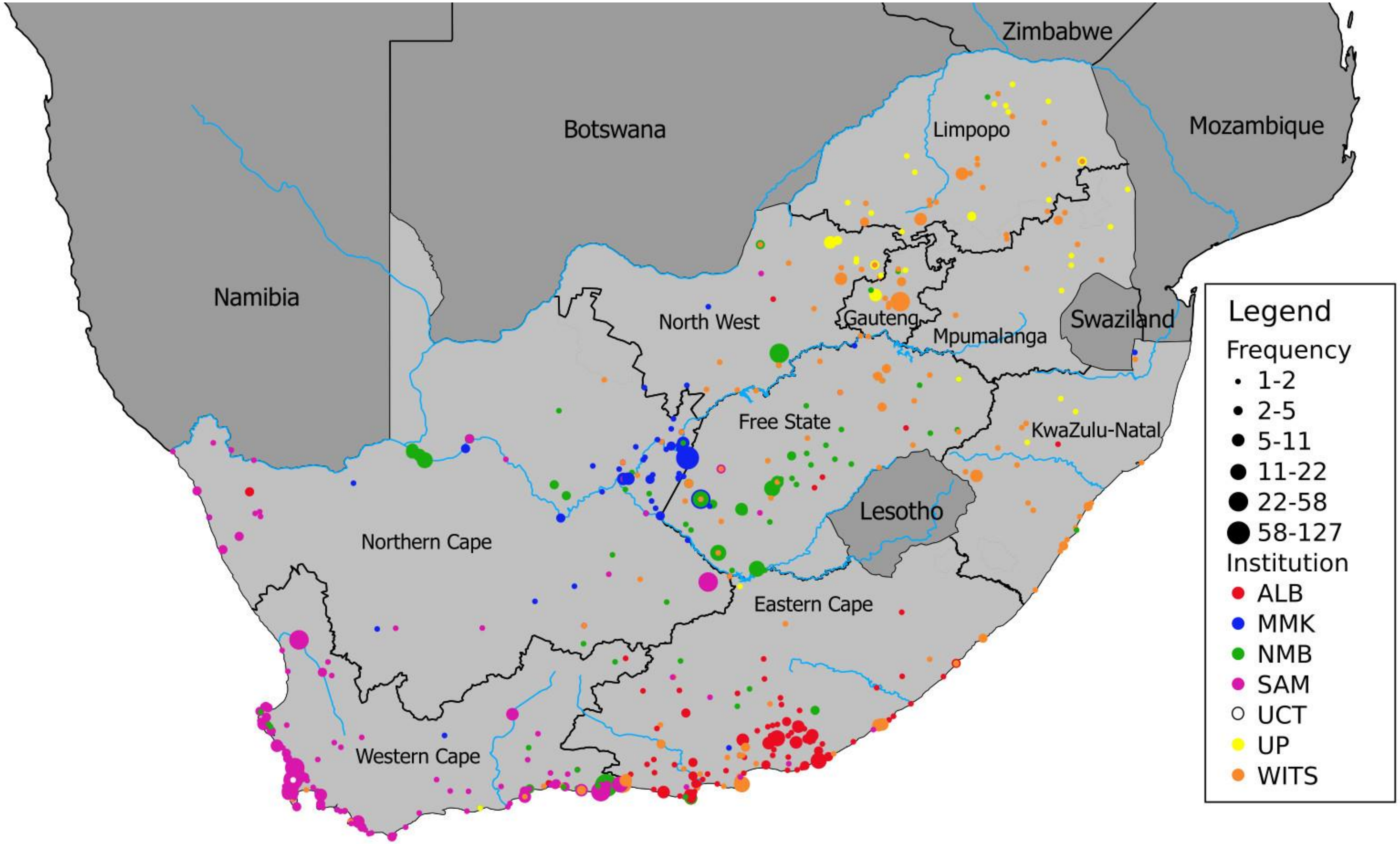


Figure 25: Location of South African samples (n = 1850). Point sizes reflect the frequency of individuals from that location and colours differentiate among curating institutions as is indicated in the legend. The frequency cut-offs were determined using the Natural Breaks (Jenks Algorithm) in QGIS.

LOCATION

For 1850 individuals (79.71%), at least some information regarding location is available; however, the degree of detail varies considerably. Figure 25 presents a map of the location of all individuals for which sufficient information was available to plot the location. The data does appear to cluster around the curating institution, at least in the case of SAM, ALB and MMK. NMB, WITS and UP show a more diffuse distribution of locations for the individuals curated within these institutions. This distribution likely predominately reflects a combination of historical population densities (with greater frequencies at the larger historical urban centres), as well as modern population densities and development (which often result in the discovery of remains). It should be noted that a large proportion of the south African skeletal remains stem from isolated or small groupings of burials.

PALEOPATHOLOGY

Given the large number of individuals examined, a presentation of the differential diagnoses is not feasible. The most likely diagnosis is presented and any pathology for which a diagnosis was not readily forthcoming was either omitted from the analysis or presented as ambiguous. These results should be viewed as guide to the type and frequency of pathology that have been observed in this dataset.

TRAUMA

A total of 221 traumatic events were observed in 166 (7.2%, N = 2321) individuals. Of these, fractures were the most frequently observed and constituted 88.2% of the total trauma (Table 11). Heterotropic ossifications were the next most frequent (9.1%) followed by dislocations (1.4%). Osteochondritis dissecans and an amputation were each observed in a single individual only. Multiple fractures and/or combinations of trauma were observed in 44 individuals. Twenty-six individuals presented with 2 fractures, 9 individuals presented with 3 fractures and 3 individuals presented with 4 fractures. Five individuals had a combination of a single fracture with either heterotopic ossification (n=2), dislocation (n=1), osteochondritis dissecans (n=1) or both dislocation and heterotopic ossification (n=1). The remaining individuals had two elements with heterotopic ossifications. An additional 12 fractures were identified in 6 individuals; however, these were associated with predisposing/pre-existing conditions. One individual had a fracture of a unilaterally elongated styloid process. One individual had numerous fractures associated with a possible diagnosis of Paget's disease (Stynder, 2002), while another had unilateral spondylolysis in association with spina bifida occulta. Finally, three individuals had numerous vertebral body fractures associated with osteoporosis. These pathological fractures were not included in the presentation of trauma bellow. There are a number of discrepancies between van der Merwe et al. (2010c) publication and the Gladstone individuals included in this report. Please note that for some of the trauma cases reported by van der Merwe et al. (2010c) (including some fractures and all of the amputations reported in that study), the skeletal elements involved or, in some cases, the entire skeleton was not physically present/available to be included in this analysis.

Table 11: Frequency table of types of trauma

Trauma - Type	Frequency (%)	Number of individuals
fracture	195 (88.2)	142
heterotopic ossification	20 (9.1)	19
dislocation	3 (1.4)	3
osteochondritis dissecans	2 (0.9)	1
amputation	1 (0.5)	1
Total	221	166

Table 12 presents the frequency of trauma type stratified by element and side. The most frequently observed site for fractures is the vertebral column (25.6%). This is followed, in decreasing frequency, by the ribs (9.7%), metacarpals (9.2%), frontals (8.2%), ulnae (7.7%), parietals (7.2%) and radii (6.2%). For the remaining elements, frequencies are all below 10 (< 5%). Differences between left and right sides are relatively small. The largest difference occurs between the parietals and ulnae, both of which have a greater frequency of fractures on the left side; however, a Fisher's exact test (on sided elements only) reveals differences are not significant ($p = 0.78$, two-sided). Heterotopic ossification was observed most frequently for the femora (35%), followed by the humeri (20%), os coxae (15%), tibiae (15%), metatarsals (10%) and occipital (5%). All dislocations involved the coccyx (n = 3) with ankylosis to the sacrum. Finally, one possible amputation of the ulna was observed and one individual presented with osteochondritis dissecans on both distal femora and both distal tibiae.

The majority of vertebral fractures (n = 31) were cases of spondylolysis (28 bilateral, 2 unilateral (right), 1 undetermined). Of the remaining fractures, 11 were compression fractures of the vertebral body, 4 were compression/avulsion fractures of the anterior superior vertebral body and 4 were fractures of the neural arch not involving the pars interarticularis. The spondylolysis cases predominantly involved the 5th lumbar vertebra (n = 19), with a few cases involving the 4th (n = 8) and 3rd (n = 2) lumbar vertebra. The two remaining cases involved the 3rd-5th lumbar vertebrae as well. Compression fractures were equally distributed between the 12th thoracic and 1st lumbar vertebrae (n = 4), with a single case for each of the 3rd, 10th and 11th thoracic vertebrae. Compression/avulsion fractures are represented by a single case for each of the 12th thoracic, 3rd, 4th, and 5th lumbar vertebrae. For the 4 neural arch fractures, 2 instances involved the unilateral (left) inferior facet. Both of these occurred in the same individual for the 1st and 2nd lumbar vertebrae, with nonunion and ankylosis to the superior facet of the adjacent vertebrae. Another individual had a unilateral (left) fracture of the 5th lumbar vertebra with nonunion just inferior to the superior facet but not at pars interarticularis. The final fracture occurred at the left transverse process of the 2nd lumbar vertebra. Multiple vertebral fractures were present in 8 individuals and in all cases 2 vertebral fractures were present. Three individuals had 2 vertebrae with spondylolysis. In two cases the vertebrae were the 4th and 5th lumbar vertebrae, while for the third individual, the 3rd and 5th vertebra were affected. Two individuals had multiple compression fractures. In one individual the 10th and12th thoracic vertebra were affected, while in the other the 12th thoracic and 1st lumbar vertebrae were affected. One individual had the two fractures of the inferior facets for contiguous vertebra described above. For the last 2 individuals, one had spondylolysis of the 5th lumbar vertebra and fracture of the transverse process for the 2nd lumbar vertebra, while the other had a compression fracture of the 3rd lumbar vertebra with a compression/avulsion fracture of the 5th lumbar vertebra. The most frequent observed complication was nonunion (n = 37). In

Table 12: Frequency of trauma types stratified by skeletal element and side.

	N				Fracture				Heterotopic ossification				Dislocation	Amputation	OCD
Element	L	R	U	Complete (L+R)	L	R	U	Total	L	R	U	Total	U	L	U
Frontal	1525	1514		1432	8	5	3	16							
Parietal	1492	1464		1342	9	3	2	14							
Occipital	1228	1241		1189							1	1			
Temporal	1411	1406		1234	1			1							
Maxilla	1260	1259		1164	1			1							
Zygomatic	1287	1295		1120	1			1							
Nasal						1	6	7							
Mandible	1211	1196		1091	1			1							
Vertebral Column*			510	1109	4	3	43	50							
Coccyx*			59	59									3		
Ribs*	397	385		836	9	10		19							
Clavicle	760	759		633	3	2		5							
Humerus	713	690		562	2	3		5	2	2		4			
Radius	613	610		489	7	5		12							
Ulna	589	590		469	10	5		15						1	
Carpals*	226	252		463		1		1							
Metacarpals*	406	441	34	740	7	11		18							
Hand Phalanges*	14	11	186	839			5	5							
Os Coxa	617	613		513	1			1	3			3			
Femur	796	773		689	1			1	3	4		7			1
Tibia	745	732		652	3	3		6	3			3			1
Fibula	542	511		424	3	3		6							
Metatarsals*	402	432	33	688	2	5		7	2			2			
Foot Phalanges*	3	5	62	628			3	3							
Total	16237	16179	884	18365	73	60	62	195	13	6	1	20	3	1	2

OCD = Osteochondritis dissecans
Side: L = left, R = right, U= unsided or midline elements/grouped elements
N = Total frequency for all well preserved skeletal elements (>75% complete).
Complete (L+R) = Frequency of individuals for which both left and right elements were present and well preserved (>75% present).
*For these elements, N (L, R and U) frequencies represent the number of grouped elements that are well preserved (>75% of the entire group present) and Complete (L+R) frequencies represent the total number of grouped elements present, regardless of preservation

two cases nonunion was observed in conjunction with osteoarthritis, while in four cases nonunion and ankylosis were observed. Kyphosis, as a consequence of wedging due to compression fractures, was observed in 3 individuals.

Most of the fractures on the frontal and parietal were depressed fractures (n = 23). Three penetrating fractures were also observed, two of which were present in the same individual. Finally, two linear fractures were observed. In one individual the fracture line extended along the frontal and parietal, while in the other the fracture was present the squamous part of the left temporal. Multiple vault fractures were observed in three individuals. Most nasal fractures involved both nasal bones (n = 6); however, for one individual only the right nasal bone was involved. The maxillary fracture occurs at the frontal process and is associated with nasal fractures. The mandibular fracture occurs at the left condyle with nonunion of the articular surface.

For the rib fractures, 2 fractures are present for each of the 4th, 7th, 10th and 11th ribs, a single fracture was identified for each of the 2nd, 3rd, 6th and 9th ribs and for the remaining 7 ribs, rib number could not be determined. Nine rib fractures occurred on the left side and ten on the right. Multiple rib fractures were only observed in two individuals, both of whom had two fractured ribs. For one individual both fractured ribs were on the left side and were at different stages of healing (one was completely healed with sclerotic bone, while for the other, woven bone was present) indicating the fractures did not occur simultaneously. The other individual had one rib fractured on each side (3rd-10th ribs).

Metacarpal fractures were most frequently observed for the 5th metacarpal (n = 7). First and fourth metacarpal fractures were observed at equal frequency (n = 4), while 2nd metacarpal fractures were the least frequently observed (n = 3). Seven of the fractures occurred on the left side, while 11 occurred on the right. Three individuals had two metacarpal fractures and in each case both were on the same side. One individual had a fractured left 2nd and 5th metacarpal. The other two individuals had right metacarpal fractures of the 5th metacarpal and either the 1st or 4th metacarpal.

Metatarsal fractures were most frequently observed for the 5th metatarsal (n = 3), followed by the 2nd metatarsal (n = 2). A single fracture is reported for the 3rd and 4th metatarsals. Five metatarsal fractures were recorded for the right side and two for the left. Two metatarsal fractures were observed in only one individual. Both occurred on the right side and involved the 4th and 5th metatarsals.

Fractures of the hand phalanges were observed for the 3rd (n = 2), 4th (n = 2) and 5th (n = 1) digits. The proximal phalanx was involved in all cases except one 4th digit for which the fracture occurred in the intermediate phalanx. Phalangeal fractures of the foot were observed for the 1st (n = 1) and 2nd (n = 2) proximal phalanges. Multiple phalangeal fractures in a single individual were not observed.

Most ulnar fractures occurred in the distal 1/3rd of the shaft (n = 11). For two individuals fractures were reported for the middle 1/3rd, a fracture of the proximal 1/3rd was observed in one individual and for another the location was not determined. Similarly, for the radius most fractures were observed in the distal 1/3rd of the shaft (n=10). A proximal 1/3rd fracture was observed for a single individual and for another the location was not determined. For the humerus a single fracture was observed in each of the proximal, middle and distal thirds of the diaphysis, as well as, the distal epiphyses for 4 individuals. The location was not determined for the 5th individual. The single femoral fracture involved the femoral head with nonunion. Tibial fractures were observed at the middle diaphyseal segment for two individuals. A single tibial fracture was observed in each of the proximal and distal epiphyses, as well as, the proximal diaphyseal segment. For the final individual the site was not determined. Two fibular fractures occurred at the proximal diaphyseal segment and two were also observed at the distal diaphyseal segment. For the final individual the site of fracture was not determined. The fracture of the os coxa occurred at the midpoint of the ischiopubic ramus. Multiple fractures were observed in 36 individuals, some of which have been described above. The accession numbers and details of these individuals can be viewed in Appendix A (Table A 11).

The most frequently observed fracture complication was malunion (n = 65, this excludes vertebrae which were discussed above). Angulation was noted most frequently (n = 41) but was generally minor. Displacement/translation (n = 8), shortening (n = 5), distraction (n = 3) and impaction (n = 1) were also observed but much more infrequently. Angulation was also seen in combination with displacement (n = 2) and rotation (n = 1), while displacement was noted in combination with shortening (n = 2) and both shortening and angulation (n = 2). Nonunion was observed in eight instances, osteoarthritis in nine, ankylosis in three and infection in two. Of these, osteoarthritis was observed in conjunction with malunion (n = 2) and with nonunion (n = 3). While ankylosis was observed with malunion and infection (n = 1), as well as nonunion and osteoarthritis (n = 1).

DEVELOPMENTAL ANOMALIES/DISORDERS

ANOMALIES/DISORDERS OF THE SKULL

The most frequently observed anomaly of the skull was a persistent metopic suture (n = 15). Three individuals were immature (all >10 yrs), while the remaining 12 individuals were adults. Seven individuals were male, while six were female and for two individuals the sex was ambiguous. Two cases of persistent mendosa suture (inca bone) were also recorded. In both instances the individuals were mature males. Two individuals had elongated styloid processes. In one individual the condition was unilateral (left) and the process exceeded 60mm in length. For the other, the condition was bilateral and the styloid processes were approximately 42mm in length. Both individuals were mature males. A single case of a bipartite zygomatic bone was present (os japonicum). A horizontal suture separated this right zygomatic bone into a superior and inferior portion. The left side was not present for observation in this mature male. In one mature female, a transverse basilar cleft separated the basiocciput from the remainder of the occipital bone. Finally, one mature female had an enlarged incisive foramen. The foramen was oval shaped (approx. 1 cm wide, 1.5 cm long), no reactive bone was present and the lesion did not appear to be associated with a dental abscess. This may be a case of a nasopalatine duct cyst. The complete list of developmental anomalies/disorders of the skull with relative frequencies is presented in Table 13.

Table 13: Table of development anomalies/disorders of the skull

Developmental Anomaly/Disorder	Element	Side	Total number of complete elements examined	Number of individuals with observed condition	(%)
Persistent metopic suture	Frontal	M	1432	15	1.1
Persistent mendosa suture	Occipital	M	1189	2	0.2
Elongated styloid process	Temporal	L	1411	2	0.1
Bipartite zygomatic	Zygomatic bone	R	1295	1	0.1
Transverse basilar cleft	Occipital	M	1189	1	0.1
Enlarged incisive foramen	Maxilla	M	1164	1	0.1

Side: M = midline, L = left, R = right

ANOMALIES/DISORDERS OF THE VERTEBRAL COLUMN

Border shifts between vertebral regions were the most frequently observed vertebral developmental field anomalies (Table 14). Caudal border shifts were far more numerous than cranial shifts, as were simple border shifts (where only one border was affected). Of the caudal border shifts, shifts at the lumbosacral border were observed most frequently (n = 22), followed by the thoracolumbar border (n = 19), the sacrocaudal border (n = 18) and, finally, the occipitocervical border (n = 1). Cranial border shifts occurred most frequently at the thoracolumbar border (n = 10), followed by the lumbosacral border (n = 3) and the cervicothoracic border (n = 2). For three of the five possible sacrocaudal caudal border shifts, the coccyx was not present and these may just represent additional sacral elements rather than a border shift. Vertebral border shifts involving multiple borders or the presence of additional vertebral segments occurred less frequently. Caudal border shifts involving the thoracolumbar border and borders caudal to it were present in nine individuals, while cranial border shifts including the cervicothoracic border and borders caudal to it were present in four individuals. For three individuals both cranial and caudal border shifts were present. For two individuals this involved a cervicothoracic cranial border shift in addition to a lumbosacral caudal border shift or a possible sacrocaudal caudal border shift (/an additional sacral element – the coccyx was not present). Finally, for the third individual, an occipitocervical caudal border shift and lumbosacral cranial border shift were both observed. Vertebral border shifts were more common among males (n = 54) than females (n = 29). For four individuals sex was ambiguous and sex determination was not possible for the remaining six individuals. Sixty-six individuals were mature and 27 were immature (>10 yrs).

Vertebral developmental disorders other than border shifts were present but were observed less frequently. Disorders affecting the neural arch (n = 41) were far more common than those affecting the vertebral body (n = 10). Vertebral body disorders included hypoplasia of the vertebral body (anterior, n = 2; lateral, n = 2) and block vertebrae (n = 6). Of the disorders affecting the neural arch, 17 individuals presented with possible spina bifida occulta. In 14 of these individuals, the defect bisected the spinous process, while for the remaining 3 individuals the defect occurred just right of the spinous process. The 11th thoracic vertebra was most frequently affected (n = 6), followed by the 7th cervical (n = 3), 1st thoracic (n = 3), 4th thoracic (n = 2) and 5th lumbar (n = 2) vertebrae. The defect was recorded only once for each of the 2nd, 5th and 12th thoracic vertebrae, as well as the 4th lumbar vertebra. In four individuals, occurrences in multiple vertebrae were observed. Two adjacent vertebrae were affected in two individuals and three vertebrae (two of which were adjacent and the third located in a superior region) were affected in the other two individuals. Spina bifida occulta of the sacrum was also observed in three individuals. In two of these individuals the median crest of the 1st sacral element was affected, while in the third individual the entire median crest was bisected by the defect. Cleft neural arches of the 1st cervical vertebra were also observed in nine individuals (anterior arch = 3, posterior arch = 6). For one individual a cleft bisected an inferior zygapophyseal facet, unilaterally. Aplasia/hypoplasia of was noted in a number of individuals and involved the spinous process (n = 3, 2 associated with a cleft), transverse process (n = 3) and zygapophyseal facets (n = 2). For the zygapophyseal facets, multiple contiguous vertebrae were affected. In one case, the expression was unilateral (left) affecting T11-L3, while for the other, both unilateral and bilateral expression was observed for T12-L2.

Table 14: Developmental disorders of the vertebral column

Developmental disorder	Number of individuals affected	%
Border shifting	93	18.2
Spina bifida occulta – vertebrae	17	3.3
Spina bifida occulta – sacrum	3	0.5*
Cleft posterior arch	6	1.2
Cleft anterior arch	3	0.6
Cleft zygapophyseal facet	1	0.2
Hypoplasia – vertebra body	4	0.8
Aplasia/hypoplasia - neural arch	8	1.6
Block vertebrae	6	1.2

Percentages are calculated by dividing the number of affect individuals with the total number of individuals observed with complete (>75% present) preservation of the vertebrae (N = 510) or *sacrum (N = 554).

Table 15: Developmental disorders of the sternum and ribs.

Developmental disorder	Number of affected individuals	N	%
Sternal aperture	44	459	9.6
Cleft/Bifurcated sternal body (inferior)	6	459	1.3
Misplaced manubriosternal joint	4	302	1.3
Bifurcated rib (sternal)	2	715	0.3
Fused ribs	1	715	0.1

N = total number of individuals for which the disorder could be observed.

The most frequently observed developmental disorder affecting the sternum was a sternal aperture (n = 44, Table 15). A cleft or bifurcated sternal body was noted in six individuals and in one individual a cleft was present inferiorly, in addition to, two sternal apertures. A misplaced manubriosternal joint was observed in four individuals. Very few developmental disorders of the ribs were noted (n = 3). Two individuals presented with a bifurcated rib at the sternal end, while a third presented with two fused ribs.

The most frequently observed disorder of the upper limbs was the presence of an os acromiale of the scapulae (n = 50, Table 16). The condition was only diagnosed in adult individuals. The condition was bilateral for 18 individuals and unilateral for 10 individuals (7 left, 3 right). For the remaining 22 individuals (12 left, 10 right), only one scapula was present and the unilateral/bilateral condition could not be assessed. Os acromiale was observed in twice as many males (n = 30) as females (n = 16). The sex could not be determined for four individuals). Fused carpals were identified in 12 individuals. In all cases the lunate and triquetral were fused. The condition was bilateral in six individuals, unilateral in one and a unilateral/bilateral state could not be determined for the remaining five individuals (2 left, 2 right and 1 unknown). A coracoclavicular joint was observed in four individuals and the condition was unilateral in all cases (3 left, 1 right). A hyploplastic hamulus was observed in three individuals. The condition was bilateral for one, unilateral (right) for another and was present on the right side for the third individual but the left was not preserved. A single individual presented with a unilateral (right) os hamuli proprium or bipartite hamate. Finally, os styloideum of the 3rd metacarpal was observed for three individuals. In one case the condition was bilateral and the bone had fused to the capitate. The condition was observed on the left 3rd metacarpal for the remaining two individuals; however, only in one individual was the condition truly unilateral, as the right 3rd metacarpal was not preserved for the other.

Table 16: Developmental disorders of the upper limb.

Developmental disorder	Number of affected individuals	N	%
os acromiale	50	620	8.1
fused carpals	12	204	5.9
coracoclavicular joint	4	937	0.4
hypoplasic hamulus	3	204	1.5
os hamuli proprium/bipartite hamate	1	204	0.5
os styloideum, 3rd metacarpal	3	204	1.5

N = total number of individuals for which the disorder could be observed.

The most frequently observed developmental anomaly of the lower limbs was the bipartite patella (n = 8, Table 17). This was bilateral in one individual, unilateral in five individuals (two left, three right) and was present on the right side for the remaining two individuals but the left patellae were not present. The bilateral presence of a vastus notch was noted in one individual. Tarsal coalition and tarsometatarsal coalition were observed in a single individual, respectively. The tarsal coalition was unilateral (right) and involved the lateral and intermediate cuneiforms. The tarsometatarsal coalition was also unilateral (left) and involved the medial cuneiform and 1st metatarsal. A single bipartite sesamoid was also observed.

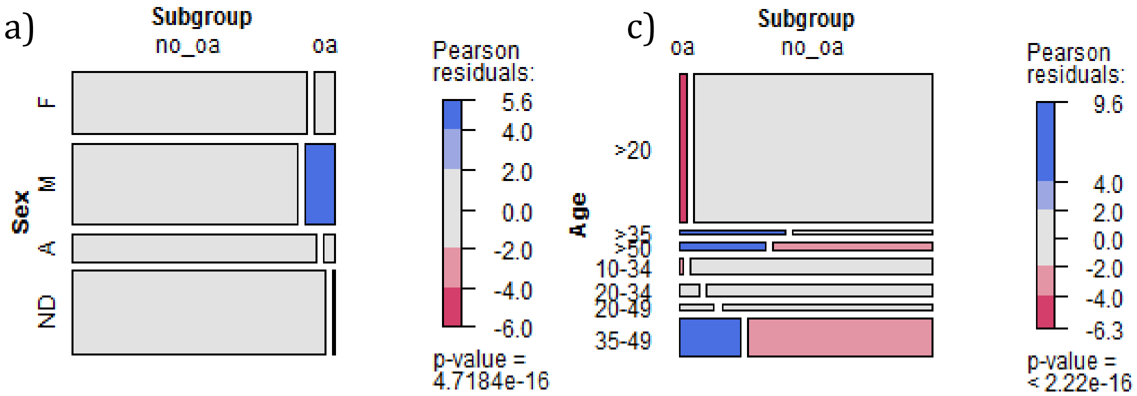
Table 17: Developmental disorders of the lower limb.

Developmental disorder	Number of affected individuals	N	%
bipartite patella	8	544	1.5
vastus notch	1	544	0.2
tarsal coalition	1	304	0.3
tarsometatarsal coalition	1	264	0.4

N = total number of individuals for which the relevant element was present and complete on the left or right side.

JOINT DISEASE

In total, 368 individuals showed some pathological changes to the joint surfaces (15.9%). Eburnation was observed in 50 individuals, grooving in only 9 individuals, surface pitting in 202 individuals, marginal osteophytes in 198 individuals, surface osteophytes in 33 individuals and alteration of the surface contour in 50 individuals (Table 18). Using Waldron's (2009a) criteria for diagnosing osteoarthritis, 153 individuals presented with eburnation or at least two other indicators of the disease (marginal osteophytes, surface osteophytes, pitting and alterations to the surface contour). This constitutes 6.6% of all individuals analyzed in this study (N = 2321). The condition was monoarticular in 14 individuals, pauciarticular in 38 individuals and polyariticular in 96 individuals. Of the 153 individuals identified with osteoarthritis, only 96 of the skeletons were complete (>75% present), which constitutes 21.1% of complete skeletal remains (N = 455). Of these 153 individuals, 87 were males and 46 were females. For an additional 12 individuals, the sex was ambiguous and for 8 individuals could not be determined. The sex distribution for the osteoarthritis subset of individuals is significantly different from the rest of the dataset (with the osteoarthritis subset removed) ($\chi^2 = 74.47$, $df = 3$, $n = 2321$, $p < .0001$). An examination of the residuals reveals this difference is driven by the greater frequency of males in the osteoarthritis subset (Figure 26a) and the odds of observing a male relative to female in the osteoarthritis group is 1.5 times that of the remaining dataset (95% CI [1.05,2.23]) (Figure 26b). Most of the individuals (n = 74) were middle adults (35-49 yrs). Osteoarthritis was observed in only 19 old adults (>50 yrs), 8 young adults (20-34 yrs) and 2 adolescents (10-34 yrs). Five, nine and thirty-six individuals fell into the broader age categories of 20-49 yrs, >35 yrs and >20 yrs, respectively. The age distribution is also significantly different from the remaining dataset (χ^2 = 232.72, *df* = 6, n = 1780, *p* < .0001), with a greater frequency of middle and old adults than expected in the osteoarthritis subgroup (Figure 26c and d).



b) **Odds ratios (95% CI)**

	Subgroup
Sex	No OA:OA
F:M	**1.53 (1.05-2.23)**
M:A	**0.37 (0.2-0.68)**
A:ND	**0.22 (0.09-0.53)**

d) **Odds ratios (95% CI)**

	Subgroup
Age	OA:No OA
>20:20-34	**0.37 (0.17-0.83)**
>35:20-34	**8.63 (2.8-26.61)**
>50:20-34	**6.24 (2.5-15.56)**
10-34:20-34	**0.19 (0.04-0.89)**
20-34:20-49	0.52 (0.16-1.72)
20-34:35-49	**0.26 (0.12-0.57)**

Figure 26: Mosaic plots and odds ratios for Osteoarthritis and non-osteoarthritis subgroups. a) Mosaic plot with shaded Pearson residuals comparing sex and data subgroups. b) Odds ratios and 95% confidence intervals comparing sex and data subgroups. c) Mosaic plot with shaded Pearson residuals comparing age-at-death and data subgroups. d) Odds ratios and 95% confidence intervals comparing age-at-death and data subgroups. Confidence intervals that do not contain the null are shown in bold. Sex: F = grouped female and probably female, A = ambiguous, M =grouped male and probably male, ND = not determined.

Changes to the articular surfaces of the vertebral bodies, including pitting and marginal osteophytes, were noted in 112 and 192 individuals, respectively (Table 19). Intervertebral disc disease was diagnosed when both features were present and 89 individuals met this criteria (3.83%, N = 2321). The condition was monoarticular in 9 individuals, pauciarticular in

Table 18: Frequency of osteoarthritis and associated features.

Preservation Score*	N	Any OA feature	Eburnation	Grooving	Pitting	Marginal osteophytes	Surface osteophytes	Alteration of bone contour	Eburnation / >2 OA features
1	455	185	23	5	111	91	23	32	96
2	627	132	18	4	64	74	8	9	40
3	1239	51	9		27	33	2	9	17
Total	2321	368	50	9	202	198	33	50	153
%	2321	15.9	2.2	0.4	8.7	8.5	1.4	2.2	6.6

*Preservation scores reflect the total skeletal preservation: 1=75-100%; 2= 25-50% and 3=0-25%.

Table 19: Frequency of intervertebral disc disease and Schmorl's nodes.

Preservation Score*	N	Pitting	Marginal osteophytes	Pitting and Marginal Osteophytes	Schmorl's Nodes
1	510	69	108	54	39
2	308	34	68	28	12
3	291	9	16	7	4
Total	2321	112	192	89	55
%	2321	4.8	8.3	3.8	2.4

*Preservation scores reflect preservation of the vertebrae: 1=75-100%; 2= 25-50% and 3=0-25%.

12 and polyarticular in 59. Of these 89 individuals, 28 were female and 52 were male. For three, the sex was ambiguous and for six the sex could not be determined. The greatest frequency of individuals (n = 49) were middle adults (35-49 yrs). Ten individuals were old adults (>50) and three were young adults (20-34 yrs). Two, seven and fifteen individuals were from the broad age categories of 20-49 yrs, >35 yrs and >20 yrs, respectively. Intervertebral disc disease was also identified in three immature individuals (10-34 yrs). The comparison between the sex and age distribution for intervertebral disc disease and the rest of the dataset mirrors that described for osteoarthritis. Both the sex distribution ($\chi^2 = 46.89$, $df = 3$, $n = 2321$, $p < .0001$) and the age-at-death distribution ($\chi^2 = 190.14$, $df = 7$, $n = 1896$, $p < .0001$) are significantly different to the remaining dataset. For the sex distribution this difference is driven a greater than expected frequency of males and for the age distribution, a greater frequency of middle and old adults (Appendix,Table A 1). The presence of Schmorl's nodes were also noted and these were identified in 55 individuals (2.4%, N = 2321).

In addition to osteoarthritis and intervertebral disc disease, one possible case of ankylosing spondylisis and one possible case of gout were observed. The right sacrum and ilium, as well as two lumbar vertebrae were fused in one individual. Post-mortem damage precluded the identification of vertebral number and observation of the left sacroiliac joint so the diagnosis here is tentative. In another individual, erosive lesions were present along the margins of the left 1st metatarsal distal articular surface, crosscutting articular and non-articular bone on both the medial and lateral surfaces resulting in overhanging margins.

OSTEOPOROSIS

The remains of 17 individuals were noted as being abnormally light in weight. Of these, two individuals showed regions of translucency (the skull in one case and the pelvis in another). For three individuals, regions of porosity were noted along the vertebral column (n = 2) and cranium (n = 1). The osteopenia/osteoporosis appeared to be secondary to another pathological condition in four individuals. For one individual, the elements of the left lower limb were much lighter in weight than those of the right indicating possible disuse osteopenia/osteoporosis, secondary to a femoral neck fracture. In two individuals, the osteopenia was noted in association with lytic destruction of the vertebrae and/or sacroiliac joint, possibly as a consequence of tuberculosis. For the remaining individual, “penciling” of numerous elements of the hands and feet are present and this may represent a case of leprosy. Waldron (2009a) suggests osteoporosis can only be securely diagnosed when osteoporotic fractures are present. In this regard, multiple fractures were noted in three individuals. Two had multiple fractures of the thoracic and lumbar vertebral bodies, while the third individual had a single vertebral body fracture in addition to a healed fracture of the ischiopubic ramus. Of the 17 cases of possible primary osteopenia/osteoporosis, seven were females, three were males and the sex was ambiguous for the remaining three. Six individuals were classified as middle adult (35-49 yrs), three as old adult (>50 yrs), one as adolescent (10-34 yrs) and the remaining three individuals could only be classified as adult (>20 yrs).

NEOPLASTIC DISEASE

The most frequently observed lesions of neoplastic origin were “button” osteomas of the external table of the skull. These well-circumscribed, dense, ivory-like nodules were observed in 24 individuals. Single osteomas were observed in 18 individuals, while multiple osteomas were observed in six. These osteomas were predominately located on the frontal (n = 8) and parietal (n = 8) bones or both (n = 7) but for one individual an osteoma was noted on the occipital. Six of these individuals were female, thirteen were male, the sex was ambiguous for four and could not be determined for one individual. One individual was an adolescent (10-34 yrs), two were young adults (20-34 yrs), five were middle adults (35-49 yrs), two were old adults (>50 yrs) and the remaining 14 individuals could only be identified as adult.

AUDITORY EXOSTOSES

Auditory exostoses were observed in four individuals. In three cases the condition was bilateral and in two of these, occluded more than 50% of the auditory canal. For the fourth individual, only the right temporal was present. All burials were located in coastal regions of the Western Cape (Struisbaai, Pearly Beach, Melkbosstrand and Gordons Bay). Two individuals were male, one was probably a female (although many of the cranial features were ambiguous) and for the final individual the sex was ambiguous. All individuals were adults. In three cases only the skull was present. A radiocarbon date for the possible female of Gordon's Bay was reported by Stynder (2009) as 2815±40 BP, while that of the male from Pearly Beach was reported as 1892±28 BP.

ATROPHY

Four cases of atrophy were observed. The elements of one side were more slender, gracile and/or shorter than their contralateral counterparts. In all cases it was the left side that was atrophic. In three of the four individuals the upper limb was involved, while in the 4th individual the lower limb was affected. In cases involving the upper limbs, the atrophic left elements were shorter than the right. Due to postmortem damage, difference in length could not be assessed for the individual in which the lower limbs were affected. In one case the atrophy was likely secondary to pathological changes to the radius and ulna that involved an abnormally shaped proximal articular surface and abnormal curvature of the ulna. Three of the individuals were male, one was female and all were middle adults (35-49 yrs).

ANKYLOSIS

Ankylosis was observed in 18 individuals. In all cases there was no evidence of trauma, infection or joint disease at the joint specifically. Partial fusion at the sacroiliac joint was observed in five individuals. The condition was unilateral (right) in three individuals, bilateral in the 4th, and only the left side could be observed for the last individual. In all cases no vertebrae were affected. In three instances, there were fractures present elsewhere in the skeleton (femoral head, fibula and proximal foot phalanx), one individual showed a lytic pathology at the right scapula and in two individuals minor to moderate marginal osteophyte formation was observed on many other skeletal elements. Partial posterior ankylosis of the left talus and calcaneus was noted in one individual (the contralateral elements were not present). There is widening of the articular surfaces with the tibia and some ossification at the entheses of the talus, calcaneus and fibula. One individual showed unilateral posterior partial fusion of the left tibia and talus. Widening of the articular surface with the fibula is observed. Another individual showed ankylosis of the zygapophyseal joints (only) of the 4th and 5th thoracic vertebrae. While there is no evidence of trauma to the affected joint, this individual does have ankylosis of the sacrum and coccyx resulting from a coccygeal dislocation. Ankylosis of the right 12th costovertebral joint is observed in one instance, while in another ankylosis of the right metarasophalangeal joint of the 1st digit is noted. The remaining cases all involved ankylosis of the proximal and intermediate phalanges. In three cases the manual digits were affected (3rd: n = 2, 2nd: n = 1). The remaining six cases all involved the pedal phalanges. The 3rd (n = 3), 4th (n = 1) and 5th (n = 1) digits were affected and in one case the digit number could not be determined. In two instances, there was evidence of trauma elsewhere in the skeleton but none at the joint itself. Finally, in two individuals (one manual and one pedal proximal interphalangeal joint) the joint was fused in a flexed position.

CRIBRA ORBITALIA

Porosity of the superior orbits was noted in 107 individuals. This constitutes 6.9% of the total number of individuals for which the frontal was >75% complete. Of these individuals, the lesions were raised in 35 individuals, in three individuals a colour change was noted when the pathological region was compared to the normal bone surface. In nine cases the bone was sclerotic.

HYPEROSTOSIS FRONTALIS INTERNA

Two possible cases of hyperostosis frontalis interna were observed. In both cases, abnormal bone formation is present along the internal table of the frontal bone superior to the orbits, resulting in an increased width. The bone is dense, sclerotic, results in a slightly irregular surface and added considerable weight to both crania. For each individual, only the skull was recovered so a complete assessment of the distribution of pathology was not possible. Both could only be identified as adult (>20 yrs). One was probably female while the sex of the other was ambiguous.

PERIOSTEAL NEW BONE FORMATION ON THE POST-CRANIAL SKELETON

In total, 44 individuals presented with periosteal new bone formation on the bones of the post-cranial skeleton. In 18 of these individual, the lesions had well circumscribed margins and the expression was unilateral in all but 2 instances (for which the occurrence was bilateral). The tibia was the most frequently affected element (n = 15), followed by the fibula (n = 2) and metacarpals (n = 1). These lesions were noted on the right tibia in eight individuals, left tibia in five and occurred bilaterally in two individuals. In nine cases the bone was sclerotic, in one case woven and for the remaining individuals, lesions of mixed bone types were observed.

Lesions affecting a single element but with poorly circumscribed margins were observed in an additional 8 individuals. Here the tibia and fibula were the most frequently affected elements (n = 3), while a single case for the femur and humerus were observed. The bone was woven in one case, lamellar or sclerotic for two, and mixed lesions were observed for three individuals.

For the remaining individuals, a more diffuse pattern was observed on multiple skeletal elements. Only the lower limbs were affected for 11 individuals, while both lower and upper limbs were affected in 5 individuals. The vertebrae and tibia were affected in two individuals. For most elements bilateral expression was most common, followed by unilateral right expression, with the fewest lesions recorded on the left side only. The tibia was most commonly affected (n = 16), followed by the fibula (n = 10), femur (n = 7), radius (n = 4) and ulna (n = 3). The metatarsals, humerus and tarsals were involved in two individuals each, while a single instance of metacarpal and os coxal involvement was observed. In four individuals the bone was woven but for the remaining individuals, lesions were of mixed or transitional bone types.

For a number of individuals (not included in the total above), the extent of new bone formation was extensive these include the Gladstone individuals identified as possible cases of scurvy and trepanematoses in van der Merwe et al. (2010b, 2010a). In these cases extensive new bone formation was noted on the bones of the lower and, less frequently, upper limbs. In two individuals bilateral bone addition to the anterior surfaces of the tibia was noted in conjunction with lytic lesions (somewhat resembling carries sicca) on the external surface of the parietal and frontal bones.

OSTEOMYELITIS

Three individuals presented with pathologies consistent with osteomyelitis. In one individual, a large cloaca was visible on the right distal tibia. Significant new bone formation was present along the entire tibial and fibular shafts with bony ankylosis of the tibia and fibula at their articulations. An immature (long bone epiphyses fusing) individual presented with pronounced morphological alteration of the left distal femur. Significant new bone formation surrounded the exposed medullary cavity. The distal third and distal epiphysis of the femur was not present. The proximal tibial epiphysis had been lost post-mortem but no pathological changes were observed on the tibial shaft. This may represent osteomyelitis secondary to a fracture of the distal femur. The final individual presented with bilateral thickening of the ulna shafts. Two small cloacae were visible on the right ulna but none were present on the left. Bilateral occurrence of osteomyelitis is rare and alternative diagnoses should be explored in this case.

OTHER

In one instance abnormal thickening of skeletal elements with no obvious periosteal reaction was noted on several skeletal elements including the internal table of the frontal bone (resulting in a very heavy cranium), several metacarpals and manual phalanges (bilateral), as well as the left ulna. This individual also had two well circumscribed lytic lesions present at the entheses on the lesser trochanter of the left femur and the left superior pubis.

SOUTH AFRICAN CASES OF POSSIBLE TUBERCULOSIS

In total, twelve possible cases of tuberculosis were identified. In all cases, the pathology is consistent with tuberculosis; however, confidence in this diagnosis varies considerably. Four individuals (GLD 8.3, UCT 552, UP 49 and MRB\20) were identified from publications or unpublished theses. The remaining individuals were identified by the author via screening of the skeletal collections within South Africa. All individuals except MRB\20 were examined by the author. MRB\20 was identified in the literature and was reburied prior to the onset of this project. The description for this individual is based solely on the published report. Table 20 presents a summary of the burial locations, demographic information and pathologies of these individuals. Detailed profiles for each individual are presented below.

These twelve cases represent 0.52% of the total number of individuals observed (N = 2321). For seven individuals, the lesions were predominantly lytic in nature, while for the remaining five individuals only proliferative lesions (abnormal periosteal bone formation) were observed (Table 21). Lytic lesions predominantly affected the vertebrae (n = 5), followed by the articulating surfaces of the sacroiliac joint (n = 2). Single observations were noted for the pubis, ulna and scapula. Three of the seven cases, for which lytic lesions predominated, showed multiple foci of lytic pathology (these do not include lesions affecting articulating surfaces of adjacent skeletal elements). GLD 8.3 had at least three foci of lytic activity which included the caudal surfaces of two lumbar vertebrae and the right ulna. The remaining two individuals presented with two foci each. NMB 1229 showed lytic destruction at articular surfaces of the right sacroiliac joint and pubic symphysis, while UCT 552 had lytic foci at the thoracic vertebrae and left sacroiliac joint surfaces. For the two individuals with involvement of the sacroiliac joint, the auricular surfaces of both the ilium and sacrum were involved with partial destruction of the joint in one case (UCT 552) and complete destruction in the other (NMB 1229). For the pathological scapula (MMK 235) and ulna (GLD 8.3), the respective humeri did not show any pathological alterations.

Of the five individuals with vertebral lesions, lytic activity was observed on single vertebra for only two individuals (SAM-AP 1271 and SAM-AP 3738). For a further two individuals, the

Table 20: Summary table of location, demographic and pathological information for possible cases of skeletal tuberculosis

Accession number	Type and location of pathological lesion(s).	Location of burial	Date of burial	Body position in burial	Population affinity	Sex	Age-at-death
MMK 235	Lytic lesion located on the right scapula	Riet River, Koffiefontein, Northern Cape	AD 1428-1666 (Radiocarbon date: 390 ± 50 BP (Pta-2894)	"crouched" / flexed	Khoesan	Female	Middle adult (35-49 yrs)
MMK 331	Bone deposition along pleural surface of some left and right ribs.	Transvaal Rd, Kimberley, Northern Cape	Late 19th century (1870-1883)	Unknown	Black African	Probably Female	Adult (20+ yrs)
GLD 38.8	Lytic lesions on the 3rd and 4th lumbar vertebrae. Abnormal bone deposition on the anterior surface of the 2nd -4th lumbar vertebrae, pleural surface of 2nd -11th right ribs, radii, tibiae and fibulae.	Outside boundaries of Gladstone Cemetery, Kimberley, Northern Cape	Late 19th century (1897-1900)	Extended on R side, arms flexed, no coffin	Black African	Male	Middle adult (35-45 yrs)
GLD 8.2	Abnormal bone deposition on pleural surface of the left 4th-6th and right 1st-5th ribs.	Outside boundaries of Gladstone Cemetery, Kimberley, Northern Cape	Late 19th century (1897-1900)	Prone, 8.3 in same grave, no coffin	Black African	Male	Middle adult (35-45 yrs)
GLD 8.3	Lytic lesions located on 4th and 5th lumbar vertebrae and olecranon process of R ulna.	Outside boundaries of Gladstone Cemetery, Kimberley, Northern Cape	Late 19th century (1897-1900)	Prone, 8.2 in same grave, no coffin.	Black African	Male	Middle adult (35-45 yrs)
NMB 1229	Complete obliteration of the right sacroiliac auricular surfaces with evidence of an abscess/sinus tract extending along the ventral surface of the sacrum. Lytic lesions are present on the right pubic symphyseal face as well.	Farm Kareeboom, Wolmaransstad, North West	Probably early 20th century	Unknown	Black African (informal cemetery for farm labourers)	Male	Middle adult (35-49 yrs)

NMB 1416	Abnormal bone deposition along the pleural surface of the left 2nd-11th ribs.	Farm Rooipad, Kakamas, Northern Cape	Probably 1750-1850	Flexed	Admixed Khoesan / Black African	Male	Middle adult (35-45 yrs)
SAM-AP 1271	Lytic lesion located on 4th lumbar vertebra.	Henkries, Northern Cape	Probably 19th century	Lying extended (Christian influence)	Khoesan	Female	Old adult (50+ yrs)
SAM-AP 3738	Lytic lesion located on the 12th thoracic vertebra.	Prestwich Street, Green Point, Cape Town, Western Cape	Probably 18th century	Unknown	Unknown	Male	Middle adult (35-49 yrs)
UCT 552	Lytic lesions located on the 6th, [7th] and 8th thoracic vertebrae and left sacroiliac joint articular surfaces. Abnormal bone formation on the 7th cervical-5th thoracic, 8th thoracic-1st lumbar vertebrae, left 4th-10th and right 2nd-11th ribs.	Cobern St, Greenpoint, Cape Town, Western Cape	Probably 18th century	Extended on back (Christian-style burial)	Admixed Black African (Likely a foreign-born individual based on isotopic data)	Male	Middle adult (35-49 yrs)
UP 49	Bone deposition on several ribs and the right scapula.	Makgope, Farm Nooitgedagcht, Rustenburg, North West	AD 1670-1784 (calibrated radiocarbon date)	Flexed	Black African (Late Iron Age agropastoralist)	Male	Old adult (50+ yrs)
MRB\20	Abnormal bone formation along the pleural surface of some right ribs.	Maroelabult, Farm De Kroon, near Brits, North West	Probably 1890-1950	Unknown	Black African (Sotho-Tswana)	Not determined (Juvenile)	3 yrs old

Table 21: Summary table of possible tuberculosis cases showing the type and location of lesions

	Element							
Accession No.	Vertebrae	Ribs	Ilium	Sacrum	Pubis	Ulna	Scapula	Long bones
MMK 235							L	
MMK 331		P						
GLD 38.8	L							P
GLD 8.2	P	P						P
GLD 8.3	L					L		P
NMB 1229			L	L	L			
NMB 1416		P						
SAM-AP 1271	L							
SAM-AP 3738	L							
UCT 552	L	L and P	L	L				
UP 49		P					P	
MRB\20		P						
Total	5	6	2	2	1	1	2	3
N*	510	715	838	497	523	590	515	327
%	0.98	0.84	0.24	0.4	0.19	0.17	0.39	0.92

Type of lesion: L = lytic lesion, P = proliferative lesion
*Total number of individuals for which the element could be observed. In all cases, excluding the ribs, the frequency of complete elements is reported (>75% present). For the ribs, this value includes partially complete rib pairs (>25% present). For the ilium and long bones, total frequencies for bilaterally complete elements are reported; however, for the pubis, ulna and scapula, only the right sides are reported.

Table 22: Vertebral elements with lytic lesions

	Vertebral Element						
Accession No.	T6	T7	T8	T12	L3	L4	L5
GLD 38.8					1	1	
GLD 8.3						1	1
SAM-AP 1271						1	
SAM-AP 3738				1			
UCT 552	1	1	1				
Total	1	1	1	1	1	3	1
N*	516	516	516	549	543	533	536
%	0.19	0.19	0.19	0.18	0.18	0.56	0.19

*Total number of individual with complete (>75% present) preservation of the vertebral element (T12, L3-L5). Values for T6-8 represent complete preservation of the grouped 1st to 9th thoracic vertebrae.

New periosteal bone formation was observed on the ribs, vertebrae, scapula and long bones. Rib lesions were observed for six individuals and were the main pathological lesion for five. For three individuals periosteal bone formation occurred bilaterally. For one individual only the left ribs were affected, while for another, only the right ribs were affected. Finally, due to post-mortem damage and pathology, for one individual the bilateral/unilateral expression could not be determined. The 4th-6th ribs were the most frequently affected (n = 5 each). Three instances for each of the 1st-3rd and 7th-10th ribs were observed, while two instances were observed for the 11th rib and only one for the 1st rib. For three individuals the rib number could not be accurately determined. No pathology was observed on the 12th ribs. In all cases the pleural surface of the ribs were affected. UCT 552 also had new bone formation extending from the pleural surface onto inferior surface and, in some instances, external rib surface as well. The entire rib shaft was affected for UP 49. Pathology was observed at the lateral aspect of the ribs for all six individuals and exclusively so for three. For two individuals the pathology extended to the vertebral end, for one individual to the sternal end and for two individuals the entire rib was affected. Abnormal bone formation was also noted on the right scapula of MMK 235. Furthermore, periosteal bone formation was noted on some of the long bones for all three of the Gladstone (GLD) individuals. In all three instances, the tibiae were affected. For one individual the radii and fibulae were also affected and for another, the femur.

New periosteal bone formation was observed on the ribs, vertebrae, scapula and long bones. Rib lesions were observed for six individuals and were the main pathological lesion for five. For three individuals periosteal bone formation occurred bilaterally. For one individual only the left ribs were affected, while for another, only the right ribs were affected. Finally, due to post-mortem damage and pathology, for one individual the bilateral/unilateral expression could not be determined. The 4th-6th ribs were the most frequently affected (n = 5 each). Three instances for each of the 1st-3rd and 7th-10th ribs were observed, while two instances were observed for the 11th rib and only one for the 1st rib. For three individuals the rib number could not be accurately determined. No pathology was observed on the 12th ribs. In all cases the pleural surface of the ribs were affected. UCT 552 also had new bone formation extending from the pleural surface onto inferior surface and, in some instances, external rib surface as well. The entire rib shaft was affected for UP 49. Pathology was observed at the lateral aspect of the ribs for all six individuals and exclusively so for three. For two individuals the pathology extended to the vertebral end, for one individual to the sternal end and for two individuals the entire rib was affected. Abnormal bone formation was also noted on the right scapula of MMK 235. Furthermore, periosteal bone formation was noted on some of the long bones for all three of the Gladstone (GLD) individuals. In all three instances, the tibiae were affected. For one individual the radii and fibulae were also affected and for another, the femur.

Most of the individuals were males (n = 8) who are estimated to have died between the ages of 35 and 49 years (middle adults) (n = 9). Only three were female and the sex of the juvenile individual was not determined. One individual was likely an old adult (>50 yrs) and the juvenile was estimated have reached its 3rd year at death. Due to post-mortem damage the age estimate for one individual could not be refined further than adult (>20 yrs). The population affinity of these individuals was determined based on contextual information about the burial and associated grave goods/inclusions. Most of the individuals were Black Africans (i.e. descended from Bantu-language speakers) (n = 7), three were likely Khoesan, one was probably not born in South Africa and the affinity of the final individual could not be determined. The locations of the burials are shown in Figure 27. Most individuals were unearthed from the Northern Cape. Specifically, four burials were located in Kimberley from an urban context and a single burial each was located near Koffiefontein, Kakamas and Henkries. Three burials were located in the North West, near the towns of Wolmaransstad, Rustenburg and Brits, respectively. Finally, two burials were located in the Western Cape and both are from the same informal burial ground in Green Point, Cape Town. All individuals are dated to the colonial period and most to the 18th or 19th centuries. At least one individual may date to the 16th-17th centuries (MMK 234) (uncalibrated radiocarbon date: 390 ± 50 BP) (Figure 28).

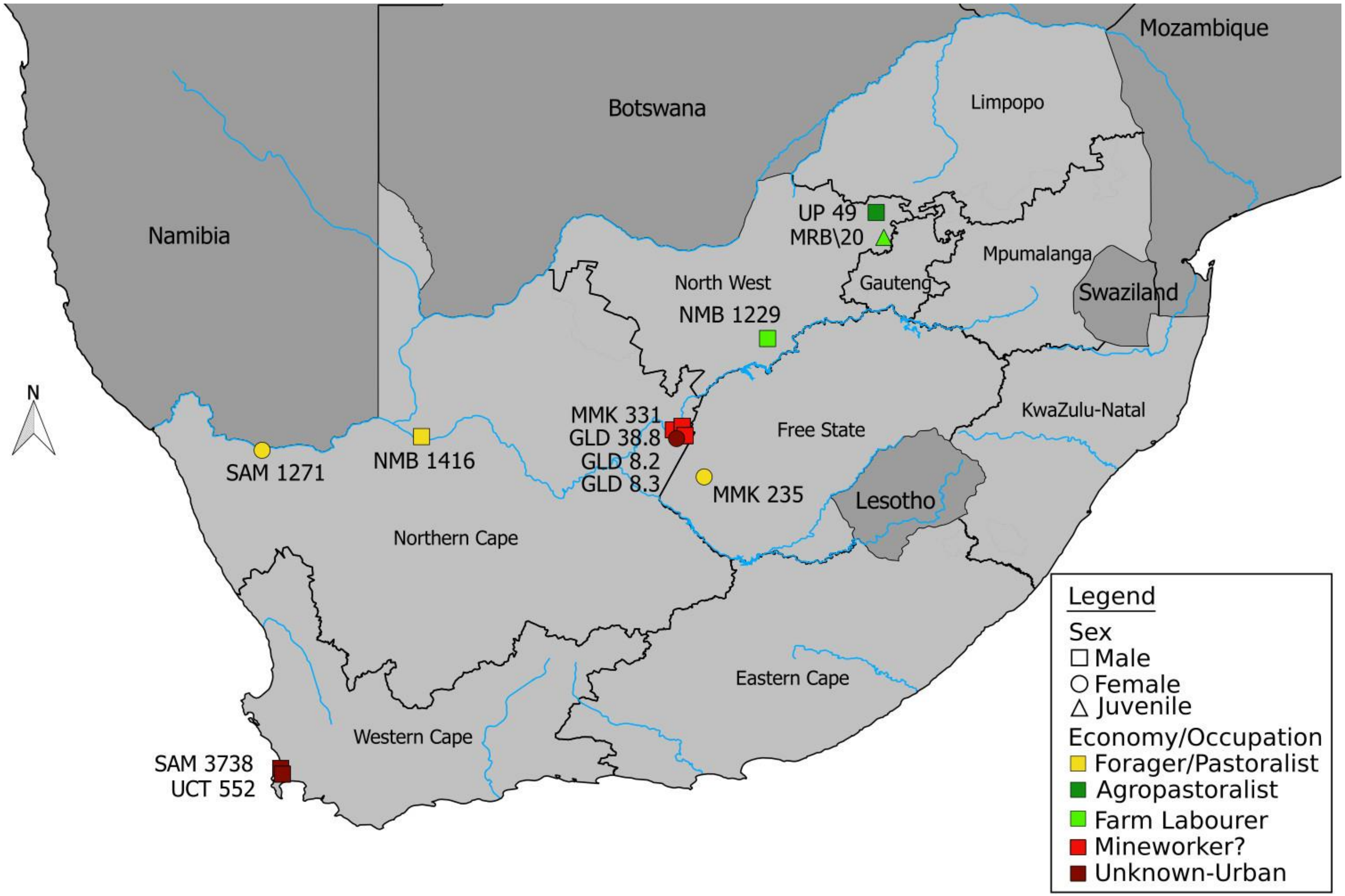


Figure 27: Map of South Africa showing the burial location of possible tuberculosis cases.

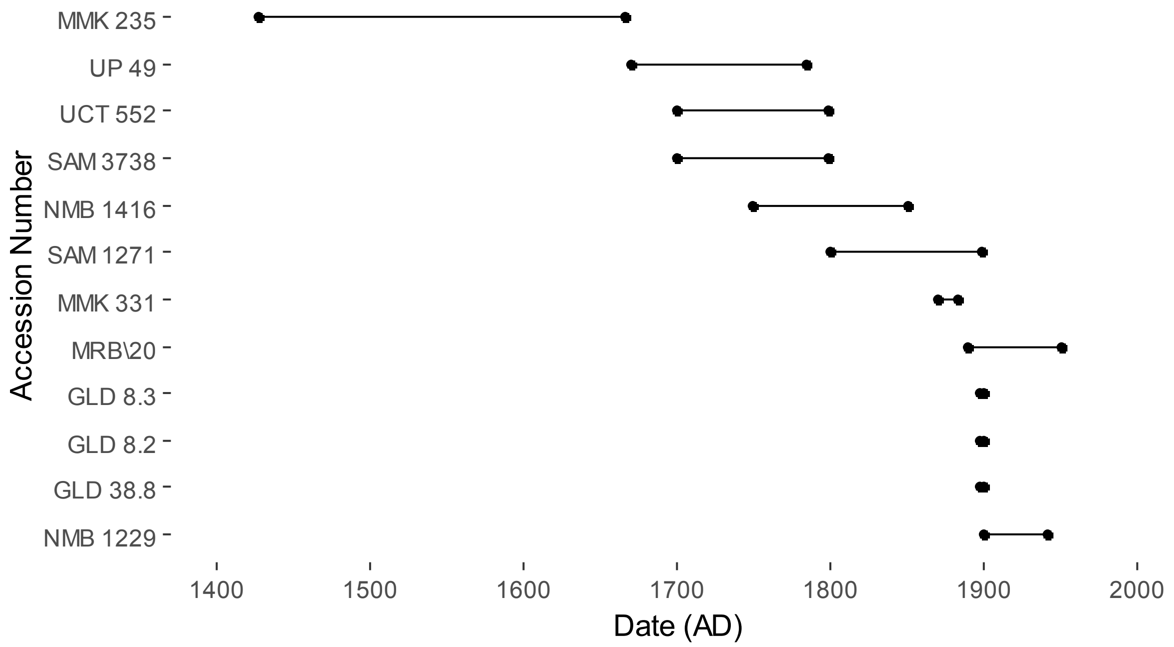


Figure 28: Chronological dates for possible tuberculosis cases.

DETAILED PROFILES

Detailed descriptions of burial context, skeletal preservation, demographic information and pathology are provided below.

MMK 235

MMK 235 is housed at the McGregor Museum, Kimberley. Information from the catalogue identifies the individual as a Korana female, accessioned in 1930 and excavated by W. Fowler. The grave was located 2 miles east of Koffiefontein and was described as a “shallow round grave”. The individual was buried in a “crouched position” and a number of associated grave goods were reported which include one stone within the shaft, a bored stone, ostrich egg-shell beads and a small pot. Humphreys (1970) reports on this individual and provides additional information not currently present in the catalogue. He describes the grave location as “¼ mile north of the [Riet] river” (pp. 105) and further describes the grave as having “stone on top with flat grinder” (pp. 105). Morris (1992) and Vogel and Fuls (1999) report an uncalibrated radiocarbon date for this individual as 390 ± 50 BP (Pta-2894). This produces a calibrated date of AD 1428 to AD 1666 (at 99.7% probability) using the SHCal13 atmospheric curve (Hogg et al., 2013) on OxCal (v4.3.2, Ramsey, 2017). Morris (1992) notes that this individual can be associated with the type-R settlements in the vicinity and that the people residing in these settlements had a predominantly hunter-gather economy with some pastoralist elements.

The skeleton is relatively complete and most skeletal elements are present. The skull, vertebral column and sternum are present and well preserved. There is some post-mortem damage to the ribs. Eight left and seven right ribs are present and most are incomplete/fragmented. The pectoral and pelvic girdles are complete and well preserved, as are the long bones of the appendages. There is some post-mortem damage to the proximal epiphysis of the right radius. Two carpals are absent from each of the left and right sides and no hand phalanges are present. A single left tarsal is absent and only one foot phalanx is present. Skull and pelvic features indicate MMK 235 is an adult female, probably between the age of 35 and 49 yrs.

The lesion of interest is present on the right scapula (Figure 29) and involves abnormal bone loss (predominantly) of the non-articular surfaces immediately inferior and ventral to the glenoid fossa and along the dorsal margin of the articular surface. Four small “openings” are present, two of which are located on the ventral surface (Figure 29c). The superior-most opening is small (<0.5 cm in diameter), circular and located ~2 cm inferior to the coracoid process and ~1 cm lateral to the glenoid fossa. Margins are well defined and sclerotic. The inferior opening is larger (0.4 cm by 0.6 cm), roughly oval and is located just anterior to the inferior aspect of the glenoid fossa. This opening has a broad, somewhat scalloped, depressed region extending medially and superiorly. There are also patches of woven bone present along this depressed region. The margins of the opening are smooth, well defined and sclerotic. The third opening is located ~1 cm inferior to the glenoid fossa along the lateral border (Figure 29b and d). This opening is oval shaped, 0.9 cm by 0.5 cm, with woven bone addition to the dorsal wall of the opening. Margins are smooth, well defined and sclerotic. There is also a 0.5-1 cm region abnormal woven-lamellar bone just ventral to the inferior opening. The fourth and final opening is present on the dorsal inferior margin of the glenoid fossa involving both the articular and non-articular surfaces (Figure 29a and b). The

Figure 29: MMK 235 – Pathology of the right scapula and right sacroiliac joint. Lytic lesion(s) are present on the right scapula a) dorsal view, b) lateral view, c) ventral view, d) inferior view. e) Partial ankylosis of the right sacroiliac joint. Scale bar = 1 cm.

lesion is small, 0.4 cm in diameter and circular. The primary lesion(s) involve abnormal loss of bone with only minor formation of woven to intermediate bone. All internal margins are sclerotic and well defined indicating a chronic process. There is also a slight increase in porosity of the bone surrounding the lesion, particularly the inferior and ventral lesions suggesting an increase in blood flow to the pathological region. It was not possible to observe the internal surface of the lesion entirely but at least three of the openings are confluent which may indicate a single focus. The fourth and superior-most opening on the ventral surface may also communicate with the others but this could not be observed; alternatively, this could represent a separate focus. There is minor osteophytic lipping of the glenoid fossa, particularly along the ventral margin and some slight irregularity of the superior articular surface; however, no pathological changes were observed on the humerus.

In addition to the scapula lesion, this individual also has partial ankylosis of the right sacroiliac joint along the superior and dorsal surfaces (Figure 29e). The internal surfaces were not visible but there is no evidence of trauma or infection and this is not likely associated with the scapula lesion. A lumbosacral cranial border shift is also observed, with complete sacralisation of the 5th lumbar vertebra resulting in 4 lumbar vertebrae and 6 fused sacral elements.

MMK 331

MMK 331 is housed at the McGregor Museum, Kimberley. The individual was unearthed in 1974 near/at the police station in Transvaal Road, Kimberley. A note in the box authored by A. Humphreys adds that the burial was discovered during the excavation of a drainage trench. The remains were located 1m below the surface and were removed by builders. Humphreys notes that the grave had been disturbed by earlier turn-of-the-century trench-works and that no grave goods were recovered. No information regarding burial position is provided. It is likely that this individual was unearthed from an African burial ground, an extension of the historic Pioneers Cemetery (David Morris, personal communication), and dates from 1870 (the diamond rush) to 1883 (when the new cemetery, Gladstone, was established) (Morris, 2004).

The remains are poorly preserved. The anterior right cranium is present and articulated. Elements present include the frontal, sphenoid, maxilla and part of the parietal. On the left side only the maxilla and part of the frontal are present and these are not in articulation with the rest of the cranium. The mandible is also present and complete. Some of the cervical (n = 4) and thoracic vertebrae (n = 10) are present and these show good to moderate preservation but the remainder of the vertebral column was not recovered. Nine left ribs are present and these included the 1st and 11th ribs. While on the right side, 6 ribs are present including the 12th rib. These are all moderately preserved (between 25-75% of the skeletal element is present). No other skeletal elements are present. Three additional bags containing small mixed cranial and post-cranial fragments, teeth and non-human bones were also present. Based on the partial cranium, this individual was probably female. Age-at-death could not be refined further than adult (>20 yrs).

Abnormal bone formation is present along the pleural surface of three left and three right ribs (Figure 30a). The identification of rib number is complicated by poor preservation. The abnormal bone consists of broad, continuous sclerotic areas focused at the angle and middle third of the rib shaft. These sclerotic areas transition into areas of small 1-3mm nodules towards the vertebral end of the rib. The sclerotic areas often have a pattern of diagonal straie on part of the surface. These lesions suggest a healed periosteal reaction affecting the pleural surface of the ribs.

There is also abnormal bone loss at the left mandibular condyle (Figure 30b). The entire condyle is absent and the remaining surface is mostly concave, porous and lamellar to slightly sclerotic with well-defined margins. There is minor “lipping” along the margin of the lesion. There is some articular surface irregularity and a small focal region of pitting (3-4mm) on the medial half of the right mandibular condyle. This may represent a healed fracture of the left mandibular condyle with non-union, alternatively it may be infective or neoplastic in origin. The condyle and temporal bone were not present. There are also deep Schmorl’s nodes present on the articulating body surfaces of the 7th and 8th thoracic vertebrae. These lesions are multiple, deep pits located centrally on the vertebral bodies.

Figure 30: MMK 331 – Pathology of one right rib and the mandible. a) Sclerotic new bone formation on the pleural surface on a right rib. b) Lesion present at the left mandibular condyle. Scale bar = 1 cm

GLD 38.8

This individual and all others with the GLD prefix are housed at the McGregor Museum in Kimberley. These individuals were exhumed in a rescue excavation that followed the discovery of unmarked graves while digging a trench outside the current boundary of the Gladstone Cemetery (van der Merwe et al., 2010b). A total of 107 individuals were unearthed from 15 graves and a minimum of 27 additional individuals were rescued from the site where the material excavated from the trench was dumped. The occupants of these graves have been identified as black migrant mineworkers who died at Kimberly hospital and were buried as paupers between 1897 and 1900 (van der Merwe et al., 2010b).

The skeletal preservation of this individual is excellent. All major skeletal elements are present and well preserved. Even smaller, fragile elements that are infrequently preserved, i.e. the xyphoid, hyoid and coccyx, were also recovered. The only elements missing are a few carpals (one left and three right), metatarsals (four left) and phalanges (one hand phalanx and most of the foot phalanges). A near complete pair of hands from another individual is stored with these remains. Based on skull and pelvic features, this is a male, and age-at-death indicators suggest this individual was a middle adult (35-49 yrs).

A number of pathological changes are present on numerous skeletal elements. Lytic lesions are present on the 3rd and 4th lumbar vertebrae. Abnormal bone formation is present on the 2nd-4th lumbar vertebrae, most of the right ribs, and some of the long bones.

A relatively shallow and oval-shaped lytic lesion (1-2 cm) is located along the anterior surface of the 3rd lumbar vertebra just left of midline extending from the caudal margin to roughly midway along the height of the vertebral body (Figure 31a and b). The lesion has exposed the underlying trabeculae and the margins are well defined but not sclerotic. Abnormal bone formation is also present along the inferior half of the anterior and lateral surfaces of the vertebral body. This bone is woven or woven to lamellar and is present in irregular patches along the cortical surface. The abnormal bone formation surrounds the lesion and extends along the inferior border laterally. Another lytic lesion is present along the superior ⅔ of the anterior surface, extending onto the cranial articular surface of the 4th lumbar vertebral body (Figure 31a and c). The lesion is irregular in shape and is somewhat lobed/scalloped in appearance with well-defined margins but no sclerosis. There is some abnormal bone formation surrounding the lesion on the anterior surface, inferior and lateral to the lesion. This bone is porous and lamellar. There may be another lytic lesion on the inferior aspect of the anterior surface of the 5th lumbar vertebrae; however, given the irregular surfaces and poorly-defined margins, it seems more likely that this lesion reflects post-mortem damage (Figure 31a and d). While there are no lytic lesions present on the 2nd lumbar vertebra, the abnormal bone formation described for the 3rd and 4th lumbar vertebrae is also present along the lateral and anterolateral surfaces (Figure 31a). For the 2nd and 3rd lumbar vertebrae, smooth, “clean” and light coloured regions along the anterior surface suggests the presence of additional areas of abnormal bone formation that have detached post-mortem due to weak adhesion to the underlying cortical surface.

There is also abnormal bone formation along the pleural surface of the right 2nd-11th ribs (Figure 31e). These proliferative lesions are typically mixed with woven, lamellar and intermediate bone present. The bone is "puffy" and porous and grades into surrounding bone as described in (Pfeiffer, 1991) expansion type. In general, this "puffy" bone is lighter in colour than the surrounding bone; however, in some regions the bone is darker, more porous and/or has small, smooth-edged depressions. For some ribs the pathology extends along the entire plural surface but the focus appears to be at the middle of the shaft or at the vertebral end.

Abnormal bone formation was also noted on the diaphysis of the radii, tibiae and fibulae. This bone is intermediate in nature (slightly more organized than woven bone but still lightly adheres to the cortical surface) and is darker in colour than the non-pathological cortical bone. On the radii this bone is present on the anterior surface of the distal diaphysis and is covers a greater area on the left side. These proliferative lesions are present on the lateral, posterior and medial surfaces of the distal ⅔ of the tibial shaft and here the lesions span a greater area on the right side (Figure 31f). Finally, the lesions present on the fibulae are located on the posteromedial surface of the diaphyseal middle ⅓.

In addition, and unrelated, to the lesions described above, this individual also has a healed fracture of the left 1st metacarpal with volar displacement of the proximal epiphysis. The articular surface is larger and the entire metacarpal is shorter than on the left side. This may represent a Salter-Harris Type 1 physeal fracture / slipped epiphysis indicating the injury occurred prior to the fusion of the growth plate.

Figure 31: GLD 38.8 – Pathology of the vertebra, ribs and tibia. a) Anterolateral view of the lumbar vertebrae with abnormal bone formation on L2-4 and lytic lesions present on L3-4 and possibly L5. b) Small oval lytic lesion present on the anterior surface of L3. c) Lytic destruction of the anterior surface and part of the cranial surface of L4. d) Possible lytic lesion on the anterior surface of L5. e) Periosteal new bone formation on the pleural surface of one right rib (upper) with numerous small depressions at the vertebral end (lower). f) Periosteal new bone formation along the lateral surface of the right tibial shaft.

GLD 8.2

See GLD 38.8 for information about the site and general information about the individuals exhumed there.

GLD 8.2 is very well preserved and most skeletal elements are present, including the xyphoid, part of the coccyx and hyoid. The only skeletal element missing is one hand phalanx. Additional elements/items stored with this individual include eight sesamoid bones, a bag containing 7 teeth, a bag of soil, a cylinder of soil, a bag with glass shards, a bag with iron beads and a piece of foil containing fabric (possibly wound dressing). Based on skull and pelvic features this is a male, and age-at-death indicators suggest this was a middle adult (35-49 yrs).

This individual has abnormal bone formation on a number of ribs (left and right), some thoracic vertebrae and the left tibia.

Abnormal bone formation is present along the left 4th-6th and right 1st-5th ribs (Figure 32a). The sternal ½ of the pleural surface is predominantly affected but on the left 4th-6th ribs proliferative lesions are also present at the angle of the rib. These proliferative lesions are also present along the external surface of the right 2nd-5th ribs near the sternal end. Similar lesions are found along the anterior surface of the 5th-9th thoracic vertebral bodies (Figure 32b). These lesions are located near the cranial and caudal margins of the nonarticular surface of the vertebral bodies. For the rib and vertebral lesions, the bone is woven, poorly organized, lighter in colour than the surrounding bone and lightly adheres to the cortical surface. There is also abnormal bone deposition on the left tibia along the lateral surface of the proximal third of the diaphysis (Figure 32c). Similar lesions are not observed on any other long bones. There is significant post-mortem damage to the lesion making it difficult to determine whether the margin is well-circumscribed. The bone of the tibial lesion shows greater organization and the lesion consists of mixed woven and woven to lamellar bone; however, attachment to the underlying cortical surface remains weak.

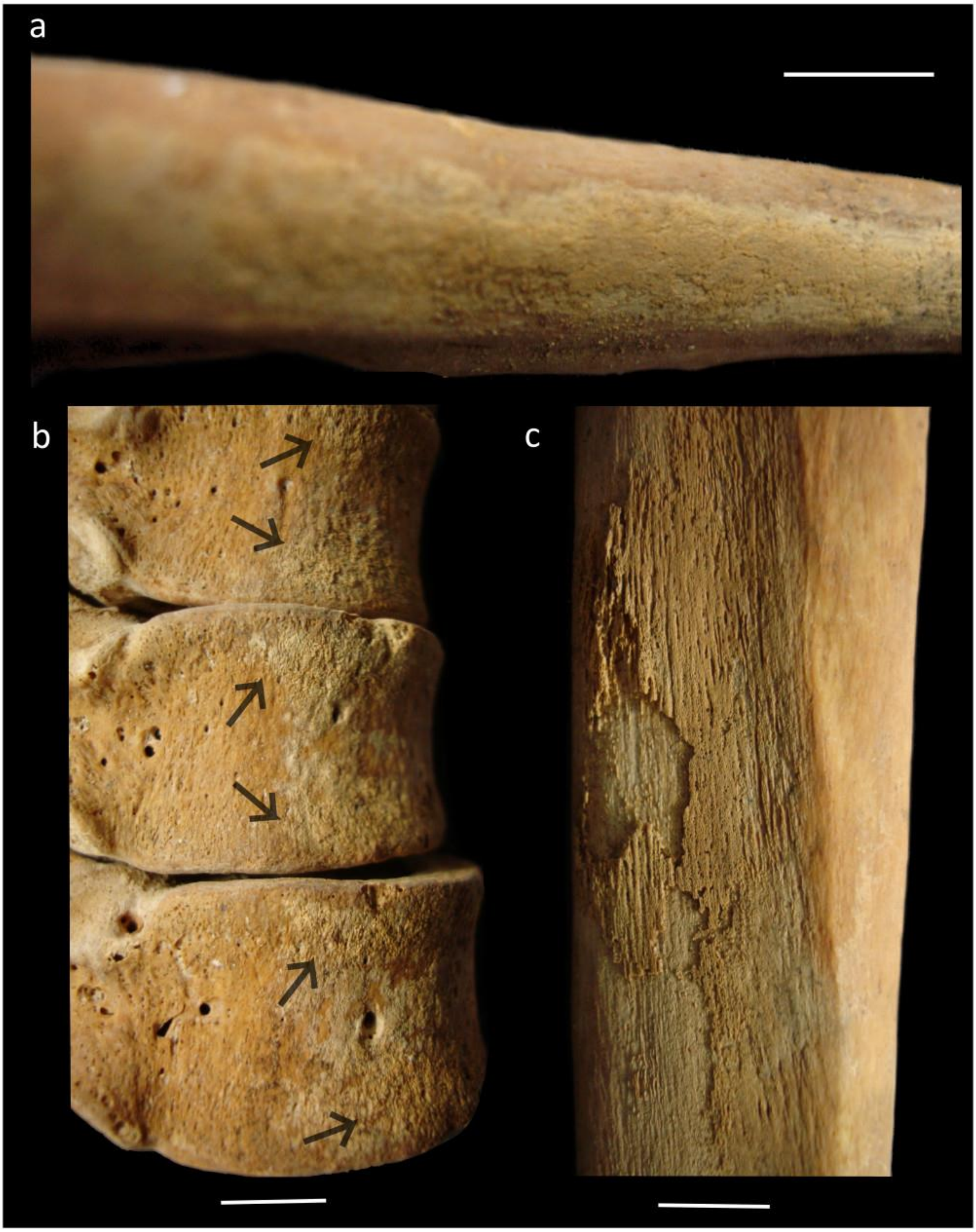


Figure 32: GLD 8.2 – Periosteal new bone formation on the ribs, vertebrae and tibia. a) Pleural surface of one pathological left rib. b) Anterolateral view of the 7th-9th thoracic vertebrae. c) Lateral surface of the left tibia. Scale bar = 1 cm.

In addition to the lesions described above there are a number of traumatic lesions and developmental field anomalies. A healed fracture is present at the left inferior facet of the

5th lumbar vertebra with non-union. The communicating surfaces are irregular and pitted with osteophytic lipping along margins. Schmorl's nodes are present on many of the thoracic and lumbar vertebral body surfaces. There is a possible healed fracture/cleft/bipartite epiphysis of two proximal foot phalanges (2nd and 3rd digits – phalanges were not sided). A somewhat linear indentation separates the left inferior 1/3rd (2nd digit) and 1/5th (3rd digit) aspect of the epiphysis (when viewed from the proximal surface with the plantar surface placed inferiorly). If these represent healed fractures, the fractures would have likely occurred prior to the fusion of the epiphyses. There are no degenerative changes noted on the articular surfaces of either phalanx. The 11th thoracic vertebra presents a case of spina bifida occulta. The cleft is located just left of the spinous process which is hypoplastic. Finally, bilateral symphalangism of the 5th digit intermediate and distal phalanges is noted.

GLD 8.3

See GLD 38.8 for information about the site and general information about the individuals exhumed there.

Of the three Gladstone individuals, GLD 8.3, while complete, has the greatest degree of post-mortem damage. The cranium is complete and articulated. The entire mandible is present but is fragmented into five parts. The sternal body is present but the manubrium and xyphoid process are not. Both os coxae are complete but the left pubis has broken off post-mortem. All vertebrae and ribs are present and well preserved. Similarly, all long bones are present and well preserved with the exception of the left femur and right fibula, both of which are broken in two at the proximal epiphysis (post-mortem). The only other absent element is a single carpal. The sacrum and the left patella were not present / available for observation but according to van der Merwe et al. (2010b) these were recovered and the patella showed pathology consistent with tuberculosis. Additional elements/fragments/material present include four sesamoids, six loose teeth, a partial hyoid and coccyx, two rib cartilage ossifications, a bag of unidentified fragments, a bag of soil, a bag of glass shards and a bag containing metal (iron) items.

Lytic lesions are present on two lumbar vertebrae and the right ulna. A large lytic lesion has destroyed just over half of the caudal surface of the 4th lumbar vertebra (Figure 33a and b). The lesion is C-shaped and is 1-2 cm in width and relatively deep, resulting in the destruction of ⅓-½ the height of the vertebral body. The lesion is bound by the margin along the left lateral side but opens onto the anterior surface of the vertebral body just right of midline, whereafter it tapers in width slightly as it extends along the right lateral surface ~0.2-0.3 cm from the margin. The posterior third of the vertebral body is unaffected. The lesion surface has a slightly scalloped or lobed appearance. The surface appears porous and well-defined but not sclerotic; however, destructive sampling revealed a 0.1-0.5 cm zone of dense sclerotic bone bordering the lesion internally (Figure 33e). There is moderate to severe osteophytic lipping of the caudal margin and minor to moderate lipping of the cranial margin. The lesion extends to ~1 cm opening on the anterior surface and is bordered by a region of abnormal bone formation. This bone is porous near the lesion margin but otherwise well-integrated into the surrounding cortex.

There is no corresponding lesion on the cranial surface of the 5th lumbar vertebra; however, there is a region of increased porosity along the anterolateral third of the cranial surface (Figure 33d). This porosity is most pronounced anteriorly, dissipating as the lesion extends laterally. There is, however, a very similar lytic lesion present on the caudal surface (Figure 33c). This C-shaped lytic lesion resulted in the destruction of the left lateral third of the caudal surface. The lesion is similar in shape and appearance to the lesion on the caudal surface of the 4th lumbar vertebra but differs slightly in location. The 5th lumbar is broadest and deepest posteriorly, extending from the midline of the posterior surface, along the left lateral margin and tapering slightly before terminating at the midline of the anterior margin. The lesion surface here is similar to that described for the lesion on the 4th lumbar vertebra but the posterior surface does appear sclerotic with increased porosity anteriorly. There is also minor to moderate osteophytic lipping along the cranial and caudal margins for this vertebra. Abnormal bone deposition is present on the anterior surface of the vertebral body. This bone results in an irregular surface commonly seen in chronic osteomyelitis.

A third lytic lesion is present on the right ulna (Figure 33f and g). This lesion is located on the proximal surface of the olecranon and consists of two relatively deep lytic depressions that affect the entire proximal surface. The lateral depression is larger (1.8 cm long and 0.9 cm wide) than the medial depression (1.2 cm long and 0.6 cm wide) and both are deeper posteriorly. The lesion surfaces are porous, well-defined but not sclerotic. Additional lytic activity is present on the medial surface, just adjacent to the articular surface. Here the lytic destruction is more superficial creating a slightly irregular and slightly sclerotic surface. Minor osteophytic lipping is present at the proximal articular surface.

van der Merwe et al. (2010b) reports that the left patella also shows pathology consistent with tuberculosis, but a description is not provided and the patella was not available for inspection in this study. Similarly, the sacrum, while recovered, was also not available for inspection. These were in the possession of van der Merwe. If the patella lesion is lytic in nature, at least four foci are suggested by these similar pathological lesions.

Abnormal bone formation is present on the left femur and tibia. A fine layer of woven bone is present in small concentrated patches on the diaphysis of the left femur (medial surface of the middle third) and tibia (anterior surface of the proximal third). The precise distribution of these lesions may be complicated by the post-mortem destruction of this pathological bone, which only weakly adheres to the underlying cortical surface.

In addition to the pathology described thus far, there are a number of likely unrelated pathologies including trauma, developmental field anomalies and degenerative joint disease. A healed fracture of the right 3rd metatarsal is present (Figure 33i). The fracture occurs at the distal third of the diaphysis and malunion has resulted as a consequence of a slight translation and angulation of the distal fragment. Acroosteolysis of the left hallux distal phalanx is present (Figure 33h). This narrowing of the shaft and tuft has many aetiologies including trauma, which may be likely given the fracture present on the 3rd metatarsal; although, this fracture occurs on the opposite foot. There are some degenerative changes on the proximal phalanx of the left hallux. The proximal articular

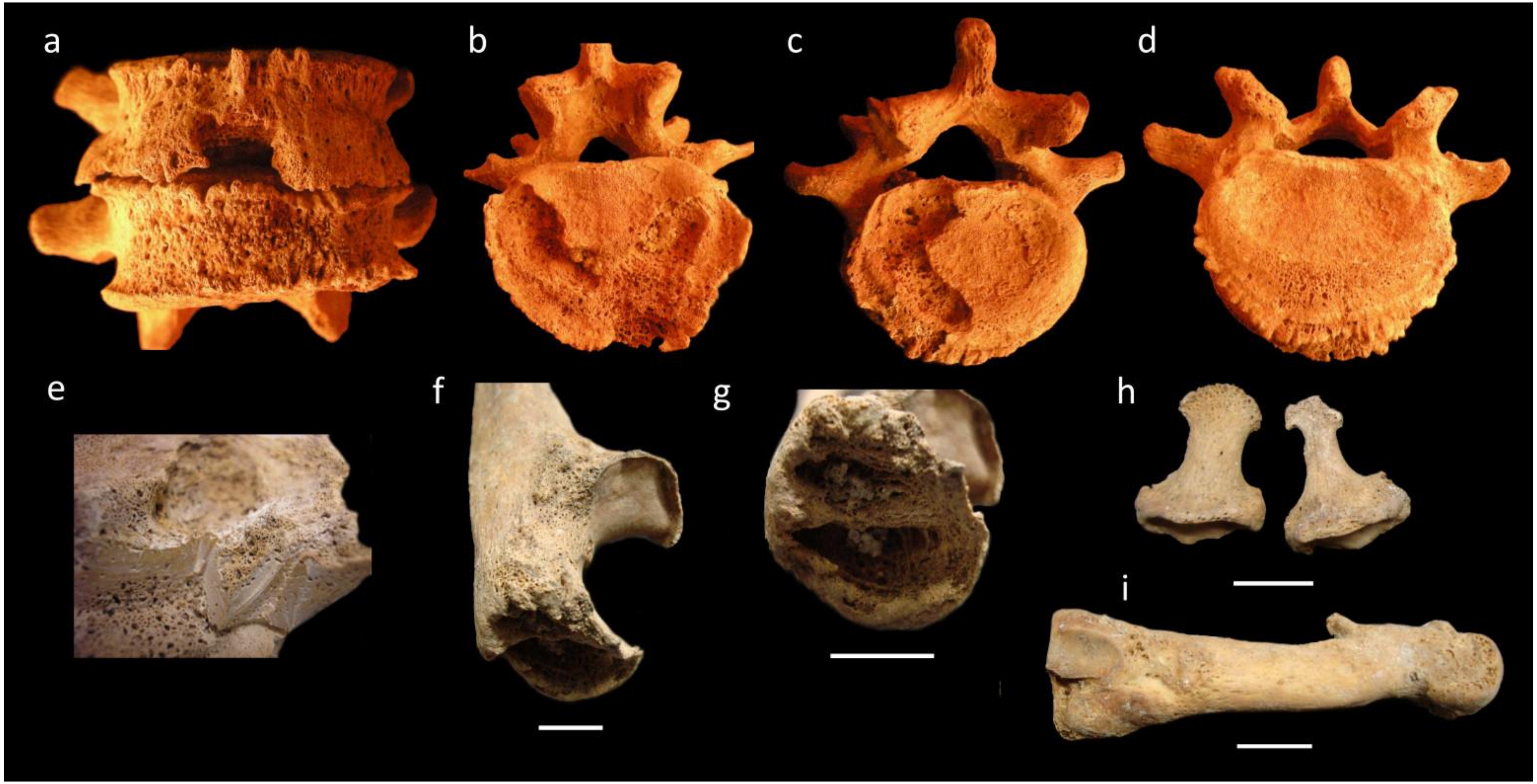


Figure 33: GLD 8.3 – Pathology of the vertebrae, ulna, metatarsal and phalanx. a) Abnormal bone formation along the anterior surfaced of L4 and L5. b) Caudal view of a lytic lesion that has destroyed the anterior and lateral aspects of the articular surface of L4. c) Caudal surface of L5 showing lytic destruction of the left lateral aspect. d) Porosity along the anterior aspect of the cranial surface of L5. e) Zone of sclerotic bone bordering the internal surface of the lesion of L4. f) Lytic activity along the medial surface of the right ulna adjacent to the proximal articular surface. g) Proximal view of the lytic lesion present at the olecranon on the right ulna. h) Acroosteolysis of the left hallux distal phalanx (right) compared to its normal counterpart (left). i) Healed fracture of the right 3rd metatarsal. Scale bar = 1 cm.

surface is irregular and lipped along its margin. The right hamulus is completely separate from the rest of the hamate. This may represent an os hamuli proprium/bipartite hamate or a healed fracture with non-union. There are no degenerative changes noted on any of the hand articular surfaces. Since there is no evidence of trauma to either of the hands, a congenital aetiology may be more likely. This individual also has an elongated styloid process of the left temporal (~5 cm long). The styloid process is completely separated from its base. This may represent a healed fracture with non-union or a segmented calcification of the stylohyoid chain with pseudoarticulation. Bilateral symphalangism of the intermediate and distal phalanges of the 5th pedal digit is also observed. Changes to the articular surfaces of a few elements are noted and these include all or a combination of the following features: lipping, surface irregularity and pitting. The affected skeletal elements include the proximal surface of the left hallux proximal phalanx, both mandibular condyles, both temporal glenoid fossae, both 12th vertebral heads, both clavicle sternal ends, both acromial processes of the scapulae and the auricular surfaces of the ilia.

NMB 1229

This individual was exhumed in 1942 by A.C. Hoffman and is currently housed at the Florisbad Quaternary Research Station of the National Museum Bloemfontein. The individual was exhumed from an informal burial ground belonging to African farm workers (Hoffman, 1942). The burial ground was situated on the farm, Kareeboom, in Wolmaransstad but was used by workers residing in the general vicinity. The burials likely date to the early 20th century (Peckmann, 2002).

Most skeletal elements are present and relatively well preserved. The left pubis is absent and there is post-mortem damage to the left ischium. The right pubis is present but was separated from the remaining os coxa post-mortem. All cervical vertebrae are present but only two thoracic vertebrae (1st-9th) and the 5th lumbar vertebra are present. A fragment of the manubrium is all that was recovered of the sternum. Six left ribs (including the 2nd rib) and seven right ribs (including the 1st and 2nd ribs) were present. All long bones were present and most were complete but there was some post-mortem damage to the left humerus proximal epiphysis, the left ulna distal epiphysis, left femur distal epiphysis and right fibula proximal epiphysis. Of the right hand, one carpal and one metacarpal are absent and one unsided hand phalanx is absent. One left and three right metatarsals are absent, in addition to half of the pedal phalanges. Otherwise, all of the remaining skeletal elements are present and well preserved. Based on skull and pelvic features, this individual is a male, and age-at-death indicators suggest this was a middle adult (35-49 yrs).

There is severe lytic destruction of the auricular surface of the right ilium and sacrum, as well as the symphyseal face of the right pubis (Figure 34). A large and multi-lobed lytic lesion has resulted in the complete obliteration of the auricular surface (no normal articular surface remains) of the right ilium (Figure 34a). In addition to the general lytic destruction of the joint surface, there are three depressions. The largest of these is located at the apex and is ~2.5 cm in diameter. This lesion is relatively deep and contains a possible sequestrum at its superior half. The other two depressions are smaller (<1 cm). The bone is porous and appears well-defined but not sclerotic; however, destructive sampling revealed a thickening and densification of the trabeculae and cortex internal/adjacent to the lesion. There is no visible reactive bone on the external surface. A new/additional articulation with the sacrum has formed at the superior posterior iliac spine. This small, oval "facet" is slightly raised above the normal cortical surface.

The auricular surface of the right sacrum shows near complete obliteration of the articular surface (Figure 34c). Only ~2 cm of the inferiormost surface remains. There does appear to be some post-mortem damage of the pathological bone, particularly along the anterior surface of the lesion. There are two deep depressions within the pathological region. The larger superior depression corresponds with the depression at the apex of the ilium and the smaller inferior depression roughly corresponds with the smaller posterior lesion on the ilium. A new point of articulation is present on the lateral side of the lateral crest. A smooth depressed region is present along the superior and posterior aspect of the lesion. This depressed region is bordered on both sides by a ridge of bone and may represent a sinus tract. A smooth depression is visible on the anterior surface of the 2nd sacral body (Figure 34d). At the inferior aspect of this lesion the cortex has thinned revealing some of the underlying trabecular bone. This depression is bordered on the right lateral surface by a slight ridge which terminates at the S1-S2 right anterior sacral foramen. Small patches of woven bone are visible along the margins of this foramen. The 1st sacral element body may also be slightly depressed but this assessment is complicated by post-mortem damage. The depression located on the anterior sacral surface may represent a pressure erosion caused by an overlying mass (possibly an abscess or sinus). Most of the thoracic and lumbar vertebrae were absent; however, no pathology was observed on the 5th lumbar vertebra.

There are also pathological changes noted on the right pubis, although the full extent of the pathology is difficult to ascertain due to post-mortem damage (Figure 34b). There may be some lytic destruction of the articular surface. What remains of the surface appears porous, slightly sclerotic and does not resemble normal articular surface. There is also some abnormal bone formation along the ventral surface adjacent to the symphyseal face. There may be a lytic lesion inferior to the symphyseal face. Some of the exposed trabeculae and the ventral margin of the lesion do appear smooth and well-defined; however, there is also clearly post-mortem damage reducing confidence in the veracity or extent of this lesion. There are also patches of woven bone along the dorsal surface of the pubic body. The left pubis was not present.

The skeletal elements of this individual feel abnormally light in weight. This could indicate disuse osteopenia possibly as a result of impaired mobility due to the pathology at the pubic symphysis and right sacroiliac joint. There is also a significant amount of alveolar resorption of the mandible and maxilla exposing ~½ to ⅔ of the tooth root (Figure 35). This could be linked to periodontal disease. A possible periapical granuloma is associated with the left mandibular 1st molar. There is greater alveolar resorption in this region with a smooth, rounded, sclerotic alveolar surface and the tooth has been glued in place. There is also calculus present on most teeth.

Figure 34: NMB 1229 - Lytic destruction and associated pathology of the right ilium, pubis and sacrum. a) Lytic destruction of the auricular surface of the right ilium. The white arrow indicates a new surface of articulation with the sacrum. b) Lytic destruction of the right pubic symphyseal face with abnormal bone formation along the inferior half of thee ventral margin and a possible lytic lesion located inferiorly. c) Lytic destruction of the superior aspect of the sacral auricular surface. The white dashed line indicates the

path/presence of a possible sinus/abscess. The white arrow indicates a new surface of articulation with the ilium. d) Anterior surface of the sacrum showing slight depression of the S1-2 bodies. Black arrows indicated ridge bordering this depression inferiorly and laterally. Scale bar = 1 cm.

Figure 35: NMB 1229 - Significant alveolar resorption of the mandible and maxillae. Some calculus is visible on most teeth and a possible abscess/granuloma is associated with the left 1st mandibular molar. Scale bar = 1 cm.

Degenerative changes were noted on some articular surfaces. This includes minor pitting of the styloid process of the right 3rd proximal metacarpal, as well as osteophytic lipping of the articular surfaces of the 1st cervical vertebra dens facet, the dens of the 2nd cervical vertebra, and the glenoid fossae of the scapulae. A unilateral instance of os acromiale of the left scapula is also noted.

NMB 1416

This individual was exhumed in 1936 by T. F. Dreyer and A. J. D. Meiring from a grave along the south bank of the Orange River on the farm, Rooipad, Kakamas and is curated at the Florisbad Quaternary Research Station, Bloemfontein. The grave is described as isolated, large, round and 0.5m deep. The individual was buried in a flexed position and stones and ash were reportedly present in the grave shaft (Morris, 1992a, 1992b). These remains and others located nearby are believed to belong to Khoekhoen individuals of the Einiqua and date to the late 18th and/or early 19th centuries (Morris, 1992b). Morris' (1992b) analysis of the Kakamas individuals indicates an admixed population sharing morphological affinities with both Khoesan and Bantu-speaking peoples.

The skeletal remains are relatively complete and well preserved. The cranium is complete and articulated. The mandible is missing the right condyle but is otherwise complete. The left patella is absent. There is some post-mortem damage to the body of the 3rd lumbar vertebra and the right 1st rib is absent but all other vertebrae and ribs are present and well preserved. There is some post-mortem damage to the right humerus distal epiphysis (which has separated from the rest of the element) and the left fibula proximal epiphysis. A post-mortem break of the right radius is observed at the proximal third. Most hand bones are absent. Only four right metacarpals and five hand phalanges were recovered. The feet are slightly better preserved. Only one tarsal and most (n = 18) of the pedal phalanges are missing. The loose teeth are stored in a small box and a left zygomatic bone from another individual was also present. All other skeletal elements are present and well preserved. Skull and pelvic features indicate this individual is male and death occurred between the ages of 35 and 49 years (middle adult).

Abnormal bone formation is present on the pleural surface of the left 2nd -11th ribs (Figure 36a and b). The bone is mostly woven or woven to lamellar and is weakly adhered to the cortical surface. The 2nd to 4th ribs have the greatest extent of new bone formation - extending more or less along the entire pleural surface of the ribs. The remaining ribs have smaller patches of new bone formation distributed mostly along the vertebral end and angle. There are regions where the bone is slightly more organized and better integrated into the cortical surface and these are located on the 4th to 7th ribs, particularly. There is no pathology on the right ribs or vertebrae.

This individual also has complete ankylosis of the right carpals, 2nd-5th metacarpals, distal radius and ulna (Figure 36d and e). A bony bridge connects the ulna and radius at the distal ¼ of the diaphysis and likely represents partial ossification of the interosseous membrane. There is a healed fracture of the distal ulna in the region of the ossification. A fracture line is visible at the medial aspect of the dorsal surface and there may be a slight angulation of the distal fragment. The ankylosed bone appears smooth and integrated on the dorsal surface but on the palmar surface the bone is dense, sclerotic and slightly irregular. This dense, sclerotic and irregular bone is also present along the dorsal surface of the ulna.

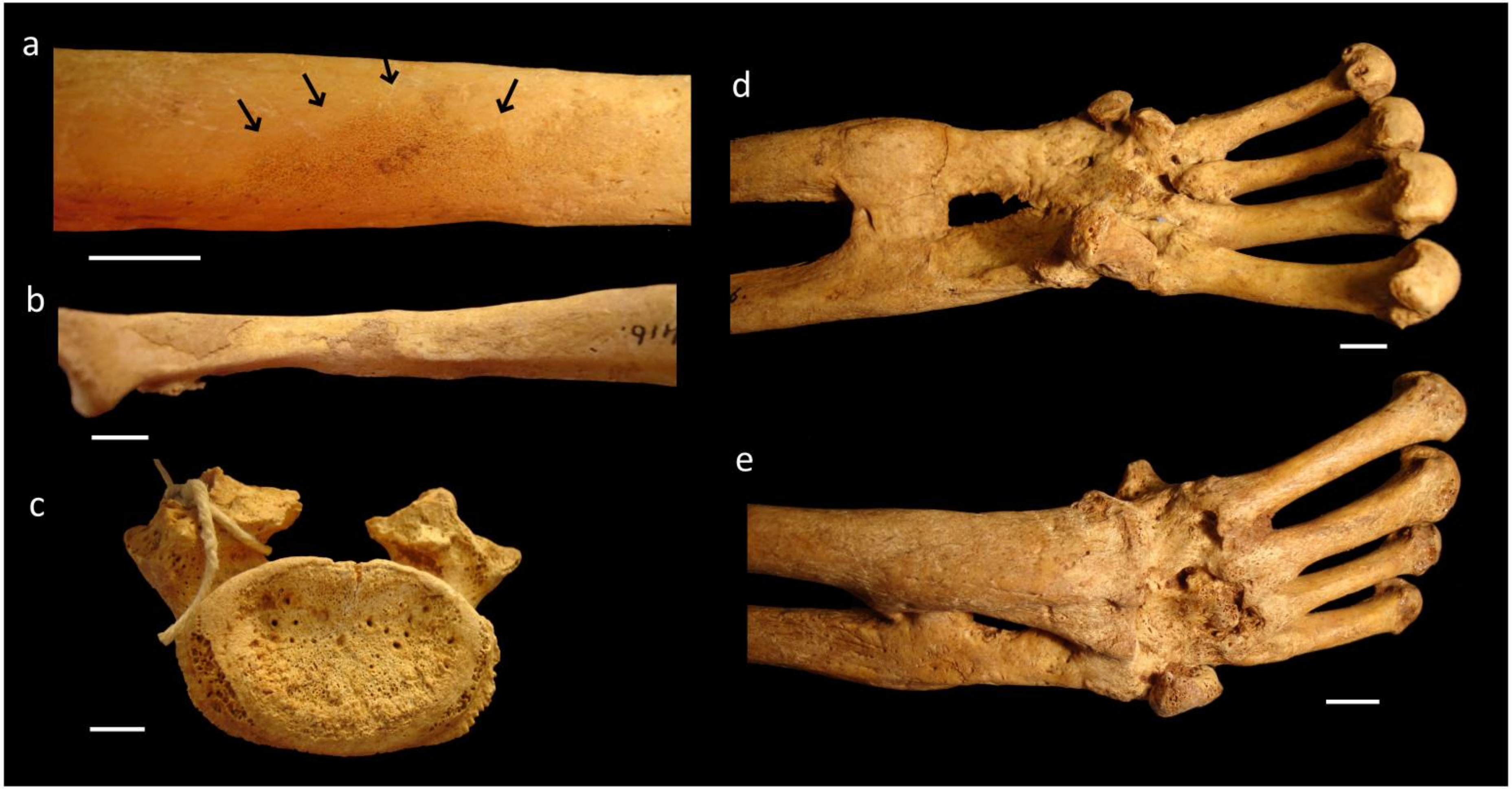


Figure 36: NMB 1416 – Pathology of the ribs, vertebra and right wrist. a) Abnormal periosteal bone formation on the pleural surface of a left rib shaft, with b) similar changes noted at the vertebral end of another. c) Bilateral spondylolysis of L5. d) Palmar view and e) Dorsal view of the ankylosis of the right carpals, 2nd-5th metacarpals, distal ulna and radius. Scale bar = 1 cm.

Furthermore, numerous, small bony spicules are observed between the radius and ulna between the bony bridge connecting the elements and the fused wrist. (Morris, 1995) suggests this is septic arthritis. There are no visible lytic lesions. Based on the stage of healing, this pathology appears to predate the rib lesions and, given the fracture present at the ulna, is most likely linked to trauma.

There is also bilateral spondylolysis at the pars interarticularis of the 5th lumbar vertebra (Figure 36c). Minor lipping and pitting is observed along the lateral margins of the vertebral body caudal surface. Degenerative changes are also noted on other vertebral body articular surfaces. Pitting is noted on some of the lumbar vertebrae and minor lipping on some of the thoracic vertebrae. Schmorl's nodes are also observed on some lower thoracic vertebrae. Small focal regions of surface irregularity are also noted on the glenoid fossae of the scapulae.

SAM-AP 1271

This individual was donated/exhumed in 1911 by F. C. Shrubsall and E. Pittard and is curated by the Iziko South African Museum in Cape Town. The burial was located in Henkries, Namaqualand and the remains were "lying extended" (Morris, 1992a). Associated grave goods include a grave stone, as well as glass and sandstone beads. Information present on the skeletal elements states this is a "Hottentot female". Given the burial position (extended burials indicate a Christian influence) and date of accession, this individual probably dates to the 19th century.

The remains are only partially complete and show moderate preservation. Most cranial elements, while present, have been damaged post-mortem and the cranium is fragmented. The only cranial elements not recovered are the left temporal and both sphenoids. The mandible is present but only part of the left side and most of the right side is preserved. Both clavicles are present and well preserved; however, the scapulae show moderate post-mortem damage. Vertebral preservation is incomplete. The vertebral bodies of the 1st cervical and three 1st-9th thoracic vertebra are not present, nor are the neural arches of one 3-6th cervical vertebra, six 1st-9th vertebrae, and the 10th thoracic vertebra. The sacrum is present and well preserved. The manubrium and sternal body are present but the body has been damaged post-mortem. Most ribs are present but preservation is generally poor. The 2nd left rib, two left 3rd-10th ribs and three right 3rd-10th ribs are absent. Both os coxae are present but both ilia show post-mortem damage and most of the right pubis is absent. The patellae are both present and well preserved. The large long bones of the limbs are all present but most are fragmented and many epiphyses are absent. Specifically, the proximal epiphyses of both humeri, and the distal epiphyses of the left radius, right ulna and right femur are all absent. The bones of the hands and feet are mostly present and well preserved, with the exception of the phalanges. Only four carpals are absent, in addition to six manual and twelve pedal phalanges. Based on the features of the skull and pelvis, this individual was a female; age-at-death indicators suggest an old adult (>50 yrs).

A large lytic lesion is present on the caudal surface of the 4th lumbar vertebra (Figure 37a and b). The lesion is c-shaped and follows the lateral and anterior margins of the caudal surface, extending onto the lateral and anterior (nonarticular) surfaces as well. The lesion is broadest anteriorly (~2 cm) and tapers as it extends laterally affecting ~⅔ of the caudal surface. While the anterior and lateral borders of the lesion are smooth, the posterior margin is irregular and characterized by a number of smaller pits or scallops. The lesion surface is generally smooth and slightly sclerotic, although the underlying trabeculae revealed by the lytic destruction are still visible. Similar to the other lytic lesions described above, destructive sampling revealed a zone of sclerotic bone internal to the lesion (Figure 37c). There is some abnormal, sclerotic bone formation along the anterior and lateral surfaces of the vertebral body bordering the lesion. There is significant post-mortem damage to the cortical surface of the 4th lumbar cranial surface but the exposed trabeculae appear normal. No pathologic changes are observed on the cranial surface of the 5th lumbar vertebra; however, abnormal bone formation is present on the anterior surface. An oblique ridge of bone is present on the left side of the anterior surface and on the right side, a bony projection extends anteriorly. These may represent entheseal ossifications of the anterior longitudinal ligament.

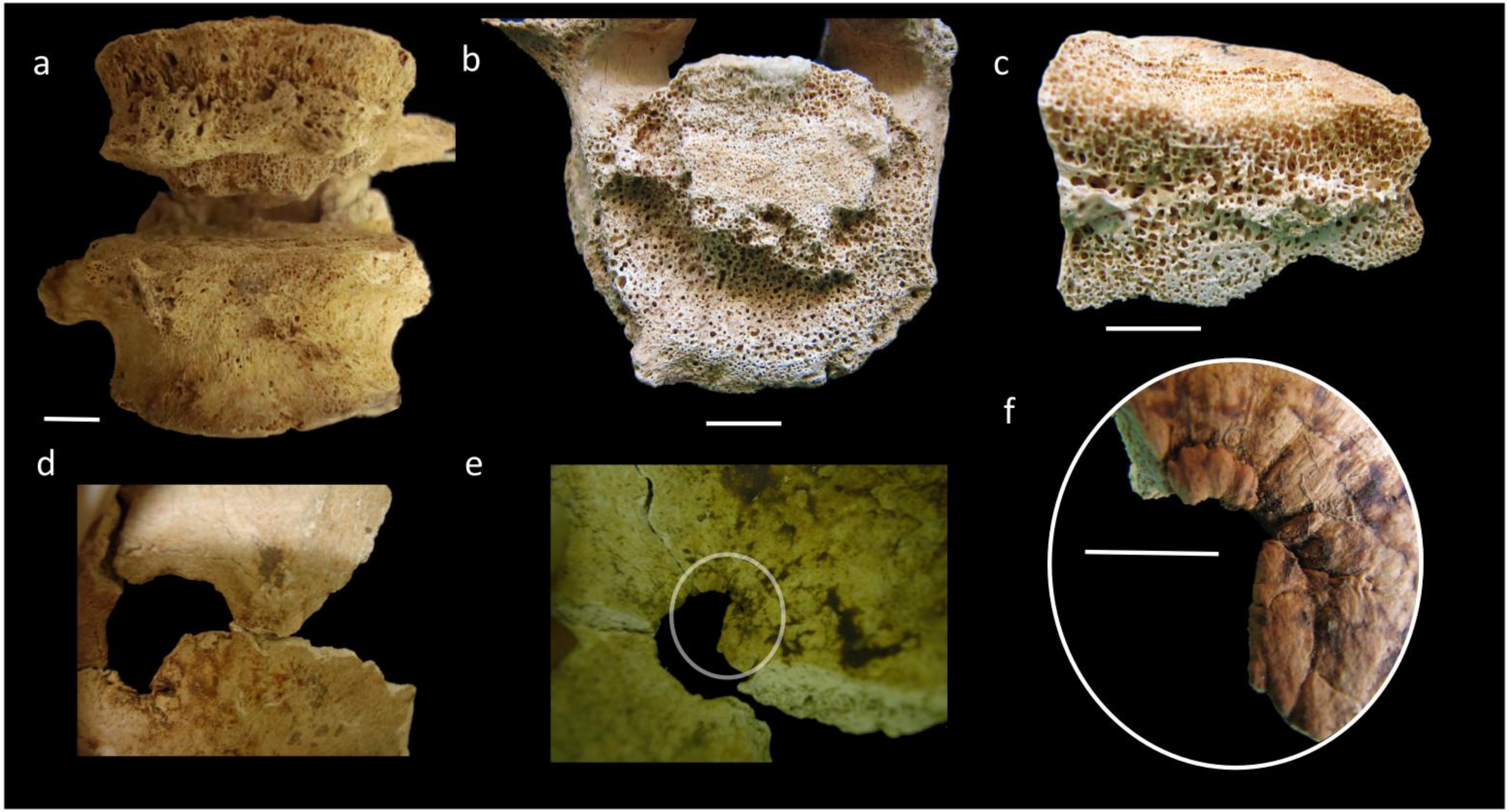


Figure 37: SAM-AP 1271 – Pathology of the vertebra and right parietal. a) Abnormal bone formation on the anterior surfaces of the 4th and 5th lumbar vertebrae. b) Lytic destruction of the anterior and lateral aspects of the caudal surface of the L4. c) Sclerotic bone bordering the lesion internally (lesions surface is located inferiorly). Healed trauma to the right parietal visible on the d) external and e) internal tables. f) Close-up of the internal table showing two fragments that had dislodged during the initial trauma. Scale bar = 1 cm.

In addition to the lytic lesions present on the 4th lumbar vertebra, a healed penetrating trauma is present on the right parietal in the region of the eminence (Figure 37d, e and f). This roughly circular aperture is ~2 cm in diameter with rounded, smooth and sclerotic margins. On the internal table, striae are visible radiating from the aperture. In addition, at least two small fragments had separated from the internal table at the margin of the aperture. Post-mortem damage has separated the right parietal and, consequently, the pathological lesion into three fragments. This pathology is consistent with impact by a high-velocity projectile.

SAM-AP 3738

This individual is housed at the Iziko Museum South Africa and was unearthed from Prestwich Street in Green Point, Cape Town. No additional information is available concerning burial position, date of accession or excavator/donor. In 2003 a large number of human remains were uncovered during earthmoving activities associated with the construction of a new development on Prestwich Street (Finnegan et al., 2011). These individuals, as well as those uncovered in the nearby Napier and Cobern Streets, all form part of an informal burial ground that existed outside the boundaries of the known formal cemeteries in the area (Apollonio, 1998; Cox, 1999; Cox et al., 2001; Finnegan et al., 2011). This individual was not exhumed as part of the highly controversial 2003/2004 Prestwich Street rescue excavations, however it may belong to the same informal burial ground, which was active predominantly during the 18th century (Apollonio, 1998). Cox et al. (2001) estimate a more specific range, indicating the burial ground was in use between 1750 and 1827. The burial ground is thought have been use by the lower class of the Cape Colonial society and/or those who could not be buried in consecrated ground including “paupers and slaves, “free blacks” and seamstresses, domestic servants and hawkers, suicides and the executed, and victims of shipwrecks and epidemics” (Finnegan et al., 2011, p. 138).

The remains of this individual are relatively complete and well preserved. The complete and articulated cranium and mandible are present and well preserved. There is some post-mortem destruction of both scapular bodies. There is post-mortem damage to the right ilium and pubis and the left pubis is mostly absent. One cervical vertebra (3rd-6th) is missing from an otherwise complete vertebral column. While both 12th ribs and one right 3rd-10th rib are absent, all other ribs are present, however, most are fragmented. All long bones are present but for most the epiphyses are either damaged or absent. A fragment of the left humerus is present but the proximal half of the shaft is absent. The distal epiphyses of the left radius, left ulna, left fibula and right fibula are absent. Similarly, the proximal epiphyses of the right radius, right ulna, right tibia and left fibula are also absent. Preservation of the hand bones is poor and no pedal skeletal elements were recovered. For the hand, only two left metacarpals, all right metacarpals and three phalanges were recovered. Additional elements/fragments/items present include a bag of loose teeth, 1 non-human bone, unidentified postcranial fragments, 2 fragments of wood and one stone. Based on skull and pelvic features, this is a male; age-at-death indicators suggest a middle adult (35-49 yrs).

The anterior half to two-thirds of the 12th thoracic vertebral body shows severe lytic destruction (Figure 38a). The lesion appears as a number of scallops or lobes that largely affect the cranial, anterior and lateral surfaces; however, a portion extends through the body, creating a ~1 cm opening onto the caudal surface (Figure 38b). The lesion surfaces are well-defined and mostly sclerotic, although some remnant of the exposed trabeculae remains. The bone of the right lateral surface, while well-defined, is not sclerotic. The boundaries of the lesion are sharp and well-demarcated from the unaffected tissue. There is little to no reactive bone present. Minor lipping is present along the margin of the caudal surface and a possible Schmorl's node is visible at the left posterolateral margin of the lytic lesion. There is some pitting/lytic activity along the anterior and lateral margins of the 11th thoracic vertebra caudal surface (Figure 38c). However, this is relatively superficial. Another possible small, shallow region of lytic activity is present on the bone "island" located centrally. The circumference of destruction surrounding this "island" may be post-mortem damage. The margins are sharp, irregular and lighter in colour. Abnormal woven bone is present along the anterior and lateral surfaces of the 11th thoracic vertebra. There is also minor lipping along the margin of the caudal surface.

Two relatively large osteophytes project and meet at the right lateral articulating surfaces of the 5th and 6th thoracic vertebrae. Minor lipping is observed at the articulating body surfaces of the 2nd and 3rd cervical vertebra and the caudal surface of the 1st lumbar vertebra. Pitting, associated with degenerative joint disease, is noted on the right inferior articular facet of the 7th cervical vertebra and both inferior facets of the 4th lumbar vertebra, with osteophytic lipping of the superior facets of the 5th lumbar vertebra. The 1st lumbar vertebra has transitional facets, there is partial lumbarization of the 1st sacral element and partial sacralisation of the 1st coccygeal element. These anomalies indicate caudal border shifting involving the thoracolumbar, lumbosacral and sacrocaudal junctions.

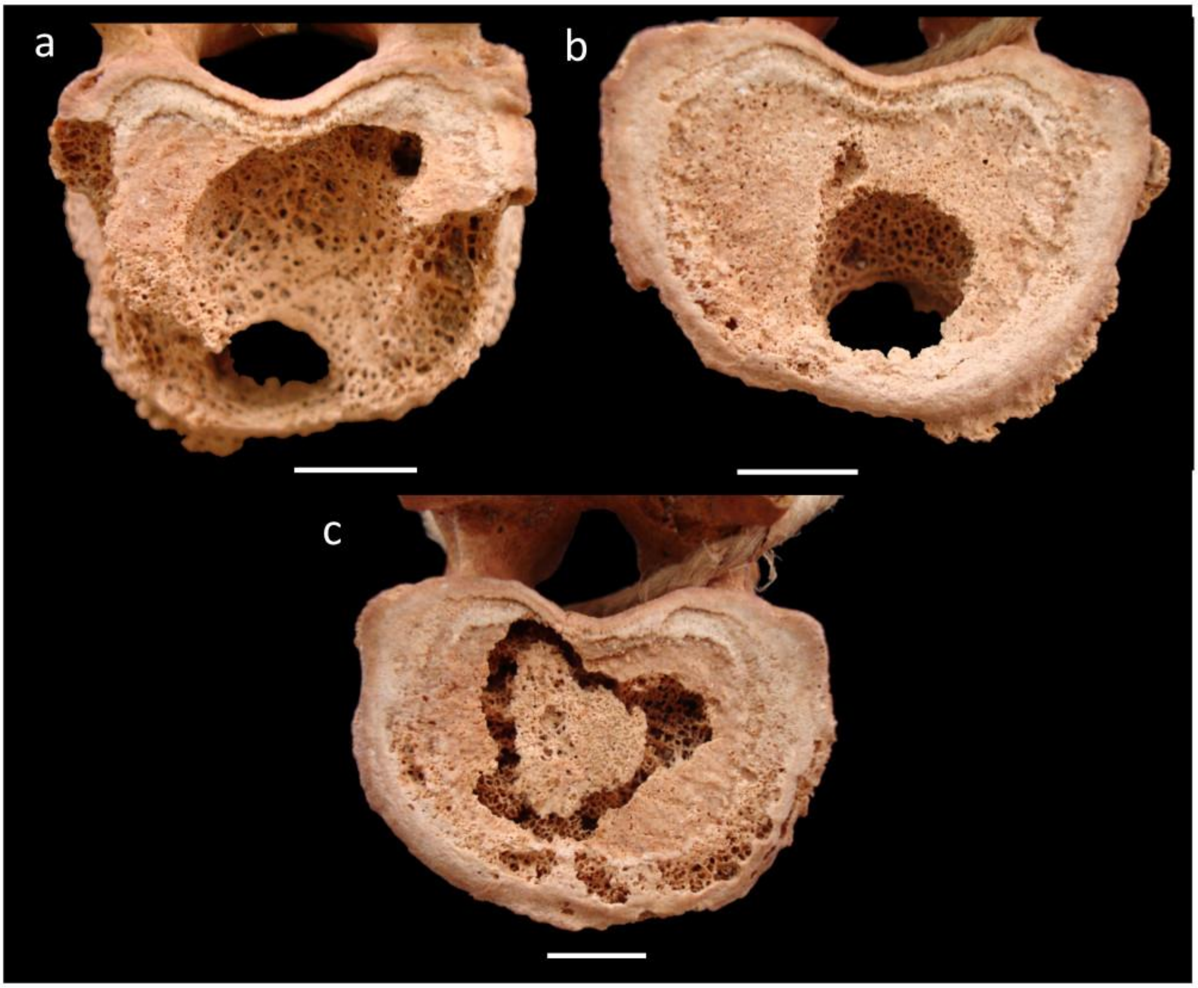


Figure 38: SAM-AP 3738 – Pathology of the 11th and 12th thoracic vertebrae. a) Cranial view of the lytic destruction of T12. b) Caudal surface of T12 with Schmorl's node located at the posterior margin of the lesion opening. c) Caudal surface of T11 with pitting located centrally, as well as along the anterior and lateral margins. Scale bar = 1 cm.

UCT 552

UCT 552 was unearthed in 1994 from Cobern Street, Green Point during a rescue excavation after a number of graves were disturbed by construction activity. In total 121 individuals were exhumed from 65 graves. These individuals are part of a larger 18th century informal burial ground that also includes the Prestwich Street (SAM-AP 3738) individual. The individual was buried supine and extended with the hands folded over the pelvis. Additional items recovered from the grave include a pipe stem, a bone-handled knife, a shovel blade and a musket ball (Apollonio, 1998). Isotopic analysis conducted by Cox (1999) indicates that this individual had a change in diet from a more terrestrial diet in childhood to one that included more seafood in later life. Cox postulates that this individual hails from a more temperate environment. Kootker et al. (2016) reached a similar conclusion. The isotopic analysis in this study indicated a greater proportion of C_3 foods in childhood than expected for the region, leading the authors to concur with a non-local origin for this individual. Cranial features suggest an admixed Black African ancestry and preliminary genetic results suggest this individual may have been born in Madagascar (personal communication, Kelly Harkins).

The remains of this individual are relatively complete and well-preserved. The cranium and mandible are complete but both have been fragmented post-mortem and subsequently glued back together. The complete hyoid is present. Post-mortem damage affects both scapular bodies. Both pubes have separated from the os coxae and, while the right side is complete, most of the left side is absent. The vertebrae are all present and complete. The ribs are fragmented but all are present and complete. All of the large long limb bones are present and complete except the left fibula which is missing its proximal epiphysis. The hand and foot bones are fairly complete. Four right carpals, one manual and nine pedal phalanges are absent. Three sesamoid bones were also recovered. In addition to this individual, four juvenile skeletal elements from another individual are also present. These include the left cuboid, one 5th metatarsal, one unidentified metatarsal shaft and one distal hand phalanx. Based on the skull and pelvic features, UCT 552 was a male; age-at-death indicators suggest a middle aged adult. However, the individual may have been slightly younger (30-40 yrs) as joint surfaces appear quite youthful.

A number of pathological lesions both lytic and proliferative are observed for this individual (Figure 39 and Figure 40). There is complete destruction of the 7th thoracic vertebral body, partial destruction of the 6th thoracic vertebral body and small lytic lesions present on the 8th thoracic vertebral body. Abnormal bone formation is observed on the vertebral bodies of the 7th cervical, 1st - 5th thoracic, 8th - 12th thoracic and 1st lumbar vertebral bodies, as well as most left and right ribs. Some ribs have lytic lesions/depressions along the pleural surface of the vertebral ends. There is also lytic destruction of the left sacroiliac joint and abnormal increase in diameter of the proximal left tibial shaft (Figure 41a).

Approximately half of the 6th thoracic vertebral body has been destroyed by a lytic lesion (Figure 39c). As a consequence, the vertebral body is somewhat wedge-shaped. Multiple scalloped lesions are visible resulting in the destruction of the entire caudal and anterior vertebral body surfaces with partial destruction of the cranial surface (anteriorly) and a small circular opening on the posterior surface (centrally). The lesion surfaces are smooth, well defined and slightly sclerotic but remnants of the trabecular structure are still visible. No reactive bone formation is noted on the vertebral body. Depressions, with exposure of the underlying trabeculae, are present just superior to the inferior facets. There is also abnormal bone formation along the anterior surface between the inferior facets. These changes, as well as those present on T7 and T8, suggest the presence of an unreduced subluxation at both zygapophyseal joints of T7, probably as a consequence of the destruction of the vertebral bodies of T6 and T7.

Given the extent of the lytic destruction of the 6th thoracic vertebra, it is likely that the 7th thoracic vertebral body has been completely destroyed by this pathological process. However, it is also possible that the vertebral body was present but not preserved/recovered. Nevertheless, there was at least complete antemortem separation of the vertebral body and neural arch as is evidenced by the remodelled surfaces at the pedicles (Figure 39d). Similar, but more severe, depressions abut the zygapophyseal facets of this vertebra as was described for T6. Furthermore, abnormal, sclerotic bone formation is noted on the anterior surfaces of spinous process, the transverse processes and zygapophyseal facets. The pathology associated with the zygapophyseal facets is likely a consequence of the unreduced subluxation described above. While remodelling as a consequence of this may explain the pathology at the transverse and spinous processes, it is also possible that this pathology is associated with the pathological process occurring at the vertebral bodies. It seems likely, given the pathology of the vertebral bodies and neural arches, that some kyphosis was present.

A number of smaller (<1 cm) lytic lesions are present on the body of the 8th thoracic vertebra (Figure 39e). A lytic lesion is present on the anterior surface of the vertebral body just inferior to the superior margin. Another lesion is present on the superior surface at the right lateral margin. This lesion extends through the bone and opens onto the lateral surface. A number of shallower lesions are present on the superior surface resulting in an irregular topography. The margins of these lesions are smooth, well-defined and slightly sclerotic. There is also abnormal bone formation along the lateral and anterior surfaces of the vertebral body. The abnormal bone is porous and lamellar to sclerotic. There is a large enthesophyte on the left lateral surface that extends along the lateral surface of T8, inferiorly, for 1-2 cm to approximately midway along the T9 vertebral body. The enthesophyte is not attached to T9. Also on the left lateral surface, is a roughly circular, depressed region (Figure 39f). This depressed region is bordered by dense, smooth sclerotic bone anteriorly, posteriorly and inferiorly but the surface of the depression is porous and somewhat spiculated. This lesion may suggest the presence of an overlying mass. There is no pathology on the inferior surface of the vertebral body. The only changes visible on the neural arch are the depressions just inferior to the superior facets which have been described on T6 and T7 above.

Abnormal bone formation present along the left lateral and anterior surfaces of vertebral bodies of C7-T5 and T8-L1 (Figure 39a and b). The abnormal bone formation is restricted to the anterior surface for C7, T1, T10-L1 and is present on the left lateral surfaces as well for the remaining vertebrae (T2-5 and T8-9). The periosteal new bone formation becomes progressively worse in appearance and extent as one moves caudally to T5 (lytic destruction predominates for T6 to T7) and then improves caudally to L1. This new bone is irregular, porous and lamellar to sclerotic. There is minor osteophytic lipping of the facets for the rib heads on T12.

Both new periosteal bone formation and lytic lesions are present on the left and right ribs. The pathology is predominantly focused along the vertebral third of the ribs and the lytic lesions are concentrated at the head and neck. On the left side, pathology is observed on the 4th-10th ribs and is most severe on the 6th-8th ribs. Pathological changes are almost exclusively present on the pleural surface of the ribs with the exception of the 6th rib. The pathology on the 4th and 5th ribs is minor and consists of a single and relative small patch of intermediate woven-lamellar bone (Figure 40e). For the remaining ribs, the new bone formation is more extensive resulting in anteroposterior thickening. The bone is intermediate (woven-lamellar) to lamellar and lighter in colour than the surrounding unaffected cortex. Multiple small, smooth, circular depressions are present on the 6th and 8th ribs in addition to the proliferative lesions. There is complete lytic destruction of the 7th rib head (providing additional support for the destruction of the 7th thoracic vertebral body) (Figure 40d). This lesion differs from those on the 6th and 8th ribs. The margins are sharp and well-demarcated and the lesion surface is well defined but not sclerotic. From the 8th-10th ribs the proliferative lesions lessen in extent and become increasingly sclerotic.

In general, right ribs show more severe pathology in extent and presentation than left side. Pathology is present on the 2nd to 11th right ribs and most severe on the 6th-8th ribs, affecting the vertebral half predominantly (Figure 40c). Lesions are mainly observed along the pleural surface; however, for some ribs pathology extends onto the inferior and sometimes even external surfaces. The nature of these pathological changes are similar to those described for the left side. Abnormal periosteal bone deposition, resulting in anteroposterior thickening of, particularly, the head, neck and angle is observed. However, there are more lytic lesions and more frequent alteration to the inferior surface, than occurred for the left ribs. Smooth-walled depression and lytic lesions are present on the 6th to 9th ribs and are

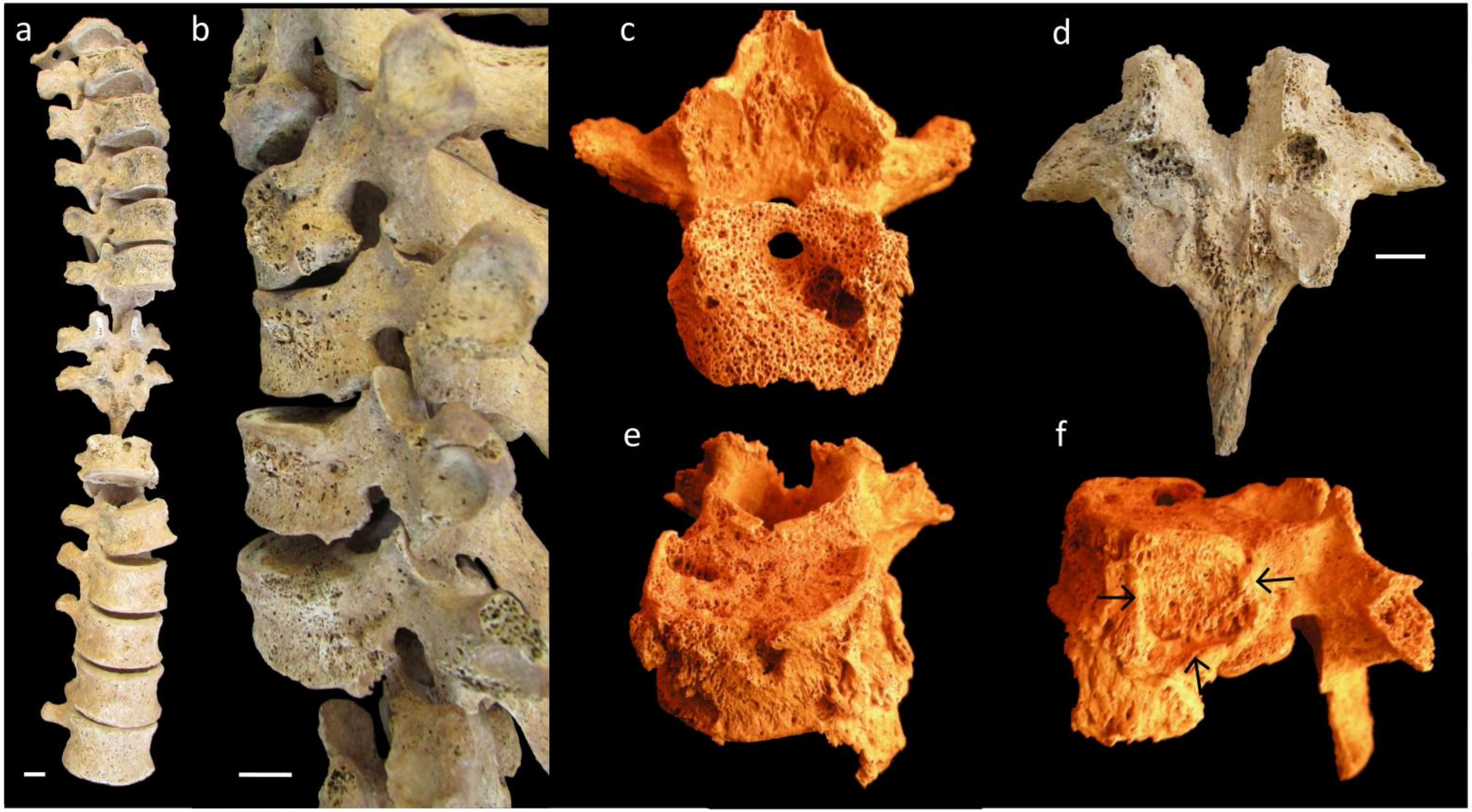


Figure 39: UCT 552 - Lytic lesions and abnormal bone formation present on some of the thoracic and lumbar vertebrae. a) Anterolateral view of all vertebrae with abnormal bone formation and/or lytic activity (C7-L1). b) Left lateral view of C7-T1 showing abnormal bone formation along vertebral body surfaces. c) Lytic destruction of the anterior and caudal surfaces of T6. d) Anterior surface of T7 neural arch. e) Lytic lesions and abnormal bone formation on the anterior and cranial surfaces of T8. f) Right lateral view of T8 with a large enthesophyte. Arrows indicated the margins of a circular depression.

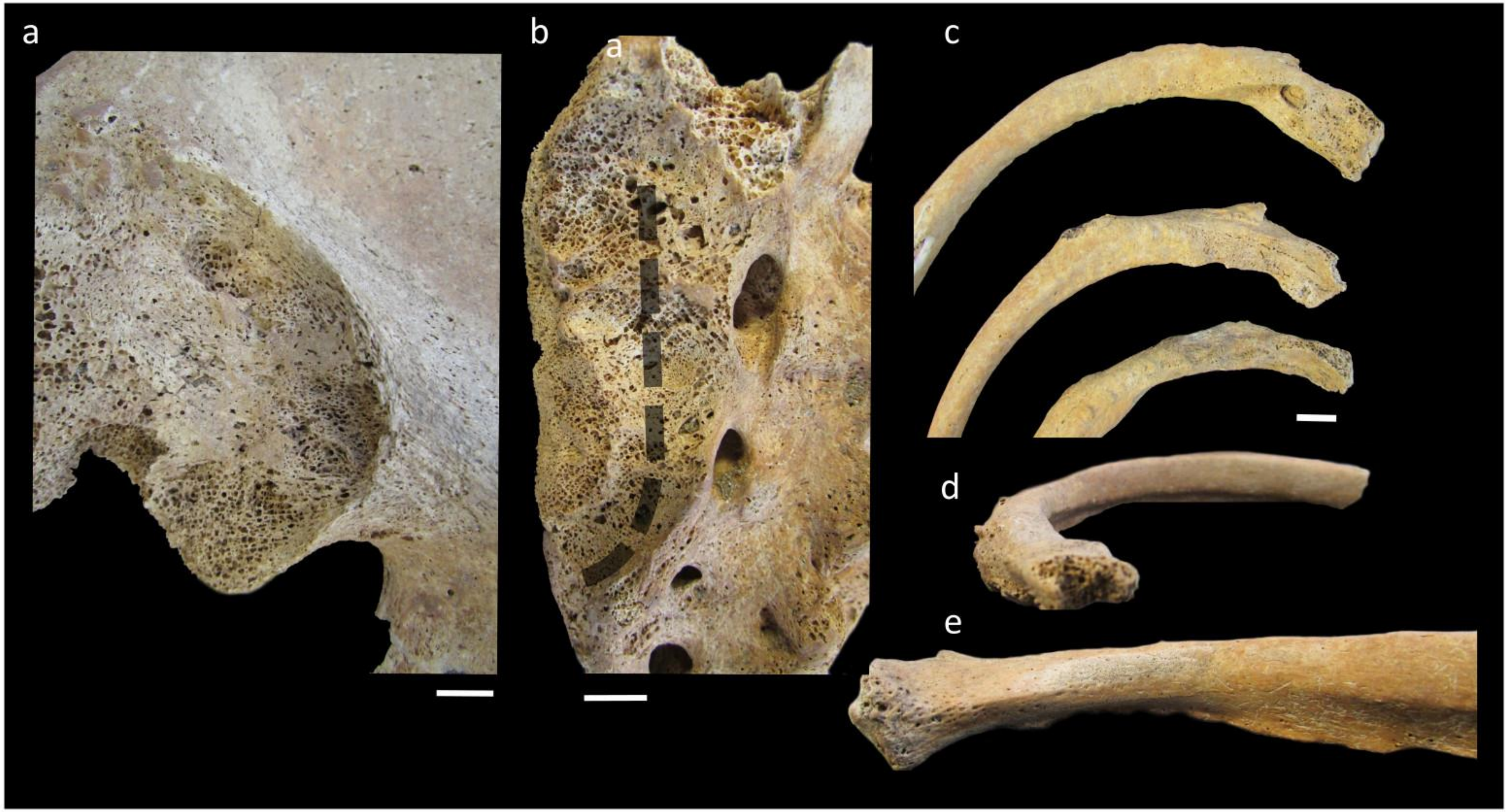


Figure 40: UCT 552 – Pathology of the ilium, sacrum and ribs. a) Partial lytic destruction of the left auricular surface of the ilium. b) Depressed region between the auricular surface and lateral crest of the left sacrum. c) Lytic lesions and abnormal bone formation at the vertebral ends of three right ribs. d) Lytic destruction of the 7th left rib head. e) Woven bone along the pleural surface of one left rib. Scale bar = 1 cm.

Figure 41: UCT 552 - Pathology of the left tibia and sternal aperture. a) The proximal third of the left tibia is much thicker than that of the right (anterior view). b) Sternal aperture present at the distal aspect of the sternal body. Scale bar = 1 cm.

more severe (more numerous and larger) than the left side. The lesion surfaces are lamellar to sclerotic and some are slightly porous. None of the ribs on the right side have patches of woven bone on the pleural surface as was observed for some ribs on the left side.

There is partial lytic destruction of the left sacroiliac joint. The posterior aspect of the sacral auricular surface is particularly affected, although the entire joint surface is abnormally porous. The lytic destruction is most pronounced at the superior half of the auricular surface. On the sacrum, the nonarticular surface between the lateral crest and auricular surface is more porous and slightly depressed relative to the right side with thinning of the cortical bone to the point of trabecular exposure in parts (possible pressure erosion due to an overlying mass) (Figure 40b). There is some postmortem damage in the region and along the anterior surface. There is significant postmortem damage to the posterior part of the left ilium and auricular surface obscuring the true extent of the pathology. Despite the postmortem damage, smooth-walled, well defined lytic lesions are visible along parts of the auricular surface (Figure 40a). A single small patch of woven bone is present along the internal and external surface of the left ilium. In general, the cortical bone adjacent to the auricular surface and the articular bone appear much thinner along the internal surface of the ilium.

Abnormal shape of the proximal third of the left tibial shaft is noted (Figure 41a). This region of the left tibia is much thicker than that of the right, particularly posteriorly, laterally and medially (largely sparing the anterior border). There are no visible fracture lines, and the cortical surface appears normal/integrated. Minor ossification is observed at the soleal line. It is unclear whether this lesion is related to the pathological process occurring at the vertebra and sacroiliac joint.

Two developmental field anomalies are also observed for this individual. The first is a sternal aperture (Figure 41b). The aperture, a small round hole, is located at the distal third of the sternal body. The other anomaly is the bilateral presentation of a patellar vastus notch. The superolateral aspect of both patellae are absent.

Finally, the bones of this individual are light in weight. This may indicate disuse osteopenia suggesting immobility probably as a consequence of the vertebral and iliosacral pathology. This likely represents a case of Pott's disease with an associated paravertebral abscess. The rib lesions suggest pulmonary involvement as well. The lesion at the sacroiliac joint indicates the disease is multifocal and the pathology at the tibia may be an additional focus, although the evidence for this is weak.

UP 49

This individual is curated at the University of Pretoria and was unearthed during 1995-1996 archaeological excavations of a Batswana settlement at the Late Iron-Age site, NTGOO1, at the base of Makgope Hill (Pistorius et al., 1998). Makgope Hill is situated on the farm Nooitgedacht (287JQ), 20km north of the town Marikana in the North West. Two burials were uncovered during these excavations and this burial was located near the entrance of a stock enclosure in the Kraal complex. The individual was buried in a flexed position, facing the south-west. A clay pot was found in the grave resting on a pedestal of stones. Three ranges of calibrated dates were reported (uncalibrated dates were not reported) AD 1670(1682)1702, AD 1716(1745)1784 and AD l792(1807)1822 (Pta-7379). The authors suggest the burial is most consistent with the earlier two ranges.

This individual is only partially complete and preservation is moderate. The cranium is partially fragmented and while mostly complete, significant post-mortem damage is observed to the frontal and sphenoids. The mandible is present but the condyles are absent. The clavicles are present and complete. Some postmortem damage is observed for the scapulae bodies and the glenoid fossa of the left scapula. Vertebral preservation is poor. The 1st cervical vertebra and the 2nd cervical vertebral body, as well as three cervical, eight thoracic and two lumbar neural arch fragments are all that were recovered. The sacrum and sternum are absent. The ribs are also generally poorly preserved. Six left and eight right ribs are present but only the 1st and 2nd ribs for each side are relatively well preserved. The remaining ribs are only represented by small fragments. Partially preserved ilia are all that remain of the os coxae. Both patellae are present and complete. All large limb long bones are present but many have post-mortem breaks and the epiphyses are generally poorly preserved or absent. One exception is the right fibula, for which only a fragment of the distal epiphysis is present. There is moderate preservation of the hand and foot bones. One right and six left carpals are present, all except one right metacarpal are present and 17 manual phalanges are present. For the foot, five left and five right tarsals, three left and three right metatarsals and one phalanx are present. Additional items present include a bag of teeth and one non-human bone. Due to poor preservation of the pelvis, sex determination is based on the skull only. Other than large mastoid processes, the features of the skull are relatively ambiguous. However, given the location of the burial this individual is likely male. Age-at-death could not be estimated due to the poor state of preservation; however, all epiphyses are fused and the individual is mature/adult. Based on dental wear and burial location, Pistorius et al. (1998) report this individual is an old adult.

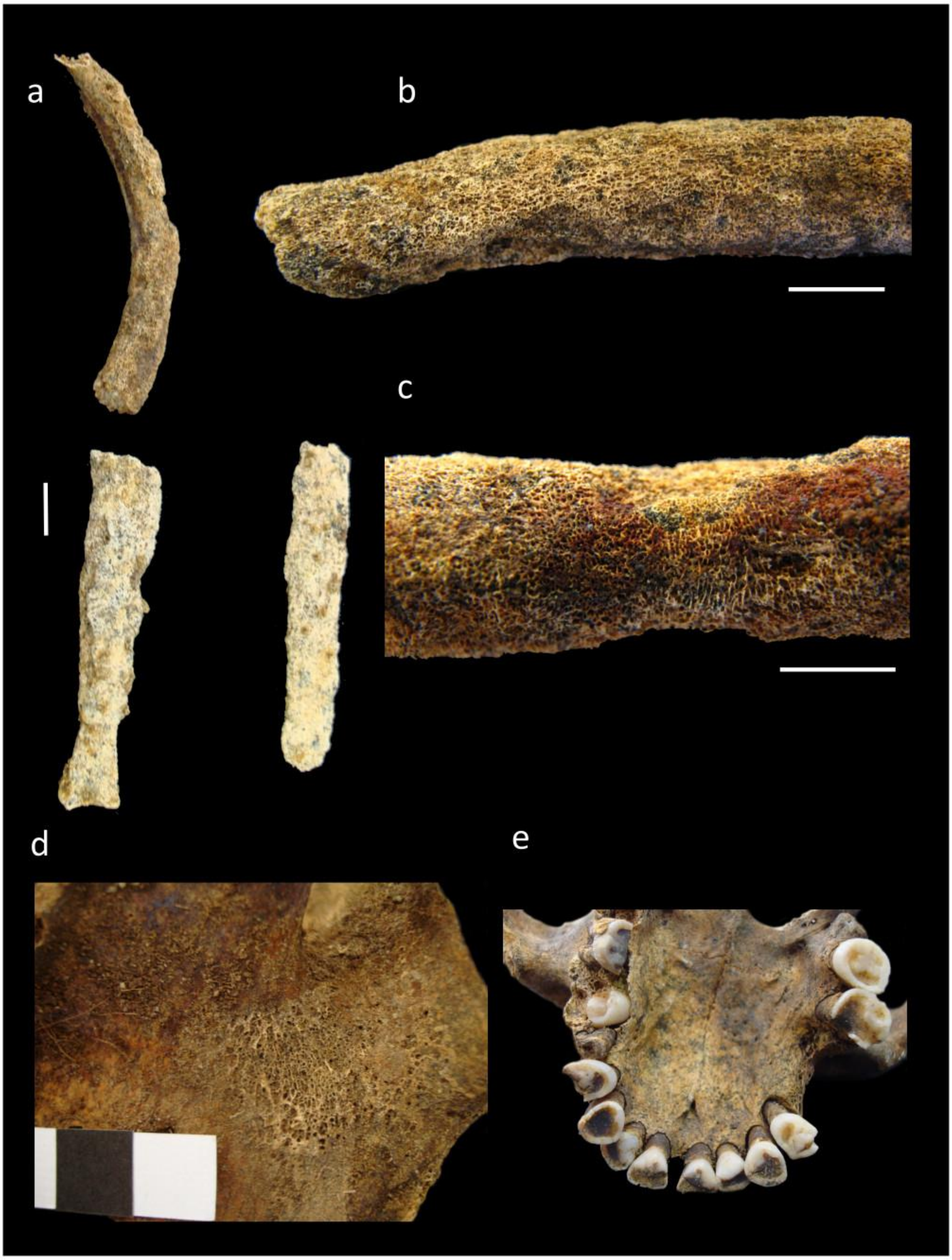


Figure 42: UP 49 - Pathology of the ribs and scapula and a supernumerary tooth visible within the left maxillary alveolus. a) Three pathological ribs showing loss of normal anatomy, increased shaft diameter and irregular surface. b) and c) Rib surfaces at greater magnification. d) Abnormal bone formation along the dorsal surface of the right scapula. e) Supernumerary premolar embedded in the alveolus of the left maxilla. Scale bar = 1 cm.

Abnormal bone formation is present on multiple ribs resulting in a thickening of the entire rib shaft (Figure 42a, b and c). Only rib fragments have been recovered and this, in addition to, the loss of almost all anatomical features due to the pathology, renders it impossible to determine the number and, in most cases, the side of affected ribs. However, it seems likely that ribs on both sides are affected. For most pathological rib fragments, the pathology affects the entire fragment resulting in a thickened shaft with an irregular surface (particularly at the pleural surface). The entire shaft (both the pleural and external surface) consists of a coarse, porous and 'puffy' bone. Due to the pathology, the costal grooves are no longer visible and, in most cases, the vertebral and sternal ends of the ribs are absent due to postmortem damage. The ribs also lack structure internally where there is no distinction between cortical and trabecular bone.

Abnormal bone formation is also present on right scapula (Figure 42d). The bone is very porous and similar to, but much milder in expression than, the rib lesions. There is no pathology on the left scapula. This abnormal bone is present at three locations on the right scapula body. Firstly, a lesion is located at the subscapular fossa, medial to the glenoid fossa and just inferior to the coracoid process. Another lesion is located at the infraspinous fossa, just inferior to the spine, and the final lesion is located at the supraspinous fossa, where the spine meets the superior blade. These may be expression of the same lesion on multiple surfaces. It is not possible to determine whether the bone is added to the cortical surface or replaces it and the lesion margins are poor circumscribed.

Two developmental anomalies are noted and include a cleft of the posterior arch of the 1st cervical vertebra and a supernumerary premolar. The premolar is located in the right maxilla beneath the 1st molar (which has been lost antemortem) (Figure 42e). While the crown of the tooth is visible, it is still embedded in the alveolar bone.

MRB\20

All of the information presented for this individual was sourced from Steyn et al. (2002). MRB\20 was unearthed from an informal graveyard located at Maroelabult, on the farm De Kroon 444 Partition 199, near Brits in the North West Province. Two graveyards containing 47 graves were discovered during an archaeological impact assessment conducted prior to the establishment of open-cast mining activities. The graves are thought to belong to Sotho-Tswana individuals who resided in the area. Based on consultations with nearby communities the graveyard was likely in use from the last decade of the 19th century to the first half of the 20th century. MRB\20 was a juvenile, estimated to be 3 years old at death. A number of pathologies are reported for this individual, including an open fontanelle, subperiosteal bone growth on the ribs and cribra orbitalia. The new periosteal bone was noted on the pleural surface of the lower right ribs along the posterior and lateral aspects and resulted in a thickening of the rib. The authors note that the rib lesions indicate a chronic pulmonary or pleural disease and consider tuberculosis as part of the differential diagnosis.

GENETIC RESULTS

Destructive analysis and export permits were obtained for nine of the twelve individuals (Table 23). Table 24 presents a list of these specimens as well as the skeletal element from which the sample was taken. Human mitochondrial DNA (mtDNA) was detected in all samples using conventional PCR (Table 25). Only five individuals, GLD 38.8, MMK331, UCT552, NMB 1416 and GLD 8.3, showed positive amplification of one or more of the *Mycobacterium tuberculosis* complex (MTBC) assays. All five individuals showed positive amplification of the *rpoB*2 assay, while two individuals showed positive amplification of the IS*6110* and IS*1081* assays, respectively. GLD 38.8 was the only individual that produced amplicons for all three assays. Furthermore, for this individual, DNA was successfully amplified from all three replicates for both the IS*6110* and *rpoB*2 assays, while two of the three replicates were positive for the IS*1081* assay. MMK 331 and UCT 552 showed positive amplification for two assays. The *rpoB*2 marker was detected in both cases but only one of the three replicates produced amplicons. MMK 331 also showed positive amplification of the IS*6110* marker and all three replicates were successfully amplified. UCT 552 showed positive amplification of the IS*6110* assay, with two of the three replicates yielding a DNA product. Finally, NMB 1416 and GLD 8.3 were only successfully amplified from the *rpoB*2 assay. Only one of the replicates were positive for GLD 8.3, while all three replicates were positive for NMB 1416. For the remaining four individuals, GLD 8.2, NMB 1229, SAM-1271 and UP 49, none of the assays yielded an amplified product for any of the MTBC assays.

It should be noted that the results reported here were for a second extraction of the samples. The first extraction was performed using a different method (Boom et al., 1999). None of the samples produced an amplification product for the 9bp-deletion primers. Quantitative PCR assays were also performed to test for the presence of human mitochondrial and nuclear DNA. These showed limited success. Four of the samples routinely produced amplicon for the mtDNA assay, while only two were positive for the nuclear assay. None of the samples yielded amplicons for any of the MTBC assays, which targeted slightly different regions of the IS*1081* and *rpoB* genes. The results will not be discussed further.

The qPCR was performed as a screening tool to identify candidates for next-generation sequencing with the hope of obtaining strain informative sequence information. Five individuals were selected for next-generation sequencing which was conducted in an independent laboratory (Prof. Johannes Krause, Max Planck). GLD 8.2 and SAM-AP 1271 were selected prior to the successful qPCR screening and were subjected to a PCR-based targeted enrichment approach. However, neither sample yielded DNA sequences that could specifically be attributed to members of the MTBC. A second attempt to obtain sequences occurred after qPCR screening. These samples were selected based on the positive amplification of DNA in more than one MTBC assay. A different method of enrichment, hybrid capture, was attempted for these samples; however, similar to the previous attempt, no sequences were obtained that could be uniquely identified as belonging to the MTBC.

Table 23: Destructive analysis and permanent export permit numbers for possible cases of skeletal tuberculosis.

Accession Number	Destructive analysis permit: authorizing institution[a]	Destructive analysis permit number	Export permit number[b]
MMK 331	SAHRA	80/11/01/002/51	80/11/01/003/52
GLD 38.8	SAHRA	80/11/01/002/51	80/11/01/003/52
GLD 8.2	SAHRA	80/11/01/002/51	80/11/01/003/52
GLD 8.3	SAHRA	80/08/04/009/52	80/08/04/009/52
NMB 1229	SAHRA	80/11/01/002/51	80/11/01/003/52
NMB 1416	SAHRA	80/11/01/002/51	80/11/01/003/52
SAM-AP 1271	SAHRA	80/11/01/002/51	80/11/01/003/52
UCT 552	HWC	2008-04-004	80/08/03/004/52
UP 49	SAHRA	80/08/04/010/52	80/08/04/010/52

[a] SAHRA = South African Heritage Resource Agency, HWC = Heritage Western Cape

[b] All permanent export permits were obtained from SAHRA.

Table 24: Individuals and skeletal elements sampled for ancient DNA analysis.

Accession Number	Element	Side	Region and/or number	Additional information
MMK 331	rib	R	5th-10th?	
GLD 38.8	rib	R	3rd	
GLD 8.2	rib	L	5th	
GLD 8.3	vertebra		lumbar, 4th	Caudal anterior region of body
NMB 1229	os coxa	R	ilium	Auricular surface
NMB 1416	rib	L	5th	
SAM-AP 1271	vertebra		lumbar, 4th	left half of body
UCT 552	vertebra		thoracic, 6th	entire body
UP 49	rib	ND	ND	

ND = Not determined

Table 25: Summary of conventional PCR and qPCR results

Specimen Name	Conventional PCR (Human 9bp deletion primers)	qPCR (number of positive replicates out of 3)			NGS – MTBC enrichment
		IS*6110*	IS*1081*	*RpoB*2	
GLD 38.8	+	3	2	3	--
MMK 331	+	-	3	1	--
UCT 552	+	2	-	1	--
NMB 1416	+	-	-	3	
GLD 8.3	+	-	-	1	
GLD 8.2	+	-	-	-	--
NMB 1229	+	-	-	-	
SAM-AP 1271	+	-	-	-	--
UP 49	+	-	-	-	

+ = amplification product detected

- = no amplification product detected

-- = no MTBC-specific sequences detected

Blank cells = analysis not performed

CHAPTER 6: DISCUSSION

As was the case with the results chapter, this discussion is presented in two parts. First, a brief discussion of the South African skeletal assemblage as a whole is presented in order to provide insight into the preservation and demographics of the assemblage, and help frame the context within which the identified tuberculosis cases can be discussed. Second, the bulk of the discussion will focus on the diagnosis of tuberculosis, and the implications of this study for our understanding of the emergence and spread of tuberculosis in South Africa.

COMPLETE ASSEMBLAGE

Most of the individuals in the total dataset (N = 2321) were unearthed from single or small groupings of burials interred during the Holocene, including the colonial period. Larger cemetery derived samples, of which the largest is Gladstone (n = 106), are also present but only form a minor component of the sample. Thus, this dataset represents a geographically and temporally aggregate sample of burials in South Africa. There are advantages and disadvantages to analysing such a massive, heterogenous sample. One advantage is that it allows for an understanding of patterns over broad geographic and temporal ranges (Milner and Boldsen, 2017). Furthermore, the large size and aggregate nature of the dataset may help overcome issues created by extrinsic taphonomic factors (environmental factors like water availability, soil type, pH, temperature, etc.) that differentially affect preservation at a local scale. In doing so, the analysis of this dataset may provide baseline proportions for variables of interest to which more geographically and temporally restricted samples can be compared. On the other hand, one loses the ability to associate local conditions with observed patterns. Furthermore, no similar studies have been identified in the current literature to serve as a comparison. Comparisons are made to other studies where possible but there are often sufficient differences in the methodology or nature of these datasets to warrant caution when making inferences from these comparisons.

TOTAL SKELETAL PRESERVATION

A number of intrinsic and extrinsic factors affect the preservation of human skeletal remains (see Henderson, 1987; Manifold, 2012). Intrinsic factors include the age of the individual, bone size and type, the presence of pathology and, finally, bone porosity and density. Extrinsic factors include the amount of groundwater, the soil type and pH, ambient temperature, surrounding flora and fauna, as well as anthropogenic factors like burial practices and depth of the grave. A number of additional anthropogenic factors occurring during and after recovery can also affect preservation and skeletal representation in assemblages and these include incomplete archaeological sampling, incomplete recovery, biased retention and poor curatorial practices (Waldron, 1987).

While many researchers record skeletal preservation as the first step in an osteological analysis, patterns of preservation are typically only reported when addressing the topic specifically. Such studies have focused on factors affecting skeletal preservation / assemblage composition, including environmental taphonomic and anthropogenic factors, and/or understanding how this might reflect burial practices in both bioarchaeological and forensic contexts. These studies differ from what is presented here in that they have either focused on single sites (e.g. Mays, 1992; Waldron, 1987), a collection of cemetery/mass/crypt/tomb burials (e.g. Beckett, 2011; Bello and Andrews, 2006; Cox and Bell, 1999), commingled remains (e.g. Willey et al., 1997), unusual taphonomic conditions (e.g. Stojanowski et al., 2002) or a particular demographic subgroup, e.g. juveniles (e.g. Manifold, 2015).

Total skeletal preservation is a crude measure of preservation by individual and is determined by taking the weighted average score of all the elements for each individual; therefore, missing and partial elements are taken into account when calculating an individual's preservation score. For the dataset as a whole, total skeletal preservation was relatively poor. More than half of the skeletons present in this dataset were <25% complete. Although, it should be noted that this measure has not be reported previously and so there is nothing to compare it to in the literature.

When comparing total skeletal preservation among institutions, most follow a similar pattern to that observed for the dataset as a whole, with the exception of three institutions, MMK, UP and NMB. The MMK remains show a very different pattern of preservation. Here most of the remains are well preserved but this divergence is driven by the good preservation of the Gladstone individuals which make up almost 40% of the MMK assemblage. If the Gladstone individuals are removed, the pattern of preservation for the remaining individuals is not significantly different to the other institutions. NMB and UP both have significantly more poorly preserved individuals relative to the other institutions. The reasons for this discrepancy are unclear. In the case of UP, the smallest assemblage (n = 103), this may be an artefact of small sample size. This is not a suitable explanation for the poor preservation at NMB, which is the second largest assemblage (n = 424). Another explanation might be differences in local taphonomic factors since there does appear to be a relationship between burial location and curating institution; however, there is considerable geographical overlap between UP and WITS and, similarly, between NMB and MMK (see Figure 27, chapter 5). Given that poor preservation is not observed at WITS and MMK, this explanation also seems unlikely. Finally, differences in curatorial practices may explain the discrepancy but this was not investigated and may be difficult to assess.

PRESERVATION BY ELEMENT, SIDE AND SUB-DIVIDED ELEMENT

The frequencies of skeletal element preservation reported here are equivalent to the bone representation index (BRI) reported in Bello and Andrews (2006). The pattern of element preservation is similar to what has been reported in other studies and what might be expected based on bone size and density (Galloway et al., 1997; Mays, 1992; Stojanowski et al., 2002; Waldron, 1987; Willey et al., 1997). In general, these studies have indicated good representation of the skull and large long bones, poor representation of small and low density elements (like the bones of the hand and foot, hyoid, coccyx, sternum and patella) and variable (but often moderate) representation of the remaining elements. A similar pattern is observed in this study and a more detailed discussion follows below.

Preservation of sided elements was also considered. In a study examining bone mineral density of modern skeletonized individuals, Galloway et al. (1997) report minimal difference in density when comparing the left and right limb bones. Significant differences were noted for some of the upper limb elements but, even then, only for parts of these elements. Using these data for comparison, Willey et al. (1997) note an abundance of right sided elements among the commingled remains from the Crow Creek Massacre site and, consequently, attribute this pattern to human modification rather than taphonomic processes. Thus, side disparities may reveal unexpected burial or curatorial practices. In this study no obvious pattern of side disparity is noted for the skeleton as a whole. A relative abundance of left large long bones is observed but the differences are small and are not statistically significant. These results, therefore, conform to expectations based on studies of contemporary samples and support the conclusion that side and preservation are not correlated.

A detailed discussion of element preservation follows and is presented in the general order of abundance observed in this assemblage.

The most abundant skeletal elements were those of the skull. The cranium was better represented than the mandible, and the bones of the neurocranium were, generally, more abundant than those of the face. Bello and Andrews (2006) in their analysis of six medieval and post-medieval European cemetery/crypt/mass burial sites, note a similar pattern of preservation. Robb (2016) citing Waldron (1987) argues that crania and major long bones (like the femora and tibia) have a comparable ability to resist destruction and should, therefore, be expected to be equally represented in skeletal assemblages. Bello and Andrews (2006) similarly note good representation of crania and femora in theirs and other studies. Robb (2016) adopts a simulation approach to examine the relationship between MNI and funerary ritual and based on the results of his analysis, posits that a major discrepancy between cranial and long bone frequencies may represent preferential curation of skulls. A relatively big difference between the most abundant cranial element and femoral element (~30%) is noted in this analysis. In fact, 38.5% (n = 893) of individuals in this assemblage are represented only by crania and/or mandibles. It is, therefore, possible that this assemblage may reflect a bias towards the preferential retention of crania possibly linked to early curatorial or acciquisition practices.

As was noted above, studies have reported good representation of the large long bones (humerus, radius, ulna, femur, tibia and fibula) (Bello and Andrews, 2006; Manifold, 2015; Mays, 1992; Stojanowski et al., 2002; Waldron, 1987). In this study the long bones were relatively well represented (with the exception of the fibula) and follow the previously reported pattern (Bello and Andrews, 2006) of better representation of proximal compared to distal elements (although this pattern likely reflects bone size and density rather than anatomical location). While the femur is typically the best represented of the long bones (as is the case in this study), the order of long bone abundance does vary from one study/assemblage to the next. The fibula does typically have the lowest representation among the long bones; however, its representation is particularly low in this study. Although, as Bello and Andrews (2006) suggest, this may be due to the fact that the fibula is easily fragmented which may affect the preservation and recording of this element. The representation of element segment is also congruent with previous reports. For all elements, the diaphyses were more abundant and better preserved (fewer preservation scores of 2 and 3) than the epiphyses (Bello and Andrews, 2006; Stojanowski et al., 2002). While there were no significant differences in representation of diaphyseal segments, the middle third was better preserved than either proximal or distal third. Contrary to the findings of Stojanowski et al. (2002), who noted differences in representation for the proximal and distal epiphyses of the humerus, tibia and femur, proximal and distal epiphyseal frequencies were similar for all elements except the ulna and fibula. Proximal ulnar epiphyses were more abundant than distal epiphyses, while the converse was true for the fibula. It should, however, be noted that distal epiphyses were better preserved (fewer preservation scores of 2 and 3) for all elements except the femur (where the proximal epiphysis was better preserved) and this may be a minor reflection of the pattern observed in Stojanowski et al. (2002). The authors of that paper argue that bone density studies support such a pattern; however, in this (larger) sample the effects appear to be relatively minor.

The vertebrae, as a group, were well represented in this assemblage but preservation (reflecting both missing vertebral elements and incomplete elements) was relatively poor. The first and second cervical vertebrae were the most abundant of the vertebrae for which individual scores were recorded. Neural arches were typically more abundant than vertebral bodies, and for the thoracic and lumbar vertebrae the neural arches were also better preserved. The converse was true of the cervical vertebrae where vertebral bodies were better preserved than neural arches. Bello and Andrews (2006) and Manifold (2015) also note good representation of the vertebrae and, particularly, the first and second cervical vertebrae. Bello and Andrews (2006) speculate that this may be attributed to the protection afforded by the cranium. The authors also note better representation of the neural arches compared to the vertebral bodies. Furthermore, they observed a greater abundance of lumbar relative to thoracic vertebrae. A similar pattern was not observed in this study but T1-T9 were not scored individually, leaving only T10-T12 for comparison with the individually scored lumbar vertebrae. The similarities between the larger and denser lower thoracic and lumbar vertebrae may not extend to the entire thoracic region.

Among the postcranial bones, the os coxae are also relatively well preserved. Of the os coxae segments, the ilium is the best represented, followed by the ischium. The pubis, in addition to being the least abundant segment, also shows the worst preservation. Of the articular surfaces, the auricular surface is recovered more frequently than the acetabulum. Other studies have also reported good representation of the os coxae (Bello and Andrews, 2006; Manifold, 2015; Stojanowski et al., 2002). These reports have detailed preservation patterns of features not recorded in this study, limiting further comparison. Bello and Andrews (2006) do, however, note that the sciatic notch and acetabulum were the most abundant os coxae segments identified in that study and Stojanowski et al. (2002) notes that the pubic region is less well preserved than the sciatic notch and auricular surface – both of which seem to indicate a lower representation of the pubis.

The clavicle and scapula were relatively well represented (compared to long bone preservation) and are equally abundant in the assemblage. A similar pattern was observed by Bello and Andrews (2006) and Manifold (2015) who also note relatively good representation of these elements. Stojanowski et al. (2002) reports poor preservation of the scapula which suggests there may be variation in the preservation of this element; however, that study was investigating skeletal preservation in anaerobic peat which may contribute to the observed differences.

The ribs showed moderate representation but, similar to the vertebrae, were poorly preserved. The 3-10th ribs, which were scored collectively, were the most abundant but also least well preserved (again this includes both missing elements and incomplete elements). Of the ribs scored individually (1st, 2nd, 11th and 12th ribs), abundance decreased as rib number increased. The 1st and 12th ribs, when present, were, however, well preserved. Ribs representation and preservation are rarely reported in any detail. Mays (1992) and Waldron (1987) report poor representation of the ribs. Manifold (2015), however, reports good representation of the ribs and, furthermore, notes that the best preserved ribs were the first and last (which the author speculates may be due to the location in the body) but this study examined subadult remains exclusively and patterns of preservation may differ from what is seen in adults or mixed assemblages.

The sternum is poorly represented. Of the sternal elements the manubrium is most frequently recovered, while the xyphoid is recovered only infrequently; however, both of these elements show better preservation when recovered than the sternal body. Low sternal representation is reported in other studies (Beckett, 2011; Stojanowski et al., 2002) and can be predicted based on the low density and high proportion of trabecular bone (Stojanowski et al., 2002). The pattern of abundance for the manubrium, sternal body and xyphoid may also be predicted based on differences in size and density but poor xyphoid representation may also result from poor recovery during excavation. Bello and Andrews (2006) report variable representation of sterna ranging from 26-62% in the six skeletal collections under investigation in that study. In agreement with this study, the authors also report a greater abundance of manubria compared to sternal bodies.

Previous studies indicate that the sacrum shows moderate representation, while the coccyx, hyoid, patella, and bones of the hands and feet are poorly represented (Bello and Andrews, 2006; Mays, 1992; Stojanowski et al., 2002; Waldron, 1987). Here, the coccyx and hyoid were the least abundant skeletal elements. As was noted by Bello and Andrews (2006) and also observed in this study, the patella, although poorly represented, is generally well preserved when recovered. In addition, no poorly preserved (<25% present) elements of patella were noted in this study. The hand and foot bones show patterns of representation that are similar to previous reports (Bello and Andrews, 2006; Mays, 1992). The larger tarsals are recovered more frequently than the smaller carpals but this pattern is reversed for the metapodials and phalanges, where manual elements are present in greater abundance than pedal elements. Both Mays (1992) and Bello and Andrews (2006) argue that, in addition to structural characteristics of these elements, excavation bias may play a role in the poor recovery of patella and the pattern of preservation for the hand and foot bones. The small bones of the foot are more easily missed during excavation, than those of the hand. Excavation bias could also partially explain the low representation of the coccyx and hyoid.

In summary, the pattern of preservation observed in this assemblage is largely congruent with other studies. In addition, skeletal representation follows a predictable pattern based on bone size and density. One exception is the greater representation of crania. Early curatorial practices of preferentially retaining crania may explain the abundance of this element. As has been noted elsewhere, excavation bias may play an additional role in explaining the poor recovery of smaller elements.

SEX AND AGE-AT DEATH

Determining demographic information is a staple of osteological analyses. An understanding of the demographic structure of an assemblage allows researchers to investigate evolutionary, ecological and cultural questions (DeWitte, 2018). The application of demographic information is often confounded by a number of issues including small sample size and incomplete, inaccurate or biased data (DeWitte, 2018). These biases could stem from differential preservation or anthropogenic factors like mortuary, excavation or curatorial practices, as well as those inherent to the methods used. In the context of paleopathology, they allow one to identify age or sex specific patterns of disease, trauma or dento-skeletal modifications. In this study, only sex and age-at-death data are reported.

The skeletal assemblage as a whole shows a greater frequency of males (41%) relative to females (32%). This pattern is consistently observed in four of the six institutions included in the analyses (ALB, MMK, NMB and SAM). MMK shows the greatest disparity between males and females but again this is driven by the Gladstone individuals who are presumed to represent a mining community of predominantly men. UP and WITS were the only two institutions with a greater percentage of females relative to males; however, the difference in frequency between males and females was not statistically significant. As Weiss (1972) reports, an abundance of males is often noted in skeletal assemblages and may reflect a bias in the methods used to visually determine sex. This is particularly true of the skull where there is a tendency to attribute skulls with intermediate features to males (Weiss, 1972) and a trend for the skulls of older females to appear more masculine (Meindl et al., 1985). In an assemblage with such a high proportion of skulls, this may be particularly relevant. Differences in the sex ratio may reflect differences in preservation, mortuary practices, mortality or demographic structure of the living population, as well as incomplete sampling and small sample size (DeWitte, 2018; Weiss, 1972). The Gladstone Cemetery finds are a good example of how a heavily biased sex ratio can help lead to inferences about the identity of individuals within an assemblage (van der Merwe et al., 2010b). There is, however, no evidence to suggest that any of the aforementioned factors are responsible for the male bias in the assemblage as a whole, other than the bias resulting from the methods used.

An analysis of the relationship between sex and preservation reveals a predictable pattern. Greater confidence in sex determination is noted with better preservation of the skull and pelvis (the elements used in sex determination for this study). Individuals identified as male/female are generally better preserved than those identified as probably male / probably female, who in turn are better preserved than individuals identified as ambiguous. Poor preservation is the primary underlying factor for individuals for whom sex could not be determined.

The age profile for this assemblage was reported in two ways: binary and ordinal. The binary assessment revealed that the majority of individuals were mature (70.2%). Placement into the more refined ordinal categories was relatively poor and most individuals could only be placed into broader/combined age categories. For example, approximately half the individuals in the assemblage could only be classified as adult (>20 yrs). For adult ordinal categories, the greatest frequencies were observed for middle adults (12.9%), followed by young adults (4.3%) and the lowest frequencies were observed for old adults (2.3%). Similar issues were apparent when classifying subadult remains; however, these age categories were narrower and in general only two or three categories were combined. Frequencies for subadult ordinal categories ranged from 0-6% and no age related pattern of representation was discernible.

Adult aging methods typically evaluate degenerative skeletal changes. This process is influenced by genetic and environmental factors and is only broadly associated with chronological age (see Mays, 2015). Consequently, popular methods for age assessment result in broad age categories which are very imprecise, or narrower age ranges that are more inaccurate (see Buckberry, 2015). Age estimations are further hampered by poor preservation, as well as the inaccuracy and bias introduced by the methods used. Concerning methods, a well-recognised problem is "age mimicry", where the age estimates of the study population are influenced by the age structure of the reference population on which the method was developed (Bocquet-Appel and Masset, 1982; Hoppa and Vaupel, 2002). Another well-known problem is an "attraction of the middle" (Masset, 1989). A phenomenon where the age of young adults is typically overestimated and that of old adults underestimated, resulting in an overestimation of middle-age adults. It is, therefore, likely that the high proportion of middle adults determined in this study may be a reflection of this method induced bias.

The broad age ranges produced by some adult aging methods often overlap multiple ordinal categories and this, in part, explains why the assignment of individuals into discrete categories was so poor. This problem is compounded by poor preservation which affects the number and quality of skeletal age indicators available for assessment. Again, the high percentage of skulls in this collection further contributes to the problem. The relationship between cranial suture closure and chronological age has been questioned (Hershkovitz et al., 1997) and many researchers are aware of the limitations of this method (Falys and

Lewis, 2011). Dayal (2009), investigating a modern South African skeletal population found no significant relationship between suture closure and age.

Juveniles are often underrepresented in skeletal assemblages (e.g. Bello and Andrews, 2006; Buckberry, 2000; Guy et al., 1997; Manifold, 2015; Walker et al., 1988). This has largely been attributed to poor preservation and excavation practices (Guy et al., 1997; Manifold, 2015). Age estimation of juveniles is generally considered more accurate and reliable than adults. There appears to be greater genetic control over skeletal and, particularly, dental development (which underpin juvenile aging methods) than the degenerative changes observed in adults. This results in juvenile ordinal age categories that are narrower than adult categories and allows for more refined age estimates. In this study, juvenile ordinal age estimates were also complicated by poor preservation resulting in estimates that overlap multiple age categories. In addition to greater representation, adult remains are better preserved (greater frequencies of well-preserved individuals) than juvenile remains. This has been reported in other studies (Bello and Andrews, 2006; Walker et al., 1988) and has been linked to the decreased bone density, mineralization and size of juvenile skeletal elements. Some researchers have noted preservation differences among different juvenile ordinal categories (Bello and Andrews, 2006; Guy et al., 1997); however, due to small sample sizes, a similar analysis was not possible for this study.

NON-TB RELATED PALAEOPATHOLOGY

The primary reason for reporting the non-TB related palaeopathology observed in the assemblage was to present the data collected in the process of determining the nature and extent of tuberculosis. A detailed discussion of these observed pathologies is beyond the scope of this thesis.

PALEOPATHOLOGY: TUBERCULOSIS

DIFFERENTIAL DIAGNOSIS

A large number of pathological conditions may cause skeletal changes similar to tuberculosis. These include osteomyelitis, brucellosis, fungal disease (actinomycosis, coccidiodomycosis, blastomycosis), echinococcosis, typhoid spine, neoplastic metastases, primary neoplasms, histocytosis-X, sarcoidosis, rheumatoid arthritis, traumatic arthritis and compression fractures (Buikstra, 1976; Harisinghani et al., 2000; Tuli, 2016). It is possible to exclude some of these conditions, on a case by case basis, depending on the appearance and location of the pathological lesion. Others are rare and infrequently reported. But in many instances the precise aetiology cannot be determined. Given the number of samples presented, the full breadth of possible alternative diagnoses will not be explored in detail for each individual. Instead, the discussion will be restricted to differential diagnoses that are frequently referred to in the paleopathological literature, are common in modern clinical settings or may have been important in the past. All of these individuals have been selected because the lesions are, at least in part, consistent with a diagnosis of tuberculosis; however, diagnostic confidence is not equal in all cases. The goal of this discussion is to highlight which features are consistent with tuberculosis and provide an indication of diagnostic confidence. In the selection of possible cases of tuberculosis, the diagnostic criteria were purposefully kept broad with the hope that ancient DNA analyses would aid in the diagnosis of less confident cases. To aid discussion individuals have been divided into four categories: those showing only proliferative rib lesions, those showing rib lesions and proliferative lesions elsewhere on the skeleton, those with extra-vertebral lytic lesions and those with vertebral lytic lesions.

PROLIFERATIVE RIB LESIONS

Proliferative rib lesions were identified as the sole pathological indicator of tuberculosis in three individuals (MMK 331, NMB 1416 and MRB\20). However, rib lesions in isolation are not sufficient to warrant a diagnosis of tuberculosis. While studies have shown that rib lesions occur more frequently in individuals that have died of tuberculosis, they can occur as a consequence of other pulmonary and non-pulmonary diseases (Kelley and Micozzi, 1984; Matos and Santos, 2006; Roberts et al., 1994; Santos and Roberts, 2006). Thus, in these three cases, a diagnosis of tuberculosis cannot be definitively asserted based on the pathology alone.

Rib involvement in tuberculosis may present as two forms – a lytic form, spread via haematogenous dissemination or direct expansion from an adjacent affected tissue (Ip et al., 1989), or proliferative form, thought to be a diffuse periostitis associated with pulmonary disease (Kelley and Micozzi, 1984). There are few reports of proliferative rib lesions associated with tuberculosis in the clinical literature (Eyler et al., 1996; Guttentag and Salwen, 1999; Varona Porres et al., 2017), while they are frequently reported from anatomical/identified collections (e.g. Kelley and Micozzi, 1984; Roberts et al., 1998; Santos and Roberts, 2006; Steyn et al., 2013). This may be because the proliferative form of rib lesions are of little diagnostic value to the clinician and/or may not easily be observed on chest roentgenograms (Roberts et al., 1998; Santos and Roberts, 2006). As was stated above, studies of anatomical or identified skeletal collections show that rib lesions are most commonly found in individuals known to have suffered tuberculosis but they are not specific to tuberculosis and can occur as a consequence of other pulmonary and non-pulmonary disease. Proliferative ribs lesions occur bilaterally in most cases (Matos and Santos, 2006; Santos and Roberts, 2006) but when expressed unilaterally, it is the left side that is most frequently affected (Kelley and Micozzi, 1984; Matos and Santos, 2006; Santos and Roberts, 2006, 2001). However, Steyn et al. (2013) have reported a greater frequency of unilateral expression with predominantly right rib involvement in a South African series. Furthermore, the vertebral end of the middle ribs are most frequently affected (Matos and Santos, 2006; Santos and Roberts, 2006, 2001) and it has been suggested that, although not pathognomonic, this may aid in distinguishing tuberculosis from non-tuberculosis pulmonary forms (Matos and Santos, 2006).

MMK 331 is an interesting case because this is the only individual for whom the lesions did not appear active (all lesions were completely sclerotic), yet *Mycobacterium tuberculosis* complex (MTBC) DNA was successfully amplified from two different assays (IS*1081* and *rpoB*2). This result may be interpreted in a number of ways. The detection of ancient MTBC DNA in the absence of active lesions may suggest a recurrence of the disease prior to death without visible skeletal involvement. Alternatively, the healed lesions may have a non-tuberculosis aetiology. Another possible explanation is that dormant bacilli were still present within the lesion despite the apparent healing. Lastly, contamination or non-specificity of the aDNA assays is a possibility. Contamination in this instance seems unlikely. No contamination was detected in any of the no-template controls. Master Mix and sample DNA were added to the centre of the plates in the ancient DNA laboratory, whereafter, the plates were sealed. The seal was not removed on this portion of the plate when the DNA standards were added to the plate margin in the modern DNA laboratory. This was the first time that the author had performed the assay and all tests and optimizations had been conducted by other researchers weeks prior to the analysis of these samples, thus reducing the risk posed by carry-over contamination. Non-specificity of the assays is a possibility. Homologues of IS*1081* have been identified in other bacterial species (Mchugh et al., 1997; Picardeau et al., 1996) and the *rpoB* gene is present in many closely related mycobacterial species (Harkins et al., 2015). Although these primers were designed to be specific to the MTBC, our knowledge of bacterial diversity is limited and instances of non-specific amplification may still arise (Harkins et al., 2015). However, while the detection of a single marker may represent spurious amplification, the amplification of two markers lends greater support to the authentic presence of MTBC DNA.

In addition to the rib pathology, MMK 331 also presented with antemortem loss of the left mandibular condyle. The aetiology of this pathology is unclear. One possible explanation for the pathological changes observed is a healed fracture with non-union of the mandibular condylar head. However, pathology as a consequence of neoplastic disease or an infective agent cannot be excluded. Clinical reports on tuberculosis of the mandibular condyle have predominantly focused on primary lesions and these are rare (Assouan et al., 2014). Moreover, while a possible case of tuberculosis of the mandible has been reported in the paleopathological literature, this is not located at the condyle (Lewis, 2011). Steyn and Buskes (2016), in their analysis of skeletal tuberculosis lesions in modern South African anatomical skeletal collections, report a relatively high percentage of individuals (11.5%, n=6) with destruction of one or both mandibular condyles. The authors note that some of these observations could be associated with complete antemortem tooth loss but present at least one case that may be attributed to tuberculosis. Here the authors provide an image of the pathological region and note an associated periosteal reaction. The pathology observed for MMK 331 does not resemble that presented by Steyn and Buskes (2016) and no periosteal reaction was observed in association with the mandibular lesion. While it remains possible that the mandibular pathology observed for MMK 331 represents lytic destruction of the left mandibular condyle due to tuberculosis infection, a traumatic aetiology seems more likely based on the appearance of the lesion. Regardless of the cause, the limited pathology present on the contralateral condyle would suggest the joint remained relatively stable. In summary, although the morphological evidence for tuberculosis is not strong for this individual, the successful amplification of MTBC DNA from two assays lends confidence to a diagnosis of tuberculosis in this case.

NMB 1416 also showed positive amplification but only for a single MTBC assay, *rpoB*2. While all three replicates produced amplicons, and there may well be true detection of MTBC DNA, there may be concerns regarding the specificity of the *rpoB*2 assay (as outlined below). The locations of the rib lesions are consistent with reports of rib lesions from identified skeletal collections. NMB 1416 also shows extensive pathology at the right wrist involving complete ankylosis of the carpus, proximal 2-4th metacarpals and distal ulna and radius. I concur with Morris' (1995) diagnosis of septic arthritis for the pathology at the wrist. Ankylosis is a common complication of untreated septic arthritis (Waldron, 2009) and the appearance of the pathology does suggest infection. While *M. tuberculosis* can cause septic arthritis, staphylococci and streptococci are the more common culprits (Dubost et al., 2002). The pathology at the wrist seems completely healed with no evidence of an active process and the rib lesions, which consist predominantly of woven bone, are indicative of an active process at death; thus, there is no direct association between the pathology at the ribs and wrist. The pathology at the wrist is more likely associated with the healed fracture at the distal ulna. While rib lesions may occur more commonly among individuals with tuberculosis and there is some support from the genetic analysis, a diagnosis of tuberculosis remains tentative for NMB 1416.

MRB/20 was identified from a report in the literature (Steyn et al., 2002) and was not examined by the author or subject to aDNA analyses. In this juvenile, the rib lesions were noted on the lower right ribs along the posterior and lateral aspects and resulted in a thickening of the rib. Santos and Roberts (2006) suggest the occurrence of proliferative rib lesions on the lower ribs may be suggestive of peritonitis rather than pulmonary tuberculosis; however, this conclusion was not supported in another study (Matos and Santos, 2006). Based on the description of pathology, while tuberculosis is one possible diagnosis, other pulmonary and non-pulmonary diseases have been associated with rib lesions and cannot be excluded.

In summary, while a diagnosis of tuberculosis cannot be definitely stated based on the presence of rib lesions alone, for these three individuals, the ancient DNA results suggest at least one, possibly two, of these three cases may have been associated with tuberculosis.

PROLIFERATIVE LESIONS ON THE RIBS AND OTHER POST-CRANIAL ELEMENTS

In two individuals (GLD 8.2 and UP 49), proliferative lesions of the ribs were noted in conjunction with proliferative lesions elsewhere on the skeleton. New periosteal bone formation was noted on the thoracic vertebral bodies and left tibia of GLD 8.2 and on the right scapula of UP 49. New periosteal bone formation was also observed on the long bones of the other two individuals from Gladstone Cemetery but these individuals will be discussed below in different context. A number of studies, in anatomical or identified skeletal collections, have observed or investigated the presence of new bone formation on long bones in individuals suffering from tuberculosis (Assis et al., 2011; Matos and Santos, 2006; Rothschild and Rothschild, 1998; Santos and Roberts, 2006, 2001; Steyn et al., 2013; Steyn and Buskes, 2016). In fact, many of these studies report a high frequency of these lesions. For example, Steyn et al. (2013) and Steyn and Buskes (2016) report frequencies of non-specific lesions of 23.8% (35/147) and 26.3% (54/205), respectively, among individuals that have died of tuberculosis in South African anatomical skeletal collections. However, the correlation between tuberculosis and new periosteal bone formation on long bones appears not to be as strong as has been described for rib lesions (Assis et al., 2011; Rothschild and Rothschild, 1998). The term hypertrophic osteoarthropathy has been used to describe these lesions, although usually a more severe expression thereof, and in the paleopathological literature, a number of studies have linked hypertrophic osteoarthropathy with tuberculosis infection (e.g. Hershkovitz et al., 2008; Masson et al., 2013; Mays and Taylor, 2002). These cases typically represent a more severe expression of periosteal new bone formation and none of the individuals in this study show such severe expression. However, Assis et al. (2011) in their study of the nature and prevalence of these lesions, used this term more generally to describe all symmetrical expressions of new periosteal bone formation (regardless of the extent/severity) on two or more skeletal elements. To conclude, new periosteal bone formation on skeletal elements may be associated with tuberculosis but is certainly not diagnostic of the disease.

As was noted above, new periosteal bone was observed on the ribs bilaterally, some thoracic vertebrae and the left tibia of GLD 8.2. The rib lesions were predominantly present near the sternal half of the ribs and occasionally on the external surface on the right side. The abnormal bone formation on the thoracic vertebral bodies may represent a direct extension of a pulmonary ailment or it may represent a more general expression of new bone formation. The single lesion on the tibia is not suggestive of a systemic process and the more lamellar expression differs from the woven bone observed on the ribs and vertebrae. However, these lesions only weakly adhere to the cortical surface and some may have been lost to post-mortem damage, obscuring the true distribution on the skeleton. The location of the proliferative lesions on the ribs are not typical of tuberculosis (expression on the external surface is rarely reported) but the presence of proliferative lesions on the thoracic vertebra only may suggest a pulmonary ailment. The lesion on the tibia may be unrelated but it seems more likely that it may be part of a systemic process. No MTBC DNA was detected in any of the assays for this individual. None of these lesions provides strong support for a diagnosis of tuberculosis and so the diagnosis for this individual remains tentative.

UP 49 is an unusual case and was identified as tuberculosis from the report of Pistorius et al. (1998). Proliferative lesions are observed on multiple ribs and multiple surfaces of the right scapula. The new bone formation noted on the ribs does not resemble the proliferative rib lesions described in the literature for tuberculosis. Involvement of all rib surfaces with the complete loss of internal structure is typically not noted in tuberculosis. The lesion on the scapula may be a direct extension of the pathology occurring at the ribs. Again this raised, porous and partially sclerotic lesion, visible on multiple surfaces, does not resemble the periosteal reactions typically observed. Proliferative lesions of the scapula have been noted in association with tuberculosis (Assis et al., 2011; Matos and Santos, 2006; Steyn et al., 2013); however, in all of these reports, only the ventral surface was affected. No MTBC DNA was detected in any of the assays for this individual. It is possible that this is an advanced case of periostitis of the ribs and scapula associated with tuberculosis; however, an alternative diagnosis may be more appropriate.

EXTRA-VERTEBRAL LYTIC LESIONS

For the remaining individuals, the pathological process was primarily lytic in nature. In two individuals, MMK235 and NMB 1229, no vertebral lesions were observed but lytic lesions were noted at the right scapula, as well as the right sacroiliac and pubic symphyseal joint surfaces, respectively.

Tuberculosis predominantly affects the spine, after which the joints, particularly the major weight-bearing joints of the hip and knee, are most frequently affected (Aufderheide and Rodríguez Martín, 1998; Ortner, 2003). However, tuberculosis can affect any skeletal element. The bacteria can be disseminated either through haematogenous spread or direct association with an affected soft tissue or skeletal focus (Aufderheide and Rodríguez Martín, 1998). The bacteria will typically localize to haemopoietic marrow and is, therefore, most commonly found in cancellous rich regions of skeletal elements such as the vertebral bodies and metaphyses of the long bones in adults (Aufderheide and Rodríguez Martín, 1998; Ortner, 2003). Extra vertebral tuberculosis can be expressed as tuberculosis osteomyelitis, arthritis or a combination of both (Aufderheide and Rodríguez Martín, 1998; Ortner, 2003). Joint involvement is typically unilateral, and if the synovial membrane is infected both surfaces are usually involved. There is generally minimal formation of new bone (Morse, 1961; Ortner, 2003); however, reactive bone formation can be observed, particularly as a consequence of healing or in association with an overlying abscess, and, although not common, sclerosis of the lesion margin is sometimes observed (Ortner, 2008). Aufderheide and Rodríguez Martín (1998) produce a list of features typically present in tuberculosis of the joints. These include osteopenia of surrounding skeletal elements, marginal erosion, destruction of the subchondral bone, well-demarcated, oval-shaped destructive lesions of the periarticular area, minimal new bone formation and rare sequestra formation.

MMK 235 presented with a lytic lesion on the right scapula body. The main focus of the lesion was along the lateral border, inferior to the glenoid fossa. Four “openings” were visible on the anterior, lateral and posterior surfaces and minimal new bone formation was observed. The features of this individual that are consistent with tuberculosis include the internal origin of the legion suggesting haematogenous spread and the limited presence of new bone formation. Tuberculosis of the scapula is rare in modern clinical settings with only 19 cases reported in the clinical literature, of which 11 were isolated to the scapula (Balaji et al., 2013; Tripathy et al., 2010). Reports involving lytic destruction of the scapula in the paleopathological literature are scant. Mays et al. (2001) reports the presence of a lesion on the scapula of a 35-45 yr old female (NA026) from Wharram Percy, England, who also had lesions on the vertebra and ribs, but the lesion itself is not described. Buikstra and Williams (1991) report a “large channel extending vertically through the glenoid fossa” of 35-39 yr old male from Peru. While some report tuberculosis of the shoulder (Faerman et al., 1997; Kelley and El-Najjar, 1980; Lahr and Bowman, 1992), descriptions are not provided in all cases. The features inconsistent with tuberculosis include the numerous small openings, which may represent cloacae – a typical feature of osteomylelitits; however, extensive new bone formation is often a feature of osteomyelitis which is lacking in this case (Ortner, 2003; Waldron, 2009). In addition to the lesion at the scapula, MMK 235 also presents with ankylosis of the left sacroiliac joint. Tuberculosis of the sacroiliac joint often leads to spontaneous ankylosis of the joint (Seddon and Strange, 1940; Soholt, 1951); however, in this case, the internal surface cannot be observed and there is no evidence of infection (no lytic activity or reactive bone) on the observable surfaces. Thus, it is not possible to demonstrate a relationship between the pathology of the scapula and that of the sacroiliac joint. The lesion of the scapula seems partially consistent with a diagnosis of tuberculosis but other diagnoses cannot be definitively excluded.

NMB 1229 presents with lytic destruction of the right sacroiliac joint and possibly the pubic symphysis. The presence of an abscess associated with the sacroiliac joint is suggested by the depressions visible on the anterior and posterior surfaces. The lesions here are similar, although more severe, than those observed on UCT 552 which is discussed below. Tuberculosis of the sacroiliac joint is relatively rare (although more common than that of the scapula) and it is thought to occur in 2-8% of skeletal tuberculosis cases (Aufderheide and Rodríguez Martín, 1998; Prakash, 2014). Although rare, Ortner (2003) describes it as the second most frequent joint affected by tuberculosis on the pelvis. Possible paleopathological cases are presented in Kelley and El-Najjar (1980), Buikstra and Williams (1991), Pfeiffer (1984), Roberts and Buikstra (2003) and Ortner (2003). Sclerosis at the lesion margin, as was observed in NMB 1229, while not typical, is occasionally observed (Ortner, 2008; Prakash, 2014). Involvement of the pubic symphysis is also rare. There is a report of both sacroiliac and pubic symphysis involvement in the clinical literature (Tsay et al., 1995); however, it is also possible that the pathology at the sacroiliac joint may result in changes to the pubic symphysis without coincident infection. For example, Seddon and Strange (1940), report subluxation at the pubic symphysis after subluxation at the infected sacroiliac joint in 10% (n = 18) of all sacroiliac tuberculosis cases reported in that study. The left pubis was not present/recovered and could, therefore, not be assessed for concomitant pathology. Common differential diagnoses include brucellosis and osteomyelitis. Isolated sacroiliac involvement is more common in brucellosis than tuberculosis and sclerosis of lesion margins is a typical feature of brucellosis (Ortner, 2003; Waldron, 2009); therefore, brucellosis may be a viable diagnostic alternative. Although brucellosis is present among livestock in South Africa, modern reports of human infection are rare (Wojno et al., 2016); however, the historical prevalence of the disease has not been explored in detail (Enslin and Maingard, 1936; Schrire, 1962; van Drimmelen, 1961, 1949). The lack of reactive bone formation would argue against a diagnosis of osteomyelitis or septic arthritis. No MTBC DNA was detected in any of the assays for this individual. In summary, this may represent a case of sacroiliac tuberculosis with possible involvement of the pubic symphysis, however, other diagnoses cannot be excluded.

The remaining individuals all presented with lytic lesions of the vertebrae, either in isolation or in conjunction with lytic or proliferative foci elsewhere in the skeleton. The vertebral lytic lesions for GLD 38.8, SAM-AP 3738 and UCT 552 are all similar in appearance. All have a cavitating, scalloped appearance and predominantly affect the anterior surface of the vertebral body. The lesions present on the vertebrae of GLD 8.3 and SAM-AP 1271, while more similar to each other, differ in appearance to those mentioned above. These C-shaped lesions lack a scalloped appearance and despite significant destruction have not resulted in vertebral collapse. Both cases appear to originate from the discovertebral margin, rather than from within the vertebral body and a dense zone of sclerotic bone is present at the margin of and internal to the lesion. While absent in SAM-AP 1271, large osteophytes have formed along the borders of the lesions for GLD 8.3.

In the spine tuberculosis typically spreads via haematogenous dissemination and localizes at the anterior aspect of the vertebral body near the end plate; however, infection can spread from direct contact with visceral lesions (Aufderheide and Rodríguez Martín, 1998). The disease can spread to the adjoining vertebral bodies by penetrating the intervertebral disc space, through extension beneath the anterior or posterior longitudinal ligaments or haematogenous dissemination (Aufderheide and Rodríguez Martín, 1998). The lower thoracic and lumber vertebrae are most commonly affected (Resnick and Niwayama, 1995). The process is predominately destructive with minimal new bone formation (Morse, 1961); however, extensive new bone formation can be noted in some cases and is often associated with healing or the presence of a paravertebral abscess (Ortner, 2003). Ortner (2008) also notes that lesion margins do typically exhibit some sclerosis. As the disease progresses, the destruction of the affected vertebra can no longer support the weight of the trunk and vertebral collapse ensues (Morse, 1961). This results in kyphosis and gibbus formation, features typically associated with advanced stages of the disease. Paravertebral abscesses are a common occurrence and the neural arch is rarely involved (Morse, 1961).

Vertebral osteomyelitis also results in similar pathology (Ortner, 2003; Waldron, 2009). The term osteomyelitis simply means an infection of the bone and bone marrow (Waldron,

2009) and, as such, many different bacterial infections can result in osteomyelitis, including tuberculosis. However, the term is most commonly used to refer to pyogenic infections that are caused by a group of more commonly found bacteria, most notably those of the staphylococcal family (particularly *Staphylococcus aureus*), followed by streptococcal infections (Ortner, 2003). The bacteria are disseminated to skeletal elements most often though haematogenous spread; however, direct infection can result through traumatic disruption of tissue (Waldron, 2009). The lumbar vertebrae are most frequently affected, followed by the thoracic vertebrae (Calhoun and Manring, 2005). In the spine, osteomyelitis can be indistinguishable from tuberculosis in the early stages of disease (Kelley and El-Najjar, 1980). The infection often proceeds rapidly affecting adjoining vertebra and can result in vertebral collapse (although this occurs less frequently than in tuberculosis); there is also more frequent involvement of the posterior spine, greater sclerotic bone formation and paravertebral abscess formation is less common (Aufderheide and Rodríguez Martín, 1998; Kelley and El-Najjar, 1980; Ortner, 2008). Many of these features are also observed in spinal tuberculosis, making it difficult to distinguish between the two diseases.

The spine and sacroiliac joints are the most common sites of skeletal brucellosis infection (Al-Shahed et al., 1994; Ortner, 2003; Turan et al., 2011). In the spine, the lumbar vertebrae are most frequently affected (Al-Shahed et al., 1994). Vertebral destruction usually occurs at the discovertebral junction, at the anterior aspect of the superior surface (Al-Shahed et al., 1994; Ortner, 2003). However, lesions may also be isolated to the inferior surface (Al-Shahed et al., 1994; Waldron, 2009). There is characteristic osteophyte formation bordering the lesion known as a “parrots beak” osteophyte or sign of Pedro-Pons when it occurs at the anterior margin (Al-Shahed et al., 1994; Ortner, 2003; Waldron, 2009). Also typical of brucellar infection is the formation of sclerotic bone along the internal margin of the lesion (Al-Shahed et al., 1994; Ortner, 2003; Waldron, 2009). Due to the slow progression of disease, spinal brucellosis rarely results in vertebral collapse and kyphosis, even in cases of extensive lytic destruction (Al-Shahed et al., 1994; Ortner, 2003; Waldron, 2009). Multiple non-contiguous vertebral involvement and multifocal disease is also more commonly observed in brucellosis than tuberculosis (Ortner, 2003). A number of reports exist in the paleopathological literature, some of these are summarized in D’Anastasio et al. (2011). Additional reports include Anderson (2003), Curate (2006) and Mutolo et al. (2012).

Furthermore, molecular detection from ancient remains has been reported in Mutolo et al. (2012) and Kafil et al. (2014), while Kay et al. (2014) report the retrieval of an ancient brucellar genome.

GLD 8.3 was identified from the literature (van der Merwe et al., 2010b). This individual presented with lytic lesions on the inferior surfaces of L4 and L5, as well as the olecranon of the right ulna and, possibly, the left patella. Many of the vertebral features, lesions originating at the discovertebral margin, destruction without vertebral collapse, sclerotic bone formation internal to the lesion, and osteophyte formation, are, in combination, more consistent with a diagnosis of brucellosis than tuberculosis. These lesions, however, do not resemble cases of brucellosis in the paleopathological literature, most of which present minor to moderate changes to the superior anterior margin or more diffuse forms, but it does fit the clinical description and very closely resembles a case from the clinical literature presented in Al-Shahed et al. (1994, Fig. 10). The lytic lesion present at the olecranon of the ulna may be linked to brucellar bursitis. Although rare, associated skeletal involvement has been reported in the clinical literature (see Turan et al. (2011). Many of these features, though not common, may occur in tuberculosis infection as well and, therefore, tuberculosis should not be entirely discounted. Based on the multifocal nature and appearance (limited new bone formation and destruction) of the lesion, osteomyelitis seems an unlikely diagnosis. GLD 8.3 also produced positive amplification of the *rpoB*2 assay which may lend support to a preferential diagnosis of tuberculosis; however, there may be issues of non-specificity with this assay.

In SAM-AP 1271, the lytic lesion present on the inferior surface of L4 resembles those observed on GLD 8.3. The major difference is that SAM-AP 1271 lacks osteophyte formation in association with the lesion. However, there is still significant formation of sclerotic bone internal to the lesion. Unfortunately, these remains are only partially complete and the full distribution of pathology cannot be assessed. No MTBC DNA was detected in any of the assays for this individual. While this lesion may be attributed to tuberculosis, other diagnoses, particularly brucellosis cannot be definitively excluded.

Of the three individuals with similar scalloped lesions (SAM-AP 3738, GLD 38.8 and UCT 552), SAM-AP 3738 is the only one with a single isolated vertebral focus (T12). There is limited new bone formation. There are minor changes noted on the adjacent vertebra but no evidence of vertebral collapse or abscess formation. The changes observed for the affected vertebra are consistent with tuberculosis but other causes of lytic vertebral lesions including osteomyelitis and brucellosis should not be discounted.

GLD 38.8 presents with multiple lytic lesions on at least two (L3 and L4), possibly three (L5), vertebral bodies. Periosteal new bone formation is also observed on the right ribs, as well as the diaphysis of the radii, tibiae and fibulae. The lesion present on the anterior surface of L3 and the possible lesion on the anterior surface of L5 may suggest an extension of infection beneath the anterior longitudinal ligament from an initial focus at the anterosuperior aspect of L4. The presence and nature of the vertebral lesions, together with the rib and long bone lesions are suggestive of pulmonary infection with vertebral involvement. This is also the only individual for which MTBC DNA was detected in all three assays. The DNA evidence, together with the morphological evidence, provides a sound argument for tuberculosis in this case.

Finally, UCT 552 is the only individual with evidence of vertebral collapse. Lytic destruction of three adjacent vertebrae, T6-8, is observed. Separation of the neural arch and body of T7 occurred antemortem and there is evidence of an unreduced subluxation involving all associated zygapophyseal joints. Minimal new bone formation is observed in association with healing of the collapsed vertebrae; however, significant reactive bone is observed along the anterior and left side of C1-L1 vertebral bodies, suggesting the presence of a large paravertebral abscess. Furthermore, there is partial lytic destruction of the left sacroiliac joint and new periosteal bone formation on the ribs bilaterally, in addition to lytic involvement of one rib head. The pathology at the sacroiliac joint is similar, but less severe in extent, to that observed for NMB 1229. The presence of an abscess is also suggested by the depressed and partially eroded region adjacent to the joint on the posterior surface of the sacrum. The sacroiliac lesion may represent a separate haematogenous focus, or infection may have seeded to the joint via an abscess associated with the vertebral pathology. The rib lesions predominantly affect the vertebral end of the pleural surface of the middle ribs but some new bone is observed along the external surface of severely pathological ribs. Thickening of the proximal left tibial shaft may suggest an additional focus of infection; however, the association of this lesion with the others is unclear. The morphological pathologies observed for this individual are highly consistent with a diagnosis of tuberculosis. This individual was also positive for two MTBC markers (IS*6110* and *rpoB*2) adding further support for a diagnosis of tuberculosis.

In summary, the most confident diagnoses of tuberculosis in South African Holocene collections are observed for GLD 38.8 and UCT 552. Both morphological and aDNA evidence support this diagnosis in these two cases. MMK 331, while only presenting with healed rib lesions, showed amplification of two MTBC markers, lending some confidence to a diagnosis of tuberculosis in this individual. SAM-AP 3738, SAM-AP 1271 and GLD 8.3 all present with vertebral lesions of the lower thoracic or lumbar spine, which is typical of tuberculosis; however, other diagnoses such as brucellosis and osteomyelitis can not be excluded. NMB 1229 and MMK 235 both have extra-vertebral lytic lesions that, although consistent with tuberculosis, cannot be exclusively associated with the disease. Finally, proliferative rib lesions are the predominant pathology observed for the remaining individuals (GLD 8.2, UP 49, NMB 1416, MRB\20) and, while often associated with tuberculosis, are not specific to the disease.

DNA

The successful amplification of ancient DNA (aDNA) in this study adds to a growing body of successful ancient DNA studies within South Africa. These studies have investigated a range of questions, including those concerning human origins and population affinities (Morris et al., 2014; Schlebusch et al., 2017), domestication (Horsburgh and Rhines, 2010; Horsburgh, 2008; Horsburgh et al., 2016; Horsburgh and Moreno-Mayar, 2015; Orton et al., 2013), diversity within modern mammalian taxa (Whitehouse and Harley, 2001) , and resolving phylogenetic relationships among modern and/or extinct mammalian taxa (Orlando et al., 2009). This is the first study to report successful amplification of ancient pathogen DNA within South Africa. Peckmann (2003, 2002) attempted to amplify Variola species DNA in a study of smallpox infection in a South African population; however, these attempts were unsuccessful.

The successful detection of human mitochondrial DNA (mtDNA) in all samples submitted to aDNA analysis in this study is a promising result for South African aDNA research, which had shown limited success until technological and methodological advances within the last decade (Morris, 2015). Although the results are not reported here, mitochondrial genomes were obtained for some of these individuals using next-generation sequencing (NGS). Sequencing of conventional and quantitative PCR (qPCR) amplicons was not attempted. The insertion sequences (IS*6110* and IS*1081*) share complete sequence identity within the MTBC and, while sequencing may allow the detection of non-specific amplification (e.g. Müller et al., 2015), it does not provide sufficient information to discriminate among MTBC lineages/strains or discriminate against modern laboratory, "carry-over" contamination (Wilbur et al., 2009). Quantitative PCR was used as a screening tool to identify candidate individuals for target-enriched NGS (Harkins et al., 2015). Obtaining MTBC genomes is the ultimate goal and could allow for the confirmation of paleopathological diagnoses and identification of strain type, as well as provide information concerning the evolution of this pathogen over time. Furthermore, an analysis of DNA fragment length and damage profiles can be used to detect and quantify contaminant sequences (Briggs et al., 2007; Key et al., 2017). Therefore, NGS is a more powerful tool than conventional Sanger sequencing of PCR products in aDNA studies. In this study, MTBC qPCR targets were successfully amplified in five individuals, three of which showed successful amplification for more than two assays; however, attempts to obtain MTBC-specific genomic sequences were unsuccessful. Thus, information regarding the identity, nature and evolution of the organism responsible for these infections in South Africa remains elusive.

While concerns have been raised as to the specificity of commonly used MTBC markers due to the presence of analogous sequences in other bacteria (Harkins et al., 2015; Müller et al., 2015; Wilbur et al., 2009), the detection of multiple MTBC markers in at least three individuals adds support to a diagnosis of tuberculosis for these individuals. These results should be interpreted with caution, however, since they have not been successfully replicated in an independent laboratory or confirmed through sequencing. This study has revealed concerns about the *rpoB*2 assay. The *rpoB* gene is present in a single copy, while the two insertion elements are typically present in multiple copies (although MTBC isolates lacking any IS*6110* elements have been reported (van Soolingen et al., 1993)). Although many factors may influence the success of an assay, including factors intrinsic to the assay (e.g. primer design, reagent concentrations) and those intrinsic to the template DNA (e.g. presence/absence/starting quantity of template DNA, presence/quantity/nature of inhibitors), it is concerning that an assay detecting a single copy gene, *rpoB*2, should outperform those detecting the multicopy insertion elements, IS*6110* and IS*1081*. While every attempt was made to assure the specificity of the *rpoB*2 assay, the authors note that the ability to design a specific assay is restricted by our current knowledge of bacterial diversity and the availability of bacterial sequences (Harkins et al., 2015). Therefore, the detection of *rpoB*2 in the absence of the other two markers, may indicate a lack of specificity of the assay. As a consequence, the two individuals (NMB 1416 and GLD 8.3) in which only *rpoB*2 was detected must be interpreted with caution.

These results highlight the challenges faced in ancient pathogen research, particularly in warmer climates, where DNA preservation is typically not as good as the more temperate regions (Hofreiter et al., 2015). In addition to problems common to all aDNA studies (see chapter 3 for a review), ancient tuberculosis research faces challenges of high sequence similarity among closely related species compounded by a limited knowledge of bacterial diversity. In addition, all of the individuals in this study have a post-exhumation history of at least a decade and in some cases a century, which has likely further negatively impacted DNA preservation. It should be noted that a failure to detect MTBC DNA is not proof of its absence. An incorrect diagnosis is one possible reason for unsuccessful amplification of MTBC DNA; however, variability in aDNA preservation in general, variability in pathogen preservation specifically and the co-extraction of PCR inhibitors are other factors which may affect the success of an assay. While poor DNA preservation in warm African climates may preclude successful recovery of ancient MTBC genomes in most cases, the field of ancient DNA research is constantly evolving and the application of new technological and methodological advances may yield successful results in the future.

GENERAL SUMMARY

PRESERVATION / NUMBER OF CASES IDENTIFIED

Extrapulmonary tuberculosis is observed in 8-24% of modern medical tuberculosis cases (WHO, 2018). Skeletal tuberculosis accounts for an estimated 30% of extra-pulmonary cases and 3% of the total (Ortner, 2003). Caution should be taken when extrapolating modern rates of extrapulmonary tuberculosis to the past. Differences may result due to changes in genetic and environmental factors. Furthermore, modern prevalence may be variably affected by HIV comorbidity. Factoring in differential preservation of archaeologically derived remains, the prevalence of discernible skeletal tuberculosis is expected to be low. Proliferative rib lesions are expected to be observed more frequently and represent pulmonary rather than skeletal tuberculosis. While the relative frequency of cases identified in this study may seem extremely low (0.52% of the total number of individuals observed (N=2321), and even less if restricted to convincing cases only), particularly for a country with such a high burden of disease, it is difficult to make a fair comparison with frequencies observed in other studies due to the broad geographical and temporal range represented in this dataset. The remains examined span the Holocene and for most (~3/4) the date has not been refined further. If the denominator is restricted to absolute or relatively dated remains that fall within the colonial period, a higher frequency (3.4%, N=378) is observed; however, restricting the denominator to individuals for which a date is known will likely greatly underestimate the number of historical remains in the assemblage and, therefore, overestimate the frequency of skeletal tuberculosis.

SEX

Of the twelve possible cases of tuberculosis identified in this study, the majority (n=8) were male. Only three individuals were female and one a juvenile. Globally, a gender bias is noted with a higher tuberculosis incidence and mortality among men (WHO, 2018). This pattern is also observed in South Africa but the burden of disease among women is increasing. McLaren et al. (2015) report a national female-male tuberculosis incidence sex ratio of 0.83, indicating a higher burden of disease among South African men versus women, but also highlighting a higher burden among South African women relative to the rest of Africa (0.71) and other regions like South East Asia (0.5) and Europe (0.43). This relatively high rate of tuberculosis among women is being driven by the concurrent HIV epidemic which disproportionately affects young women (McLaren et al., 2015).

A number of factors, biological and socioeconomic, have been suggested to explain the higher rates of tuberculosis globally among men. These include biological differences in disease progression, differences in health and nutritional status, an occupation-linked elevation of risk among men, larger social networks among men and, finally, gender differences in detection linked to care-seeking practices (Hudelson, 1996). In South Africa, labour migrancy to urban centres, mines and white-owned farms has disproportionately involved men and this may very well have resulted in larger social networks for men than women. Furthermore, men were likely working in areas/among populations with higher tuberculosis prevalence and the nature of the occupation itself may have carried greater risks. For example, the occupational risks associated with mining were discussed in chapter 2. It should, however, be noted that a greater frequency of males are reported in this dataset and this may be at least partly driving a higher frequency of males among the tuberculosis subset.

AGE

All but one of the individuals identified in this study were adults at death. Most were estimated to fall within the middle-adult age category with only two individuals identified as old adults and, due to poor preservation, one individual could only be identified as adult. In modern South African populations, tuberculosis predominantly affects people of reproductive age (WHO, 2018). Due to the poor, irregular and haphazard collection of statistics among the indigenous populations of South Africa during the period represented by the identified cases (colonization-early 20th century), it is difficult to ascertain a broad sense of age-related tuberculosis mortality patterns. Skeletal tuberculosis appears to have been more prevalent among juveniles in the pre-chemotheraputic era (Ortner, 2003). Although limited in scope, MacVicar (1935) report of skeletal tuberculosis in the Ciskei from 1927 to 1935 shows a greater number of juveniles treated for skeletal tuberculosis. This pattern is not, however, typically reflected in archaeological remains at least partly owing to the poor preservation of juvenile remains. In this regard, juveniles are poorly represented in this collection constituting <29.8% of the total number of remains for which age-at-death could be determined.

GEOGRAPHICAL LOCATION

The vast majority of the cases are from the northern regions of South Africa. Specifically six were from the Northern Cape, three from the North West and one from the Free State. The remaining two individuals were from the Western Cape. This pattern does not represent the modern or historical distribution of disease and may be spurious as a consequence of small sample size.

WHAT CAN WE SAY ABOUT THE EMERGENCE AND SPREAD OF TUBERCULOSIS IN SOUTH AFRICA?

NO EVIDENCE OF PRECOLONIAL TUBERCULOSIS

In chapter 2, the potential for a precolonial presence of tuberculosis was discussed in detail. In summary, genetic studies have suggested an African origin for tuberculosis based on greater genetic diversity found on the continent (Comas et al., 2015; Gutierrez et al., 2005; Hershberg et al., 2008; Wirth et al., 2008). It should, however, be noted that modern patterns of genetic diversity may not necessarily reflect past patterns of diversity and other regions of origin should not be ruled out. Based on the presence of exotic trade items at archaeological sites, we know that populations in South Africa were part of the Indian Ocean Trade Network from at least the 8th century AD. Thus, prior to colonization, opportunities for disease transmission did exist. Unfortunately, the indigenous people of South Africa did not develop an independent writing system. Consequently, our current knowledge of the emergence of tuberculosis is dependent on the observations of early colonial explorers, missionaries and settlers, as well as indirect lines of evidence that include perceived immunological naivety, population size, geographical disease distribution and the nature of contact. Inferences based on historical observations generally suggest an absence of the disease. Criticisms of these inferences and the indirect lines of evidence are discussed in chapter 2. Based on a review of the literature, the conclusion reached was that tuberculosis was either never present among the indigenous people of South Africa, was periodically introduced but did not become endemic or was present but went undetected by early settlers, explorers and missionaries. While the contribution of the skeletal record to answering this question has received much attention in the America's and Europe, outside of Egypt, little research in this regard has been conducted in Africa (Roberts and Buikstra, 2003). Thus, it has been the purview of this study to investigate the origins of tuberculosis using the South African skeletal record.

One individual, MMK 235, possibly predates the colonial period. A radiocarbon date of 390±50 BP was obtained for this individual (Morris, 1992b; Vogel and Fuls, 1999). This produces a calibrated date of AD 1428 to AD 1666 (at 99.7% probability) using the SHCal13 atmospheric curve (Hogg et al., 2013) in OxCal (v4.3.2, Ramsey, 2017). Colonial settlement postdates AD 1652; thus, while it is possible that the burial falls within the colonial period, given the location of the burial in the interior, it seems likely that at the very least the burial predates direct European contact, which occurred first at the Cape. The lytic lesion of the scapula provides only weak evidence for a diagnosis of tuberculosis and this individual was not included in the genetic analysis; further analysis of this individual may be an interesting avenue for further research. The individual was unearthed near the Riet River, Koffiefontein by W. Fowler, an amateur archaeologist, who excavated a number of burials from the Koffiefontein area in the first half of the 20th century. These burials have been associated with the type R settlements in the region that were first investigated in detail by Maggs (1971). These settlements are identified by the presence of stone-walled structures that delineate a centrally located livestock pen, with smaller structures (associated with domestic activities) located along the periphery of the central structure (Maggs, 1971). The individuals associated with these settlements are thought to have been hunter-gatherers who acquired stock and adopted the practice of stock-keeping through interactions with Tswana populations further north (Humphreys, 2009; Maggs, 1971; Morris, 1992b). Despite the evidence of stock-keeping these communities appear to be distinct from the KhoeKhoen and were following a predominantly hunter-gatherer economy (Maggs, 1971). This hypothesis, that these were hunter-gathers who kept stock, is supported by the archaeology (Maggs, 1971) and accounts from early explorers and missionaries who describe encountering “Bushmen” with livestock (see Humphreys, 2009; Maggs, 1971; Morris, 1992b). Based on the reports of European observers, by the early 19th century these “Bushmen” with stock were no longer present along the Riet River. The communities inhabiting the type R settlements were likely displaced by / incorporated into the Griqua, Kora and Tswana as a consequence of colonial encroachment and the upheavals of the *Mfecane/Difaqane* (Maggs, 1971). Studies of the Koffiefontein skeletal remains and dentition support population continuity with other Khoesan / earlier Holocene remains and suggest that there was little genetic input from Bantu-speaking populations (Black, 2014; Irish et al., 2014; Morris, 1992b). However, isotopic analyses indicate that at least some of the Koffiefontein individuals had a C_4-rich diet consistent with the Iron-Age population included in that study (Masemula, 2015). Masemula (2015) has suggest two possible explanations for this result. The favoured scenario involves the incorrporation of individuals from Iron-Age communities, albeit morphologically indistinct individuals, into the Koffiefontein population. Alternatively, the Koffiefontein individuals were acquiring food from their Iron-Age neighbours. However, only a few individuals showed high C_4 dietary signals. Morris (1992b) cautions that not all of the Koffiefontein burials can be clearly associated with the type R settlements; however, based on the grave goods recovered with this individual, MMK 235 is thought to be associated with the type R settlements. This individual is also one of the few that show an isotopic C_4 dietary signature consistent with Iron-Age populations (Masemula, 2015). Although a diagnosis of tuberculosis cannot be confidently stated for this individual based on the observed pathology alone, should this represent a case of tuberculosis it may be a very significant case. It would not only potentially pre-date colonial settlement but may also suggest a route of transmission for the disease. This is discussed in greater detail below.

A calibrated radiocarbon date is also available for UP 49. The authors suggest that the burial is most consistent with the earlier two of three calibrated dates provided: AD 1670(1682)1702 and AD 1716(1745)1784 (Pistorius et al., 1998). This individual was unearthed near the entrance of a kraal from an Iron-Age, Batswana settlement on Makgope Hill near Rustenburg in the North West Province. While both of these date ranges fall within the colonial period of South Africa, recorded European expeditions into the region are only noted from the early 1820s and large-scale European settlement in the region did not occur until the 1830s (Bergh, 1999). Therefore, while the interment occurred during the colonial period, similar to MMK 235, it likely predated direct European contact. The settlement from which these remains were uncovered is thought to be affiliated with the Bakwena Baphalane, an ancient Batswana clan (Pistorius et al., 1998). The first half of the 19th century has been described as a period of great unrest among the Bantu-speaking peoples of South Africa. This period, often termed the *Mfecane*/*Difaqane*, has been the subject of much debate. Specifically, the causes, extent of violence and displacement, and even its very existence have been questioned (e.g. Cobbing, 1988; Etherington, 2004; Hamilton, 1995). Parsons (1995), referring to the region north of the Vaal River, has posited that the turmoil of the early 19th century may be rooted in events occurring in the preceding two centuries. Similarly, Manson (1995), in his discussion of the Tswana of the Highveld, also concludes that the increased competition for resources and trade, state formation and conflict that culminate in the *Mfecane*/*Difaqane*, emerge decades earlier in this region. Morton (2008) has, however, challenged these views. His research mapping settlement location based on historical oral traditions, has indicated stability in settlement patterns among different groups in the region through the 18th century until the arrival of the Pedi in the 1820s. He has, therefore, argued that if the late 18th to early 19th centuries were marked by increased violence in the region, as Parsons and Morton suggest, this was not reflected in the settlement patterns. Although the extent and timing of these disruptions remain unresolved, it is possible that the events of the century preceding the *Mfecane*/*Difaqane* may have resulted in ideal conditions (including stress, famine and war) for the spread of tuberculosis. However, the proliferative lesions present on the ribs and scapula could have multiple causes of which tuberculosis is one. Attempts to amplify MTBC DNA from this individual were also unsuccessful. Thus, in this case, as with MMK 235, the diagnostic confidence is tenuous.

In both of these cases, MMK 235 and UP 49, the disease could have been European in origin without direct European contact. The Portuguese had, by the end of 15th century, rounded the southern tip of Africa and by the 16th century entered, and subsequently dominated, Indian Ocean trade (Thompson, 2001). By the end of the 16th century, other European nations had begun to make more regular use of this southern route to the East. Initially, trade along the eastern coast of African as far south as Delagoa Bay (now Maputo) was relatively minor and at times sporadic but by the late 18th century trade at Delagoa Bay had intensified (Newitt, 1995). By this time, the Dutch had established the already expanding colony at the Cape. There was, however, no permanent European settlement along the east coast of South Africa until the 19th century (Holden, 1855). As a consequence of the Indian Ocean trade, the settlement at the Cape and occasional shipwrecks along the South African coast, tuberculosis may have made its way into the interior via overland trade networks or direct spread with interacting populations. It is, therefore, not possible to rule out a European origin for the disease, if the lesions present are in fact representative of tuberculosis. It is interesting to note that this early possible evidence of tuberculosis occurs in Bantu-speaking, specifically Tswana populations, or communities interacting with them. McVicar (1908) reports contradictory evidence to the general consensus that tuberculosis was absent among the indigenous people of South Africa from some of the Zulu communities of Natal. While individuals in this study are located further inland and are likely of Tswana affinity, this may lend credence to an early presence of disease among the Iron Age populations of South Africa. This may implicate Indian Ocean trade as an early route of transmission whereby tuberculosis spread via contact with European or even possibly Asian or Arabic traders. The evidence for exotic trade items is, however, scant. Six glass beads were uncovered with one of the Koffiefontein individuals (MMK 228) (Humphreys, 1970), although, the temporal relationship of this individual to MMK 235 is unknown. Maggs (1971) argues that while these could have come from the Cape, trade with neighbouring Tswana populations seems more likely given Wikar’s (Mossop, 1935) historical (1779) accounts. He also attributes the marine shell ornaments to trade with the Cape or Natal, while copper is associated with trade with the Tswana. Pistorius et al. (1998) do not report any exotic trade items associated with UP 49 and the Makgope site. It could be argued that the presence of exotic trade goods demonstrates involvement in trade networks through which tuberculosis may have spread. However, the absence of these items does not preclude involvement in such trade networks (the items may simply not have been recovered). Furthermore, populations may have been in contact but trading other locally sourced/produced items. An alternative explanation for the presence of tuberculosis in these Tswana/Tswana-affiliated populations is that the disease may have followed populations as they migrated into the region. It should again, however, be stressed that the evidence for tuberculosis for these two individuals is tenuous and, therefore, these assertions remain speculative. While it is possible that these represent cases of tuberculosis, they cannot be used as support for a precolonial presence of the disease in South Africa.

EARLY FOCUS OF DISEASE AT THE CAPE

The earliest convincing evidence of tuberculosis comes from two individuals, SAM-AP 3738 and UCT 552, unearthed from an unmarked informal burial ground in Green Point, Cape Town. In the case of UCT 552, the lytic vertebral and sacroiliac lesions, the proliferative lesions on several vertebral bodies and ribs and the successful amplification of multiple MTBC markers provide strong support for a diagnosis of tuberculosis. The evidence is not quite as strong, but still convincing, for SAM-AP 3738, which shows lytic destruction of the 12th thoracic vertebra only. This individual was not subject to genetic analysis. The Green Point burial ground is located outside the formal boundaries of historically documented cemeteries in the area and is thought to have been used predominantly by the lower classes of 18th century settler society (Apollonio, 1998; Cox, 1999; Finnegan et al., 2011). An early focus of the disease in the first colonial city of South Africa correlates well with the historical literature. The large scale immigration of tuberculous individuals seeking a climatic cure for the disease did not become popular until the 19th century. It had, however, already become a notable disease in the century prior. The earliest reports in the literature date to the late 18th century and note consumption as the most frequently occurring disease among residents of the Cape (de Jong, 1802; Percival, 1804). Cape Town was not only the first colonial city but also the only formal port of entry in South Africa until the 19th century. Tuberculosis was rife in Europe during the 17th and 18th centuries (Murray, 2004b) and the disease was undoubtedly imported on numerous occasions by visiting or immigrating Europeans. In fact, the Dutch East India Company's initial intention at the Cape was to establish a garden (to provide fresh produce) and a hospital (to treat sick sailors attempting the long voyage from Europe to the East) (Davenport and Saunders, 2000). European immigrants were probably not the only source of tuberculosis for the local population. The forced immigration of African and Asian slaves, convicts and political prisoners likely also contributed to the burden of disease. These individuals may have also contracted the disease via European contact but it is also possible that tuberculosis was endemic to regions within Africa and Asia prior to European expansion in the 16th century.

The individuals interred at the informal burial ground in Green Point were those who could not be buried on consecrated ground or could not afford to purchase a burial plot within the formal cemetery of the Church. These included suicides, non-Christians, the lower classes of colonial society (including servants, seamstresses, etc), poor sailors, slaves, freed slaves and their descendants, paupers and criminals (Finnegan et al., 2011). Both individuals, UCT 552 and SAM-AP 3738, were males who are estimated to have died between the ages of 35 and 50 years.

Isotopic (Cox et al., 2001; Kootker et al., 2016) and genetic results indicate that UCT 552 was likely brought to the Cape as a victim of the slave trade. The new colony was heavily dependent on slave labour (Worden et al., 1998) and an estimated 63 000 slaves were imported to the Cape in the early colonial period (1752 to 1808) (Shell, 1994). Slaves at the Cape were diverse in origin hailing predominantly from India (25.9%), Indonesia (22.7%), Africa (26.4%, particularly East Africa) and Madagascar (25.1%) (Shell, 1994). The experience of slaves at the Cape was by no means universal (Percival, 1804). Slaves experienced different degrees of autonomy and physical violence at different levels of measure (rural vs. urban, Company (VOC) vs. private, one household vs. another) (Shell, 1994). Shell notes that “all involuntary labour is ultimately based on violence” (pp. 206), although control was not always effected through physical violence. While the exact conditions of life of UCT 552 will never be known (e.g. adequacy of nutrition and housing, extent of physical and emotional stress), including where the disease was contracted (abroad, on a slaving vessel, at the colony?), slaves were undoubtedly a population vulnerable to diseases of poverty, like tuberculosis. The brutality of slavery and crippling disease stands in stark contrast to the care of the burial, hands folded across the pelvis with personal items included (Apollonio, 1998), and serves to highlight an aspect often difficult to glean from the history books of Cape slavery, the life of the individual. Slave history is often told as a collective history. In America, the stories of emancipated slaves were collected and complied giving an individual account of slavery (Library of Congress, 2001) but this did not occur in South Africa (Murray, 2010). Instead, we must rely on limited historical accounts/records and archaeological investigations to unveil these neglected histories.

The population affinity of SAM-AP 3738 was not determined in this study; however, Black (2014) has identified a number of Khoesan dental traits present in this individual. As is noted above, early reports indicate that tuberculosis was the most common complaint among Europeans or those of European descent. Laidler and Gelfand (1971), however, note that the disease was relatively uncommon among the Europeans of the Cape (probably compared to disease prevalence in Europe) but that it was frequent among the "Hottentots". Unfortunately, no information about the burial, other than its location, is available for this individual.

Although a sample size of two hardly represents abundant evidence, nevertheless the presence of tuberculosis in these two individuals does support an early occurrence of the disease in the most impoverished and disenfranchised of the early Cape society. This included slaves, Khoesan and their descendants. This is not unexpected. Modern tuberculosis is considered a disease of poverty, and this appears to have been the case in South Africa in the past as well. This differentiates South Africa from other regions such as Europe, where the disease was much less discriminating during its peak in the 17th and 18th centuries. According to parish records in England, an estimated one in four deaths was attributed to tuberculosis (Davis, 2000). In contrast, from early in its colonial history reports of disproportionate disease burden among the poorest communities within or near the Cape are apparent; thus setting the stage for a pattern that would last for centuries and laying the base for the modern tuberculosis epidemic in South Africa today.

TUBERCULOSIS ALONG THE ORANGE RIVER IN THE 18TH AND 19TH CENTURY

Two individuals, NMB 1416 and SAM-AP 1271, were interred near the banks of the Orange River in the Northern Cape. NMB 1416 was likely a middle-adult male, while SAM-AP 1271 was an old-adult female.

The European traveller George Thompson (1827) remarked on his travels along the Orange River in 1824 that the Korana were "very subject to consumption", further noting that all the "Hottentot tribes" were. It is clear then, that by the early the 19th century tuberculosis had become established among the Khoekhoen and mixed communities living beyond the formal boundaries of the colony as far north as the Orange River. Throughout the 18th and 19th centuries, enterprising, disaffected and outlawed individuals began to migrate north from the colony at the Cape. These included runaway slaves, individuals of mixed ancestry (termed “Bastaards” or “Hottentot-Bastaards”), culturally assimilated Khoesan, pastoralist farmers of European descent (*trekboers*), European deserters, criminals/outlaws and missionaries (Leśniewski, 2010; Penn, 2005, 1995). The nature of interactions between these individuals and the San and Khoekhoen were variable. Interactions ranged from amicable, although not always equal (e.g. trade, intermarriage, assimilation, seeking/providing refuge), to violent (e.g. warfare, raiding) or involved the provision of services (e.g. clientage, religious ministration) (Penn, 1995). However, as the 18th century progressed and the colony continued to expand, these interactions came to increasingly disrupt the autonomy and lifeways of the Khoekhoen and San. By the end of the 19th century, as a consequence of disease, drought, conflict, loss of land, participation in a wage-based economy and the expanding influence of the colony, little remained of the pastoralist lifeways of the Khoekhoen (Boonzaier et al., 1997). Some Khoekhoen/descendant communities migrated to north to escape the turbulence created by the colonial frontier. Of those that remained, many were, in effect, forced to labour on settler farms (Boonzaier et al., 1997). Some were able to retain a claim to land through an association with missionaries and the establishment of permanent mission stations but as the 19th century progressed, conditions for these communities and their status in colonial society increasingly declined (Boonzaier et al., 1997; Kelso and Vogel, 2015).

NMB 1416 is one of a number of remains unearthed by Dreyer and Meiring in 1936, located near the modern town of Kakamas. This particular individual was unearthed near Augrabies Falls on the farm Rooipad, located along the south bank of the Orange River. Radiocarbon dates have been obtained for some of the Kakamas burials and these, in conjunction with imported glass trade beads, indicate a mid- to late-18th century interment for the majority of the remains (Morris, 1992b). A Christian style burial (extended on back) suggests a possible early 19th century interment for at least some of the burials. The identity of these individuals has been a somewhat debated topic. Early physical anthropologists working in South Africa were heavily influenced by a typological approach to understanding human variation (Morris, 2012) and, consequently, the Kakamas individuals were thought to represent “pure” Khoekhoen (who at the time were thought to be of Hamitic origin) of the “Kakamas type” (See Morris, 1995, 1992b). In the second half of the 20th century, researchers began to question typological assessments of this nature. (Morris, 1992b), in his statistical analysis of the morphology of the skull, has shown that the Kakamas individuals likely represent an admixed population who share morphological affinities with both Khoesan and Bantu-speakers. The historical literature has also shed some light on the identity of these individuals. A number of European explorers and missionaries visited the region in the late 18th and early 19th centuries. Based on the accounts of Wikar (Mossop, 1935) and Gordon (Gordon et al., 1988), the Kakamas burials may be associated with an Einiqua community, the Anoe eis/Klaare Kraal people, who are described as Khoekhoen who had lost their cattle due to a dispute with a neighbouring community and were reliant on hunting and fishing for subsistence (see Morris (1992b) for a review of the historical accounts). By the time the European travellers Campbell and Thompson visited the area in the early 19th century, the extent and pattern of occupation along the Orange River had changed (Campbell, 1815; Thompson, 1827). Their descriptions suggest a general depopulation of the area, likely as a consequence of the emergence and expansion of groups of mixed ancestry, in this case the Korana, who had begun to assert an influence in the area.

It is not possible to state with certainty that this individual was associated with the Anoe eis/Klaare Kraal people described by Wikar and Gordon although this association is suspected (Morris, 1992b). Given that at least one of the Kakamas burials show a missionary influence, a later association with the Korana groups that had come to occupy the region by the early 19th century, as noted by Campbell and Thompson, cannot be ruled out. It is clear though from historical accounts that the late 18th century heralded a period of turbulence and displacement for the Khoekhoen of the middle Orange River (Penn, 1995). This unrest has been attributed to frequent raids and warfare with *boer* commandos and groups of mixed-descent who became variably known as Oorlams, Bastaards, Korana and Griqua (Penn, 1995). The expanding influence and increasing contact with the Colony undoubtedly provided opportunities for the transmission of disease and as Thompson noted by the early 19th century tuberculosis was well established among the residents of the Orange River. The evidence of tuberculosis is not as strong for NMB 1416 as some of the other cases identified in this study and consists only of proliferative rib lesions and the successful amplification of a single genetic marker for the *rpoB* gene. However, should this represent a case of tuberculosis, its presence along the river at this time supports accounts in the historical literature.

SAM-AP 1271 was unearthed in Henkries, Namaqualand. Henkries is located near the Orange River but a more detailed description of the location of this burial is not available. Morris (1992a) describes the burial position as "lying extended" which, as was noted above, is associated with a Christian influence. In 1799, the first missionaries ventured north of the Colony and settled among the San in the region of the Sak River (Penn, 1995). Two years later, in 1801, at the request of the "Bastaard" and Oorlam groups that now occupied the region, missionaries had reached and settled along the banks of the Orange River (Penn, 1995). This would likely, although not exclusively, place the date of interment for SAM-AP 1271 after 1801. The remains were "donated" in 1910; thus, a 19th century, possibly early 20th century interment seems most likely. To the mixed groups along the Orange River, Christianity, missionaries and mission stations promised, although did not always deliver, a number of social, economic and political benefits. These included political legitimacy, increased stability, access/rights to land and pasturage, legitimate/accepted participation in trade with the Colony and an elevation in colonial social status through education and the acceptance of Christianity (Beck, 1989; Kelso and Vogel, 2015; Penn, 1995). Both McVicar (1908) and Millar (1908) implicate missionaries and, more specifically, missionary schools in the spread of tuberculosis; however, these reports refer to the presence of disease among Bantu-speaking communities in the late-19th and early 20th centuries. McVicar (1908) attributes the association of tuberculosis with mission schools predominantly to overcrowding in boarding school dormitories and poorly ventilated school (often converted church) buildings. Among the Khoekhoen and the mixed groups of the Orange River, the presence of the disease may predate the more formal mission stations to which McVicar (1908) and Millar (1908) refer. In the late 18th and 19th centuries, the concentration of these communities around mission stations and a closer association with colonists may have contributed to the spread of tuberculosis, although the disease may have already been endemic among these populations at that time. The single vertebral lytic lesion present on the remains of SAM-AP 1271 provides moderate support for skeletal tuberculosis and given the historical accounts confirming a presence of the disease during this period and the broad time range of interment, the presence of tuberculosis is not unexpected.

The case for skeletal tuberculosis in NMB 1416 and SAM-AP 1271 is weak to moderate; however, the presence of disease along the Orange River during the late 18th and 19th centuries is not unexpected at this time and is supported by the historical literature.

MINING IN THE LATE 19TH CENTURY

Four individuals, MMK 331, GLD 38.8, GLD 8.2 and GLD 8.3, were all unearthed in Kimberley and are thought to be associated with the discovery of minerals and the establishment of mining operations in the region.

As was discussed in detail in chapter 2, the discovery of minerals played an important role in the spread of tuberculosis among the Bantu-speaking people of South Africa. The Kimberley diamond deposits were discovered in 1867 and the “diamond rush” that ensued attracted miners from all over southern Africa and the world. Tuberculosis at this time was already established at the Cape colony and among the Khoekhoen and mixed communities further north. Thus, both local and international miners may have served as a source of disease. As Packard (1989a) notes everything about the mining industry, from recruitment, to living and working conditions at the mines promoted the development and spread of tuberculosis among the African population. By the late 19th century the rates of tuberculosis among Africans began to cause alarm among the colonial settlers resulting in the first broad scale survey of the extent of the disease by McVicar (1908).

MMK 331 is one of only three females showing possible skeletal evidence of tuberculosis identified in this study and was unearthed from an African burial ground that was in use between 1870 and 1883 (Morris, 2004). This individual, unearthed from the Transvaal Road burial ground in Kimberley, was probably not directly involved in mining activities, which were largely the purview of men, but may have laboured in one of the many ancillary industries that supported the mines and their workers. The role of women, particularly black women, in the early mining town has not received much attention in the literature. This is partly due to the pronounced gender disparity among Africans, which was a noted feature of the early Kimberley diggings (Turrell, 1987). European men travelled to the diggings with their wives. African men were, however, mostly temporary migrants and were either young and unmarried or travelled without their wives (who remained behind to manage the homestead in the rural villages) (Turrell, 1987). The result was an overwhelming predominance of black male labourers. In these early years, the few black women that did make their way to Kimberley were Griqua, Korana or of mixed descent (Turrell, 1987). Skilled and semi-skilled black women worked as seamstresses, clothing pressers/ironers and bakers (Turrell, 1987). The growth of the town attracted Tswana, Griqua and Korana families who migrated to the area to supply produce to the growing market (Turrell, 1987). While the location of the burial in an African burial ground provides some indication as to the population affinity of this individual, a morphological estimation thereof is hampered by poor preservation. In the discussion regarding the role of the mining industry in the spread of tuberculosis among the African population of South Africa, much emphasis has been placed on the working and living conditions on the mines and in the mining compounds, as well as the health of new recruits. This case serves as a reminder of the broader impact of the disease particularly in the earlier years before the establishment of mining compounds.

The three males from Gladstone Cemetery were likely interred between 1897 and 1900 (van der Merwe et al., 2010b). These individuals are thought to have been mineworkers who died in Kimberley hospital and were buried as paupers. The rescue excavations of the Gladstone remains, which totalled 107 individuals from 15 graves, revealed neatly dug graves most of which lacked coffins and contained multiple individuals who, in some instances, were placed haphazardly within the shaft (van der Merwe et al., 2010b). This suggests that all deaths occurring on a particular day were buried within a single grave and that, in most cases, little care was taken to respect the dignity of those interred (van der Merwe et al., 2010b). van der Merwe et al. (2010b) analysis of the Gladstone remains revealed a high proportion of traumatic injury, as well as dietary and pathogenic disease which point to the dangerous and strenuous nature of the work at the mines, nutritional insufficiencies and a relatively high burden of pathogenic disease. Although the practice of repatriation of sick workers to their rural homes has been blamed for the spread of tuberculosis to the rural areas, to the miners, separated from their families, repatriation would have been far preferable to the anonymous death facing many of those that remained.

The most convincing evidence of tuberculosis identified in this study comes from GLD 38.8. This individual has osteolytic vertebral lesions, proliferative lesions of the ribs and long bones, and genetic support from all three MTBC loci investigated. GLD 8.3 also has a number of vertebral and extra-vertebral lytic lesions, as well as proliferative lesions on the lower limb long bones. MTBC DNA was only detected from the *rpoB*2 assay for this individual. GLD 8.2 shows proliferative lesions of the ribs, vertebrae and tibia. Attempts to amplify MTBC DNA from this individual were unsuccessful. Finally, MMK 331 presents with possible healed proliferative lesions of the ribs and MTBC DNA was successfully amplified from two loci. The evidence for tuberculosis is not equally compelling in all of these cases; however, it is clear that by the late 19th century it is possible to convincingly identify tuberculosis in the skeletal record. The three males from Gladstone share population affinity with Bantu-speakers. This, again, supports the historical literature which implicates the mining industry in the spread of tuberculosis among the Bantu-speaking populations of southern Africa (McVicar, 1908; Packard, 1989a; U.G., 1914). Seemingly coincident, the highest frequency (n = 4) of skeletal tuberculosis in this study emerges from the late 19th century in Kimberley. This may also support assertions that the mining industry played an instrumental role in the spread of tuberculosis among the black population but sample sizes in this study are so small that this correlation should be interpreted with caution.

RURAL AGRICULTURAL BASED COMMUNITIES IN THE 20TH CENTURY

The final two individuals identified in this study, NMB 1229 and MRB\20, both hail from communities that provided labour to surrounding farms.

NMB 1229 showed lytic destruction of the left sacroiliac joint and attempts to amplify MTBC DNA were unsuccessful. This individual was one of a number of remains exhumed by A. C. Hoffman in 1942 from an informal African burial ground on the farm, Kareeboom, in Wolmaransstad. The burials likely date to the early 20th century (Peckmann, 2002) and belonged to farm workers who laboured on farms in the area (Hoffman, 1942). Peckmann

(2002), in her analysis of the Wolmaransstad remains noted a predominance of males (30/39). Based on this observed male bias, she concluded that these remains likely belonged to migrant workers who, leaving their rural homes, sought to provide wage labour on farms. Peckmann (2002) also highlights the role of the 1890's hut tax and 1913 land act, which served to force Africans into the wage labour economy.

MRB\20 showed proliferative lesions of the ribs (Steyn et al., 2002) and is the only juvenile identified in this study. This individual was reburied prior to the onset of the project and was, therefore, not directly examined or included in the genetic analysis. MRB\20 was unearthed from an informal African cemetery at Maroelabult in the North West Province which is thought to have been in use between 1890 and 1950. Similar to the Wolmaransstad burials, this cemetery is believed to have serviced a community that provided labour to farms in the area. In contrast to the Wolmaransstad burials, however, the majority of these remains were subadult indicating a settled community, rather than a migrant one.

Although a diagnosis of tuberculosis is not secure in either of these cases, this does fit the timeline of a late 19th/early 20th century spread of tuberculosis to rural African communities. The mines, or more specifically, returning migrant mineworkers, have been viewed as instrumental in the dissemination of tuberculosis to the rural areas. This is largely due to the high rates of tuberculosis among mineworkers and the sheer scale of oscillating migrancy the industry produced (Packard, 1989a). However, returning mineworkers were not the only source of disease for rural Africans. Labour migrancy to the major towns, where tuberculosis had become established early in the country's colonial history, predates the development of commercial mining and the continued expansion of settlers and colonial influence presented increasing opportunities for transmission of the disease. A number of legislative acts, like the 1890s hut tax affecting Wolmaransstad and the 1913 Natives Land Act mentioned above, served to dispossess Africans of their land and independence, restrict their movement and force them into wage labour (Bundy, 1972). Cheap labour was sought not only on the mines but on white-owned farms invigorated by the economic growth spurred by the mineral revolution. It is unclear the extent of the disease among Africans working as labourers on white farms. Dormer and Wiles (1946) specifically note the lack of statistics available for this segment of the African population (estimated at be ~40% of the

African population at the time) but do describe the incidence of tuberculosis among Africans residing in the reserves as “low, but not negligible”. It is clear, however, that by the turn of the 19th century, rural Africans were becoming increasingly impoverished creating ideal conditions for the spread of tuberculosis.

CONCLUSION

This thesis has attempted to address the contributions of the historical literature, bioarchaeological record and ancient DNA to understanding the emergence and spread of tuberculosis in South Africa. To achieve this goal, the majority of skeletal remains housed in South African institutions were surveyed to identify possible cases of skeletal tuberculosis. In itself, the large, temporally and geographically aggregate dataset analysed in this study, affords a unique opportunity to examine patterns of preservation, demographic structure and pathology. The inferences gleaned from this broader dataset have been used to aid in the interpretation of identified cases of skeletal tuberculosis. Overall, the South African skeletal assemblage shows relatively poor preservation. An abundance of skulls is observed and is likely linked, in part, to past curatorial practices of preferentially collecting and retaining these elements. This may exacerbate the probable bias observed in the sex and age structure of the assemblage. In this regard, more males than females were observed, a frequently reported phenomenon partly attributed to bias in sex determination methods. Another frequently observed phenomenon, reported in this study and many others, is the underrepresentation of juveniles. While all but two individuals could be assigned to the binary age categories, mature or immature, a refinement of this assessment was not possible for the vast majority of skeletal remains. Where the assignment to one of three ordinal categories was possible, middle aged adults far outnumbered young and old adults. This is another pattern often observed in skeletal assemblages and has also been partly linked to biases inherent in the methodology. Most of the individuals in this assemblage cannot be readily associated with a chronological date. In contrast, location information is available for 80% of individuals, but the quality of this information varies considerably. Trauma and degenerative joint disease are the most frequently observed pathologies in this assemblage and skeletal evidence of infectious diseases is only infrequently observed.

Very few (n = 12) cases of tuberculosis were identified in this study (N = 2321), of which only two are truly convincing, based on combined molecular and skeletal evidence. This is a surprisingly low frequency for a country burdened with tuberculosis for at least 3½ centuries. Poor preservation may partially account for this low frequency. The vertebral column, the most frequent location of skeletal tuberculosis, while relatively well represented in this assemblage, was rarely complete or well preserved. Diagnostic confidence is so low partly because proliferative rib lesions were the sole pathology observed in five individuals and these, while consistent with pulmonary tuberculosis, are not generally considered diagnostic of the disease. A number of the identified tuberculosis cases displayed abnormal new bone formation on the long bones in addition to lesions elsewhere on the skeleton. These lesions have been interpreted as indicative of non-specific systemic infection and a number of additional individuals showing such lesion have been identified in the general assemblage. An investigation of such lesions, while not diagnostic of tuberculosis, may increase sample size for molecular investigations.

The detection of *Mycobacterium tuberculosis* complex (MTBC) markers was successful in five of the nine individuals sampled. Quantitative PCR screening for three MTBC markers, IS*6110*, IS*1081* and *rpoB*2, was used in this study. The amplification of *rpoB*2 in the absence of other markers suggests that, although designed as an MTBC-specific marker, this assay may result in non-specific amplification. Consequently, the molecular MTBC detection in two individuals, for which only *rpoB*2 was detected, should be accepted with caution. Of the remaining three individuals, two showed successful amplification of two assays, while all three assays were successful for the third individual. Target-enriched, next-generation sequencing was attempted for these three individuals but no MTBC-specific DNA was detected. This is the first study in South Africa to report the successful detection of ancient pathogen DNA; however, information regarding MTBC lineage and strain remains elusive.

All identified cases of skeletal tuberculosis are dated to, or have date ranges that overlap with, the colonial period. This may lend support to a colonial origin for tuberculosis in South Africa. One case may potentially predate colonization and/or physical European contact but the diagnosis is very tenuous in this case. No ancient DNA studies were attempted on this individual and this may be an interesting avenue for further research. Another interesting, although speculative, observation is that the earliest cases of tuberculosis occur in Iron Age populations or those in frequent contact with Iron Age populations. This is particularly relevant in light of the fact that the only historical reports to suggest a pre-colonial presence of tuberculosis were from Bantu-speaking populations along the east coast of South Africa. This may implicate East African trade in the early origins of tuberculosis in these populations. It should, however, be noted that these suppositions are highly speculative due to the tenuous diagnoses in both of these cases. The timing and geographical spread of skeletal cases confirm or conform to expectation in the literature, but is difficult to make concrete statements regarding patterns due to the low frequencies observed and low confidence in some of the diagnoses. These patterns include an early presence of tuberculosis at the major port city of Cape Town, followed by a spread to indigenous communities along the Orange River. A later focus is observed in Kimberley, associated with the development of the mining industry, and this is followed by a spread to rural agricultural communities.

This study has contributed to a local understanding of the antiquity of tuberculosis through a novel and systematic investigation of the bioarchaeological record. It has also contributed to a growing body of ancient DNA research in South Africa and is the first study to report the successful detection of ancient pathogen DNA. While studies have suggested that tuberculosis has an African origin, there is no concrete evidence to suggest that the disease was present as far south as South Africa. Further studies in other African countries will aid our understanding of the disease’s antiquity on the continent, and contribute a better understanding of its emergence and spread globally.

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APPENDIX A

Table A 1: Observed and fitted values, as well as, working, standardized and studentized residuals for the loglinear model with main effects for element and score and an interaction between the two variables.

Element	Score	Side	Observed	Fitted	Working	Standarized	Studentized
Frontal	1	L	1520	1513	0	0.25	0.25
Frontal	1	R	1506	1513	0	-0.25	-0.25
Parietal	1	L	1487	1473.5	0.01	0.5	0.5
Parietal	1	R	1460	1473.5	-0.01	-0.5	-0.5
Occipital	1	L	1223	1229.5	-0.01	-0.26	-0.26
Occipital	1	R	1236	1229.5	0.01	0.26	0.26
Temporal	1	L	1406	1403	0	0.11	0.11
Temporal	1	R	1400	1403	0	-0.11	-0.11
Sphenoid	1	L	1098	1090	0.01	0.34	0.34
Sphenoid	1	R	1082	1090	-0.01	-0.34	-0.34
Zygomatic	1	L	1280	1284	0	-0.16	-0.16
Zygomatic	1	R	1288	1284	0	0.16	0.16
Maxilla	1	L	1254	1253	0	0.04	0.04
Maxilla	1	R	1252	1253	0	-0.04	-0.04
Palatine	1	L	980	977	0	0.14	0.14
Palatine	1	R	974	977	0	-0.14	-0.14
Mandible	1	L	1204	1196	0.01	0.33	0.33
Mandible	1	R	1188	1196	-0.01	-0.33	-0.33
Clavicle	1	L	759	758	0	0.05	0.05
Clavicle	1	R	757	758	0	-0.05	-0.05
Scapula	1	L	522	518.5	0.01	0.22	0.22
Scapula	1	R	515	518.5	-0.01	-0.22	-0.22
Patella	1	L	429	429	0	0	0
Patella	1	R	429	429	0	0	0
Sacrum	1	L	519	526.5	-0.01	-0.46	-0.46
Sacrum	1	R	534	526.5	0.01	0.46	0.46
Os Coxa	1	L	616	614	0	0.11	0.11
Os Coxa	1	R	612	614	0	-0.11	-0.11
Humerus	1	L	713	700.5	0.02	0.67	0.67
Humerus	1	R	688	700.5	-0.02	-0.67	-0.67
Radius	1	L	611	609.5	0	0.09	0.09
Radius	1	R	608	609.5	0	-0.09	-0.09
Ulna	1	L	588	588	0	0	0
Ulna	1	R	588	588	0	0	0
Femur	1	L	795	783	0.02	0.6	0.61
Femur	1	R	771	783	-0.02	-0.61	-0.61

Element	Score	Side	Observed	Fitted	Working	Standarized	Studentized
Tibia	1	L	744	737	0.01	0.36	0.36
Tibia	1	R	730	737	-0.01	-0.37	-0.36
Fibula	1	L	541	525.5	0.03	0.95	0.95
Fibula	1	R	510	525.5	-0.03	-0.96	-0.96
Ribs	1	L	397	391	0.02	0.43	0.43
Ribs	1	R	385	391	-0.02	-0.43	-0.43
Carpals	1	L	225	238.5	-0.06	-1.25	-1.24
Carpals	1	R	252	238.5	0.06	1.22	1.23
Metacarpals	1	L	406	423.5	-0.04	-1.21	-1.21
Metacarpals	1	R	441	423.5	0.04	1.19	1.2
Hand Phalanges	1	L	14	12.5	0.12	0.59	0.59
Hand Phalanges	1	R	11	12.5	-0.12	-0.61	-0.61
Tarsals	1	L	344	362.5	-0.05	-1.39	-1.38
Tarsals	1	R	381	362.5	0.05	1.36	1.37
Metatarsals	1	L	402	416	-0.03	-0.98	-0.97
Metatarsals	1	R	430	416	0.03	0.97	0.97
Foot Phalanges	1	L	3	4	-0.25	-0.74	-0.72
Foot Phalanges	1	R	5	4	0.25	0.68	0.69
Frontal	2	L	184	194	-0.05	-1.02	-1.02
Frontal	2	R	204	194	0.05	1.01	1.01
Parietal	2	L	226	237.5	-0.05	-1.06	-1.06
Parietal	2	R	249	237.5	0.05	1.05	1.05
Occipital	2	L	389	385	0.01	0.29	0.29
Occipital	2	R	381	385	-0.01	-0.29	-0.29
Temporal	2	L	188	188	0	0	0
Temporal	2	R	188	188	0	0	0
Sphenoid	2	L	205	214	-0.04	-0.88	-0.87
Sphenoid	2	R	223	214	0.04	0.86	0.87
Zygomatic	2	L	73	77.5	-0.06	-0.73	-0.73
Zygomatic	2	R	82	77.5	0.06	0.72	0.72
Maxilla	2	L	192	189	0.02	0.31	0.31
Maxilla	2	R	186	189	-0.02	-0.31	-0.31
Palatine	2	L	79	76.5	0.03	0.4	0.4
Palatine	2	R	74	76.5	-0.03	-0.41	-0.41
Mandible	2	L	163	173	-0.06	-1.09	-1.08
Mandible	2	R	183	173	0.06	1.07	1.07
Clavicle	2	L	120	110.5	0.09	1.26	1.27
Clavicle	2	R	101	110.5	-0.09	-1.3	-1.29
Scapula	2	L	257	265	-0.03	-0.7	-0.7
Scapula	2	R	273	265	0.03	0.69	0.69
Patella	2	L	25	24.5	0.02	0.14	0.14
Patella	2	R	24	24.5	-0.02	-0.14	-0.14
Sacrum	2	L	228	221	0.03	0.66	0.66
Sacrum	2	R	214	221	-0.03	-0.67	-0.67
Os Coxa	2	L	267	274.5	-0.03	-0.64	-0.64

Element	Score	Side	Observed	Fitted	Working	Standarized	Studentized
Os Coxa	2	R	282	274.5	0.03	0.64	0.64
Humerus	2	L	285	285.5	0	-0.04	-0.04
Humerus	2	R	286	285.5	0	0.04	0.04
Radius	2	L	289	285.5	0.01	0.29	0.29
Radius	2	R	282	285.5	-0.01	-0.29	-0.29
Ulna	2	L	339	328.5	0.03	0.81	0.82
Ulna	2	R	318	328.5	-0.03	-0.82	-0.82
Femur	2	L	260	255.5	0.02	0.4	0.4
Femur	2	R	251	255.5	-0.02	-0.4	-0.4
Tibia	2	L	232	228.5	0.02	0.33	0.33
Tibia	2	R	225	228.5	-0.02	-0.33	-0.33
Fibula	2	L	275	278	-0.01	-0.25	-0.25
Fibula	2	R	281	278	0.01	0.25	0.25
Ribs	2	L	286	295	-0.03	-0.74	-0.74
Ribs	2	R	304	295	0.03	0.74	0.74
Carpals	2	L	154	150.5	0.02	0.4	0.4
Carpals	2	R	147	150.5	-0.02	-0.41	-0.4
Metacarpals	2	L	176	171	0.03	0.54	0.54
Metacarpals	2	R	166	171	-0.03	-0.54	-0.54
Hand Phalanges	2	L	1	3	-0.67	-1.9	-1.77
Hand Phalanges	2	R	5	3	0.67	1.49	1.56
Tarsals	2	L	303	288.5	0.05	1.2	1.2
Tarsals	2	R	274	288.5	-0.05	-1.22	-1.21
Metatarsals	2	L	148	145	0.02	0.35	0.35
Metatarsals	2	R	142	145	-0.02	-0.35	-0.35
Foot Phalanges	2	L	9	11	-0.18	-0.88	-0.87
Foot Phalanges	2	R	13	11	0.18	0.83	0.84
Frontal	3	L	81	82.5	-0.02	-0.23	-0.23
Frontal	3	R	84	82.5	0.02	0.23	0.23
Parietal	3	L	53	59	-0.1	-1.12	-1.11
Parietal	3	R	65	59	0.1	1.09	1.1
Occipital	3	L	134	130.5	0.03	0.43	0.43
Occipital	3	R	127	130.5	-0.03	-0.44	-0.43
Temporal	3	L	36	36.5	-0.01	-0.12	-0.12
Temporal	3	R	37	36.5	0.01	0.12	0.12
Sphenoid	3	L	129	124.5	0.04	0.57	0.57
Sphenoid	3	R	120	124.5	-0.04	-0.57	-0.57
Zygomatic	3	L	22	19.5	0.13	0.78	0.79
Zygomatic	3	R	17	19.5	-0.13	-0.82	-0.81
Maxilla	3	L	88	86.5	0.02	0.23	0.23
Maxilla	3	R	85	86.5	-0.02	-0.23	-0.23
Palatine	3	L	82	92	-0.11	-1.5	-1.49
Palatine	3	R	102	92	0.11	1.45	1.46
Mandible	3	L	48	55	-0.13	-1.36	-1.35
Mandible	3	R	62	55	0.13	1.31	1.32

Element	Score	Side	Observed	Fitted	Working	Standarized	Studentized
Clavicle	3	L	22	21.5	0.02	0.15	0.15
Clavicle	3	R	21	21.5	-0.02	-0.15	-0.15
Scapula	3	L	115	109	0.06	0.81	0.81
Scapula	3	R	103	109	-0.06	-0.82	-0.82
Patella	3	L	1	0.5	1	0.88	0.94
Patella	3	R	0	0.5	-1	-1.41	-1.22
Sacrum	3	L	115	117.5	-0.02	-0.33	-0.33
Sacrum	3	R	120	117.5	0.02	0.33	0.33
Os Coxa	3	L	84	80	0.05	0.63	0.63
Os Coxa	3	R	76	80	-0.05	-0.64	-0.64
Humerus	3	L	29	32	-0.09	-0.76	-0.76
Humerus	3	R	35	32	0.09	0.74	0.74
Radius	3	L	19	21.5	-0.12	-0.78	-0.77
Radius	3	R	24	21.5	0.12	0.75	0.76
Ulna	3	L	24	23.5	0.02	0.15	0.15
Ulna	3	R	23	23.5	-0.02	-0.15	-0.15
Femur	3	L	32	29	0.1	0.77	0.78
Femur	3	R	26	29	-0.1	-0.8	-0.79
Tibia	3	L	26	29	-0.1	-0.8	-0.79
Tibia	3	R	32	29	0.1	0.77	0.78
Fibula	3	L	14	16.5	-0.15	-0.89	-0.88
Fibula	3	R	19	16.5	0.15	0.85	0.86
Ribs	3	L	186	192.5	-0.03	-0.67	-0.66
Ribs	3	R	199	192.5	0.03	0.66	0.66
Carpals	3	L	131	138	-0.05	-0.85	-0.85
Carpals	3	R	145	138	0.05	0.84	0.84
Metacarpals	3	L	66	72.5	-0.09	-1.1	-1.09
Metacarpals	3	R	79	72.5	0.09	1.06	1.07
Hand Phalanges	3	L	3	3.5	-0.14	-0.39	-0.38
Hand Phalanges	3	R	4	3.5	0.14	0.37	0.37
Tarsals	3	L	143	138	0.04	0.6	0.6
Tarsals	3	R	133	138	-0.04	-0.61	-0.6
Metatarsals	3	L	72	66.5	0.08	0.94	0.95
Metatarsals	3	R	61	66.5	-0.08	-0.97	-0.96
Foot Phalanges	3	L	15	14	0.07	0.37	0.38
Foot Phalanges	3	R	13	14	-0.07	-0.38	-0.38

Table A 2: Element and preservation score (EP) log-linear model parameters and associated standard errors, z-values and p-values.

	Estimate	exp(Estimate)*	Std. Error	z value	Pr(>|z|)
(Intercept)	7.32		0.0182	402.77	0
Element-Parietal	-0.03		0.0259	-1.02	0.3067
Element-Occipital	-0.21		0.0272	-7.64	0
Element-Temporal	-0.08		0.0262	-2.88	0.004
Element-Maxilla	-0.19		0.027	-6.98	0
Element-Zygomatic	-0.16		0.0268	-6.12	0
Element-Sphenoid	-0.33		0.0281	-11.67	0
Element-Mandible	-0.24		0.0274	-8.59	0
Element-Palatine	-0.44		0.029	-15.07	0
Element-Femur	-0.66		0.0311	-21.16	0
Element-Humerus	-0.77		0.0323	-23.83	0
Element-Tibia	-0.72		0.0318	-22.64	0
Element-Ulna	-0.95		0.0344	-27.5	0
Element-Os Coxa	-0.9		0.0338	-26.65	0
Element-Radius	-0.91		0.0339	-26.8	0
Element-Clavicle	-0.69		0.0315	-21.97	0
Element-Scapula	-1.07		0.036	-29.76	0
Element-Ribs	-1.35		0.0401	-33.73	0
Element-Fibula	-1.06		0.0358	-29.54	0
Element-Sacrum	-1.06		0.0358	-29.5	0
Element-Metacarpals	-1.27		0.0389	-32.76	0
Element-Tarsals	-1.43		0.0413	-34.55	0
Element-Hand Phalanges	-4.8		0.2008	-23.88	0
Element-Metatarsals	-1.29		0.0391	-32.98	0
Element-Foot Phalanges	-5.94		0.354	-16.77	0
Element-Carpals	-1.85		0.0493	-37.5	0
Element-Patella	-1.26		0.0387	-32.59	0
Score-2	-2.05		0.0539	-38.09	0
Score-3	-2.91		0.0799	-36.39	0
Element-Parietal:Score-2	0.23	1.26	0.0732	3.13	0.0018
Element-Occipital:Score-2	0.89	2.44	0.0679	13.15	0
Element-Temporal:Score-2	0.04	1.04	0.077	0.57	0.567
Element-Maxilla:Score-2	0.16	1.17	0.0772	2.11	0.0352
Element-Zygomatic:Score-2	-0.75	0.47	0.0987	-7.63	0
Element-Sphenoid:Score-2	0.43	1.54	0.0755	5.64	0
Element-Mandible:Score-2	0.12	1.13	0.0788	1.53	0.1263
Element-Palatine:Score-2	-0.49	0.61	0.0998	-4.94	0
Element-Femur:Score-2	0.93	2.53	0.0742	12.59	0
Element-Humerus:Score-2	1.16	3.19	0.0733	15.78	0

	Estimate	exp(Estimate)*	Std. Error	z value	Pr(>|z|)
Element-Tibia:Score-2	0.88	2.41	0.076	11.62	0
Element-Ulna:Score-2	1.47	4.35	0.0727	20.25	0
Element-Os Coxa:Score-2	1.25	3.49	0.0745	16.77	0
Element-Radius:Score-2	1.3	3.67	0.074	17.5	0
Element-Clavicle:Score-2	0.13	1.14	0.09	1.43	0.1537
Element-Scapula:Score-2	1.38	3.97	0.0759	18.22	0
Element-Ribs:Score-2	1.77	5.87	0.0767	23.11	0
Element-Fibula:Score-2	1.42	4.14	0.0752	18.84	0
Element-Sacrum:Score-2	1.19	3.29	0.0782	15.16	0
Element-Metacarpals:Score-2	1.15	3.16	0.0837	13.7	0
Element-Tarsals:Score-2	1.83	6.23	0.0776	23.53	0
Element-Hand Phalanges:Score-2	0.63	1.88	0.4578	1.37	0.1709
Element-Metatarsals:Score-2	1	2.72	0.0869	11.5	0
Element-Foot Phalanges:Score-2	3.07	21.54	0.4164	7.36	0
Element-Carpals:Score-2	1.59	4.9	0.0912	17.46	0
Element-Patella:Score-2	-0.81	0.44	0.1565	-5.17	0
Element-Parietal:Score-3	-0.31	0.73	0.1233	-2.5	0.0123
Element-Occipital:Score-3	0.67	1.95	0.1031	6.46	0
Element-Temporal:Score-3	-0.74	0.48	0.143	-5.18	0
Element-Maxilla:Score-3	0.24	1.27	0.1121	2.1	0.0354
Element-Zygomatic:Score-3	-1.28	0.28	0.1801	-7.1	0
Element-Sphenoid:Score-3	0.74	2.1	0.1042	7.09	0
Element-Mandible:Score-3	-0.17	0.84	0.1261	-1.35	0.1767
Element-Palatine:Score-3	0.55	1.73	0.1111	4.92	0
Element-Femur:Score-3	-0.39	0.68	0.1558	-2.48	0.013
Element-Humerus:Score-3	-0.18	0.84	0.1508	-1.17	0.2404
Element-Tibia:Score-3	-0.33	0.72	0.1559	-2.09	0.0364
Element-Ulna:Score-3	-0.31	0.73	0.1689	-1.84	0.0658
Element-Os Coxa:Score-3	0.87	2.39	0.116	7.51	0
Element-Radius:Score-3	-0.44	0.64	0.1745	-2.5	0.0126
Element-Clavicle:Score-3	-0.65	0.52	0.1741	-3.75	2.00E-04
Element-Scapula:Score-3	1.35	3.86	0.1093	12.35	0
Element-Ribs:Score-3	2.2	9.03	0.1013	21.72	0
Element-Fibula:Score-3	-0.55	0.58	0.194	-2.84	0.0044
Element-Sacrum:Score-3	1.41	4.1	0.1077	13.09	0
Element-Metacarpals:Score-3	1.14	3.13	0.1203	9.51	0
Element-Tarsals:Score-3	1.94	6.96	0.1067	18.21	0
Element-Hand Phalanges:Score-3	1.64	5.16	0.435	3.76	2.00E-04
Element-Metatarsals:Score-3	1.08	2.94	0.1229	8.75	0
Element-Foot Phalanges:Score-3	4.16	64.07	0.4088	10.18	0
Element-Carpals:Score-3	2.36	10.59	0.11	21.46	0
Element-Patella:Score-3	-3.85	0.02	1.0038	-3.83	1.00E-04

* Only 2-way assocations were exponentiated as these reflect the odds ratios.
Reference levels: Element – Frontal and Score – 1

Table A 3: Subdivided element frequencies (relative frequencies %) stratified by element, side and preservation score (n=2307).

			Score			
Element	Segment	Side	1	2	3	Total
Temporal	Complete	L	1406 (60.94)	188 (8.15)	36 (1.56)	1630 (70.65)
		R	1400 (60.68)	188 (8.15)	37 (1.6)	1625 (70.44)
	TMJ	L	1460 (63.29)	21 (0.91)	8 (0.35)	1489 (64.54)
		R	1458 (63.2)	20 (0.87)	7 (0.3)	1485 (64.37)
Scapula	Body	L	522 (22.63)	257 (11.14)	115 (4.98)	894 (38.75)
		R	515 (22.32)	273 (11.83)	103 (4.46)	891 (38.62)
	Glenoid fossa	L	687 (29.78)	89 (3.86)	30 (1.3)	806 (34.94)
		R	720 (31.21)	73 (3.16)	15 (0.65)	808 (35.02)
Sternum	Body	U	352 (15.26)	141 (6.11)	62 (2.69)	555 (24.06)
	Manubrium	U	459 (19.9)	73 (3.16)	25 (1.08)	557 (24.14)
	Xyphoid	U	80 (3.47)	5 (0.22)	0 (0)	85 (3.68)
Ribs	1st	L	542 (23.49)	100 (4.33)	5 (0.22)	647 (28.05)
		R	553 (23.97)	93 (4.03)	9 (0.39)	655 (28.39)
	2nd	L	399 (17.3)	205 (8.89)	15 (0.65)	619 (26.83)
		R	402 (17.43)	214 (9.28)	18 (0.78)	634 (27.48)
	11th	L	388 (16.82)	131 (5.68)	14 (0.61)	533 (23.1)
		R	396 (17.17)	105 (4.55)	8 (0.35)	509 (22.06)
	12th	L	358 (15.52)	85 (3.68)	5 (0.22)	448 (19.42)
		R	365 (15.82)	66 (2.86)	1 (0.04)	432 (18.73)
	3rd-10th	L	367 (15.91)	323 (14)	135 (5.85)	825 (35.76)
		R	365 (15.82)	328 (14.22)	149 (6.46)	842 (36.5)
		U	NA	NA	27 (1.17)	27 (1.17)
Os coxa	Acetabulum	L	510 (22.11)	153 (6.63)	68 (2.95)	731 (31.69)
		R	529 (22.93)	144 (6.24)	56 (2.43)	729 (31.6)
	Auricular surface	L	626 (27.13)	142 (6.16)	88 (3.81)	856 (37.1)
		R	677 (29.35)	113 (4.9)	71 (3.08)	861 (37.32)
	Ilium	L	709 (30.73)	174 (7.54)	45 (1.95)	928 (40.23)
		R	724 (31.38)	173 (7.5)	42 (1.82)	939 (40.7)
	Ischium	L	660 (28.61)	143 (6.2)	36 (1.56)	839 (36.37)
		R	683 (29.61)	122 (5.29)	32 (1.39)	837 (36.28)
	Pubis	L	526 (22.8)	137 (5.94)	66 (2.86)	729 (31.6)
		R	522 (22.63)	141 (6.11)	59 (2.56)	722 (31.3)
Vertebrae						
C1	Body	U	629 (27.26)	21 (0.91)	1 (0.04)	651 (28.22)
	NA	U	674 (29.22)	118 (5.11)	6 (0.26)	798 (34.59)
C2	Body	U	696 (30.17)	41 (1.78)	2 (0.09)	739 (32.03)
	NA	U	690 (29.91)	64 (2.77)	12 (0.52)	766 (33.2)
C7	Body	U	593 (25.7)	21 (0.91)	1 (0.04)	615 (26.66)
	NA	U	594 (25.75)	27 (1.17)	5 (0.22)	626 (27.13)
C3-6	Body	U	575 (24.92)	108 (4.68)	107 (4.64)	790 (34.24)
	NA	U	552 (23.93)	119 (5.16)	121 (5.24)	792 (34.33)

Element	Segment	Side	Score 1	2	3	Total
C3-7	Body	U	21 (0.91)	15 (0.65)	13 (0.56)	49 (2.12)
	NA	U	26 (1.13)	24 (1.04)	15 (0.65)	65 (2.82)
T10	Body	U	537 (23.28)	60 (2.6)	6 (0.26)	603 (26.14)
	NA	U	595 (25.79)	17 (0.74)	1 (0.04)	613 (26.57)
T11	Body	U	559 (24.23)	54 (2.34)	6 (0.26)	619 (26.83)
	NA	U	611 (26.48)	16 (0.69)	4 (0.17)	631 (27.35)
T12	Body	U	575 (24.92)	53 (2.3)	9 (0.39)	637 (27.61)
	NA	U	620 (26.87)	25 (1.08)	4 (0.17)	649 (28.13)
T1-9	Body	U	496 (21.5)	182 (7.89)	111 (4.81)	789 (34.2)
	NA	U	549 (23.8)	154 (6.68)	116 (5.03)	819 (35.5)
T1-12	Body	U	37 (1.6)	27 (1.17)	35 (1.52)	99 (4.29)
	NA	U	49 (2.12)	21 (0.91)	30 (1.3)	100 (4.33)
L1	Body	U	543 (23.54)	62 (2.69)	9 (0.39)	614 (26.61)
	NA	U	592 (25.66)	22 (0.95)	8 (0.35)	622 (26.96)
L2	Body	U	561 (24.32)	57 (2.47)	4 (0.17)	622 (26.96)
	NA	U	606 (26.27)	23 (1)	5 (0.22)	634 (27.48)
L3	Body	U	560 (24.27)	59 (2.56)	9 (0.39)	628 (27.22)
	NA	U	609 (26.4)	19 (0.82)	4 (0.17)	632 (27.39)
L4	Body	U	554 (24.01)	53 (2.3)	14 (0.61)	621 (26.92)
	NA	U	596 (25.83)	20 (0.87)	4 (0.17)	620 (26.87)
L5	Body	U	553 (23.97)	65 (2.82)	9 (0.39)	627 (27.18)
	NA	U	602 (26.09)	25 (1.08)	7 (0.3)	634 (27.48)
L1-5	Body	U	46 (1.99)	30 (1.3)	15 (0.65)	91 (3.94)
	NA	U	59 (2.56)	33 (1.43)	11 (0.48)	103 (4.46)

Table A 4: Log-linear model fit statistics for sub-divided elements of the scapula, ribs and os coxa. The variables included in the models are element/sub-element (E), preservation score (P) and side/segment (S). The best fitting/selected model is shown in bold font.

Element	Model	G^2	*df*	*p*-value	H_0	ΔG^2	*Δdf*	*Δp*-value	AIC	BIC
Scapula	EPS	0	0	1.00000	NA	NA	NA	NA	107.12	112.94
	EP,PS,ES	5.5	2	0.06393	λ_{ijk}^{EPS}=0	5.5	2	0.063928	108.62	113.47
	EP,PS	8.64	4	0.07076	λ_{ik}^{ES} =0	3.14	2	0.208045	107.76	111.64
	EP,S	8.64	5	0.12431	λ_{jk}^{PS} =0	0	1	1.000000	105.77	109.16
	E,P,S	386.31	7	0.00000	λ_{ij}^{EP} =0	377.67	2	0.000000	479.43	481.86
	EP	**8.65**	**6**	**0.19424**	**λ_k^S =0**	**0.01**	**1**	**0.920344**	**103.77**	**106.68**
Ribs	EPS	0	0	1.00000	NA	NA	NA	NA	250.78	292.82
	EP,PS,ES	9.85	8	0.27571	λ_{ijk}^{EPS}=0	9.85	8	0.275712	244.63	275.46
	EP,PS	11.47	10	0.32209	λ_{ik}^{ES} =0	1.62	2	0.444858	242.26	270.28
	EP,S	12.72	14	0.54868	λ_{jk}^{PS} =0	1.25	4	0.869800	235.5	257.92
	E,P,S	919.9	22	0.00000	λ_{ij}^{EP} =0	907.18	8	0.000000	1126.68	1137.89
	EP	**12.72**	**15**	**0.62392**	**λ_k^S =0**	**0**	**1**	**1.000000**	**233.5**	**254.52**
	EP	11.99	12	0.44680	No group	0.73	3	0.865111	182.45	196.59
Os coxa	EPS	0	0	1.00000	NA	NA	NA	NA	160.61	176.64
	EP,PS,ES	1.58	4	0.81238	λ_{ijk}^{EPS}=0	1.58	4	0.812382	154.19	166.65
	EP,PS	2.92	6	0.81882	λ_{ik}^{ES} =0	1.34	2	0.511709	151.53	162.22
	EP,S	3.02	8	0.93310	λ_{jk}^{PS} =0	0.1	2	0.951229	147.64	156.54
	E,P,S	46.59	12	0.00001	λ_{ij}^{EP} =0	43.57	4	0.000000	183.2	188.54
	EP	**3.02**	**9**	**0.96350**	**λ_k^S =0**	**0**	**1**	**1.000000**	**145.64**	**153.65**
Vertebrae	**EPS**	**0**	**0**	**1.00000**	**NA**	**NA**	**NA**	**NA**	**745.2**	**991.37**
	EPS	0	0	0.00000	Score (1,2+3)	0	0	1.000000	559.89	698.06
	EP,PS,ES	236.31	30	0.00000	λ_{ijk}^{EPS}=0	236.31	30	0.000000	921.5	1090.75
	EP,PS	271.02	32	0.00000	λ_{ik}^{ES} =0	34.71	2	0.000000	952.22	1116.34
	EP,S	285.27	47	0.00000	λ_{jk}^{PS} =0	14.25	15	0.506659	936.46	1062.12
	E,P,S	2377.89	77	0.00000	λ_{ij}^{EP} =0	2092.62	30	0.000000	2969.08	3017.81
	EP	290.64	48	0.00000	λ_k^S =0	5.37	1	0.020486	939.83	1062.92

Table A 5: Log-linear (EP) model observed fit statistics: observed values, fitted values and residuals for the scapula, ribs and os coxa.

						Residuals		
Element	Score	Segment	Side	Observed	Fitted	Working	Standardized	Studentized
Scapula	1	Body	L	522	518.5	0.007	0.217	0.217
			R	515	518.5	-0.007	-0.218	-0.217
		Glenoid fossa	L	687	703.5	-0.023	-0.883	-0.882
			R	720	703.5	0.023	0.876	0.878
	2	Body	L	257	265	-0.03	-0.699	-0.697
			R	273	265	0.03	0.692	0.693
		Glenoid fossa	L	89	81	0.099	1.237	1.247
			R	73	81	-0.099	-1.279	-1.268
	3	Body	L	115	109	0.055	0.805	0.809
			R	103	109	-0.055	-0.82	-0.817
		Glenoid fossa	L	30	22.5	0.333	2.126	2.182
			R	15	22.5	-0.333	-2.382	-2.31
Ribs	1	1st	L	542	547.5	-0.01	-0.333	-0.333
			R	553	547.5	0.01	0.332	0.332
		2nd	L	399	400.5	-0.004	-0.106	-0.106
			R	402	400.5	0.004	0.106	0.106
		11th	L	388	392	-0.01	-0.286	-0.286
			R	396	392	0.01	0.285	0.285
		12th	L	358	361.5	-0.01	-0.261	-0.261
			R	365	361.5	0.01	0.26	0.26
		3rd-10th	L	367	366	0.003	0.074	0.074
			R	365	366	-0.003	-0.074	-0.074
	2	1st	L	100	96.5	0.036	0.501	0.502
			R	93	96.5	-0.036	-0.507	-0.505
		2nd	L	205	209.5	-0.021	-0.441	-0.44
			R	214	209.5	0.021	0.438	0.439
		11th	L	131	118	0.11	1.663	1.678
			R	105	118	-0.11	-1.725	-1.709
		12th	L	85	75.5	0.126	1.515	1.531
			R	66	75.5	-0.126	-1.58	-1.563
		3rd-10th	L	323	325.5	-0.008	-0.196	-0.196
			R	328	325.5	0.008	0.196	0.196
	3	1st	L	5	7	-0.286	-1.127	-1.099
			R	9	7	0.286	1.023	1.046
		2nd	L	15	16.5	-0.091	-0.53	-0.526
			R	18	16.5	0.091	0.515	0.518
		11th	L	14	11	0.273	1.227	1.253
			R	8	11	-0.273	-1.345	-1.313
		12th	L	5	3	0.667	1.489	1.563
			R	1	3	-0.667	-1.899	-1.771

						Residuals		
Element	Score	Segment	Side	Observed	Fitted	Working	Standardized	Studentized
		3rd-10th	L	135	142	-0.049	-0.838	-0.834
			R	149	142	0.049	0.824	0.827
Os coxa	1	Ilium	L	709	716.5	-0.01	-0.397	-0.397
			R	724	716.5	0.01	0.396	0.396
		Ischium	L	660	671.5	-0.017	-0.629	-0.629
			R	683	671.5	0.017	0.626	0.627
		Pubis	L	526	524	0.004	0.123	0.124
			R	522	524	-0.004	-0.124	-0.124
	2	Ilium	L	174	173.5	0.003	0.054	0.054
			R	173	173.5	-0.003	-0.054	-0.054
		Ischium	L	143	132.5	0.079	1.274	1.282
			R	122	132.5	-0.079	-1.308	-1.299
		Pubis	L	137	139	-0.014	-0.24	-0.24
			R	141	139	0.014	0.239	0.24
	3	Ilium	L	45	43.5	0.034	0.32	0.321
			R	42	43.5	-0.034	-0.324	-0.323
		Ischium	L	36	34	0.059	0.48	0.483
			R	32	34	-0.059	-0.49	-0.488
		Pubis	L	66	62.5	0.056	0.62	0.623
			R	59	62.5	-0.056	-0.632	-0.629

Table A 6: Loglinear model (EP) parameter estimates, standard error, Wald z-statistic and p-values for the SCAPULA, RIBS and OS COXA. Reference baseline score = 2. Reference baseline Element/Segment: Scapula = body, ribs = 1^{st} , os coxa = pubis.

Element	Main effects/Interactions	Estimate	Exp(Est)*	*SE*	*z*-value	*p*-value
Scapula	Segment-Glenoid fossa	-1.19	0.31	0.09	-13.2	**<0.0001**
	Score-1	0.67	1.96	0.053	12.57	**<0.0001**
	Score-3	-0.89	0.41	0.08	-11.04	**<0.0001**
	Segment-Glenoid fossa:Score-1	1.49	4.44	0.099	15.11	**<0.0001**
	Segment-Glenoid fossa:Score-3	-0.39	0.68	0.187	-2.1	**0.03553**
Ribs	Element-2nd	0.78	2.17	0.087	8.91	**<0.0001**
	Element-11th	0.2	1.22	0.097	2.07	**0.03821**
	Element-12th	-0.25	0.78	0.109	-2.26	**0.02389**
	Element-3rd-10th	1.22	3.37	0.082	14.83	**<0.0001**
	Score-1	1.74	5.67	0.078	22.23	**<0.0001**
	Score-3	-2.62	0.07	0.277	-9.48	**<0.0001**
	Element-2nd:Score-1	-1.09	0.34	0.099	-11.03	**<0.0001**
	Element-11th:Score-1	-0.54	0.59	0.108	-4.97	**<0.0001**
	Element-12th:Score-1	-0.17	0.84	0.119	-1.43	0.15299
	Element-3rd-10th:Score-1	-1.62	0.2	0.095	-17.06	**<0.0001**
	Element-2nd:Score-3	0.08	1.09	0.331	0.25	0.80348
	Element-11th:Score-3	0.25	1.29	0.355	0.71	0.4803
	Element-12th:Score-3	-0.6	0.55	0.5	-1.2	0.22858
	Element-3rd-10th:Score-3	1.79	6.01	0.286	6.28	**<0.0001**
os coxa	Segment-Ischium	-0.05	0.95	0.086	-0.56	0.57696
	Segment-Ilium	0.22	1.25	0.08	2.75	**0.00588**
	Score-1	1.33	3.77	0.067	19.67	**<0.0001**
	Score-3	-0.8	0.45	0.108	-7.42	**<0.0001**
	Segment-Ischium:Score-1	0.3	1.34	0.095	3.11	**0.00189**
	Segment-Ilium:Score-1	0.09	1.1	0.09	1.01	0.31192
	Segment-Ischium:Score-3	-0.56	0.57	0.173	-3.23	**0.00122**
	Segment-Ilium:Score-3	-0.58	0.56	0.161	-3.62	**0.00029**

* Exponentiated Estimates: For 2-way interactions these are the odds ratios.
Significant main effects and interactions are shown in bold font.

Table A 7: Loglinear model (EPS) parameter estimates, standard error, Wald z-statistic and p-values for the VERTEBRAE. Reference baselines: score = 2, element = L5, segment = body.

Main effects/Interactions	Estimate	Exp(Estimate)	*SE*	*z*-value	*p*-value
Element-C1	-1.13	0.32	0.251	-4.5	**<0.0001**
Element-C2	-0.46	0.63	0.199	-2.31	**0.02086**
Element-C7	-1.13	0.32	0.251	-4.5	**<0.0001**
Element-C3-6	0.51	1.66	0.157	3.23	**0.00122**
Element-C3-7	-1.47	0.23	0.286	-5.12	**<0.0001**
Element-T10	-0.08	0.92	0.179	-0.45	0.65481
Element-T11	-0.19	0.83	0.184	-1.01	0.31397
Element-T12	-0.2	0.82	0.185	-1.1	0.27013
Element-T1-9	1.03	2.8	0.144	7.13	**<0.0001**
Element-T1-12	-0.88	0.42	0.229	-3.84	**0.00012**
Element-L1	-0.05	0.95	0.178	-0.27	0.7901
Element-L2	-0.13	0.88	0.181	-0.72	0.46921
Element-L3	-0.1	0.91	0.18	-0.54	0.59016
Element-L4	-0.2	0.82	0.185	-1.1	0.27013
Element-L1-5	-0.77	0.46	0.221	-3.5	**0.00046**
Score-1	2.14	8.51	0.131	16.33	**<0.0001**
Score-3	-1.98	0.14	0.356	-5.56	**<0.0001**
Segment-NA	-0.96	0.38	0.235	-4.06	**0.00005**
Element-C1:Score-1	1.26	3.52	0.258	4.88	**<0.0001**
Element-C2:Score-1	0.69	2	0.207	3.33	**0.00087**
Element-C7:Score-1	1.2	3.32	0.258	4.65	**<0.0001**
Element-C3-6:Score-1	-0.47	0.63	0.168	-2.79	**0.00524**
Element-C3-7:Score-1	-1.8	0.16	0.363	-4.98	**<0.0001**
Element-T10:Score-1	0.05	1.05	0.189	0.27	0.78858
Element-T11:Score-1	0.2	1.22	0.194	1.01	0.31099
Element-T12:Score-1	0.24	1.28	0.194	1.25	0.21115
Element-T1-9:Score-1	-1.14	0.32	0.157	-7.24	**<0.0001**
Element-T1-12:Score-1	-1.83	0.16	0.285	-6.41	**<0.0001**
Element-L1:Score-1	0.03	1.03	0.188	0.15	0.87708
Element-L2:Score-1	0.15	1.16	0.191	0.76	0.44581
Element-L3:Score-1	0.11	1.12	0.19	0.58	0.56372
Element-L4:Score-1	0.21	1.23	0.195	1.06	0.29
Element-L1-5:Score-1	-1.71	0.18	0.269	-6.37	**<0.0001**
Element-C1:Score-3	-1.07	0.34	1.084	-0.99	0.3246
Element-C2:Score-3	-1.04	0.35	0.807	-1.29	0.19597
Element-C7:Score-3	-1.07	0.34	1.084	-0.99	0.3246
Element-C3-6:Score-3	1.97	7.16	0.381	5.17	**<0.0001**
Element-C3-7:Score-3	1.83	6.26	0.52	3.53	**0.00042**
Element-T10:Score-3	-0.33	0.72	0.557	-0.58	0.55879
Element-T11:Score-3	-0.22	0.8	0.558	-0.39	0.69345
Element-T12:Score-3	0.2	1.23	0.506	0.4	0.68694

Main effects/Interactions	Estimate	Exp(Estimate)	*SE*	*z*-value	*p*-value
Element-T1-9:Score-3	1.48	4.4	0.375	3.95	**<0.0001**
Element-T1-12:Score-3	2.24	9.36	0.438	5.1	**<0.0001**
Element-L1:Score-3	0.05	1.05	0.504	0.09	0.92526
Element-L2:Score-3	-0.68	0.51	0.628	-1.08	0.27897
Element-L3:Score-3	0.1	1.1	0.505	0.19	0.84777
Element-L4:Score-3	0.65	1.91	0.466	1.39	0.16536
Element-L1-5:Score-3	1.28	3.61	0.476	2.7	**0.00698**
Element-C1:Segment-NA	2.68	14.61	0.334	8.03	**<0.0001**
Element-C2:Segment-NA	1.4	4.06	0.309	4.54	**<0.0001**
Element-C7:Segment-NA	1.21	3.34	0.374	3.22	**0.00126**
Element-C3-6:Segment-NA	1.05	2.86	0.27	3.89	**0.0001**
Element-C3-7:Segment-NA	1.43	4.16	0.405	3.52	**0.00043**
Element-T10:Segment-NA	-0.31	0.74	0.362	-0.84	0.39822
Element-T11:Segment-NA	-0.26	0.77	0.369	-0.71	0.47996
Element-T12:Segment-NA	0.2	1.23	0.338	0.6	0.54597
Element-T1-9:Segment-NA	0.79	2.2	0.26	3.04	**0.00238**
Element-T1-12:Segment-NA	0.7	2.02	0.374	1.88	0.05987
Element-L1:Segment-NA	-0.08	0.92	0.342	-0.24	0.81373
Element-L2:Segment-NA	0.05	1.05	0.341	0.14	0.88822
Element-L3:Segment-NA	-0.18	0.84	0.354	-0.5	0.61541
Element-L4:Segment-NA	-0.02	0.98	0.352	-0.05	0.9569
Element-L1-5:Segment-NA	1.05	2.86	0.345	3.05	**0.00232**
Score-1:Segment-NA	1.04	2.83	0.243	4.29	**0.00002**
Score-3:Segment-NA	0.7	2.02	0.556	1.27	0.20548
Element-C1:Score-1:Segment-NA	-2.7	0.07	0.344	-7.85	**<0.0001**
Element-C2:Score-1:Segment-NA	-1.49	0.22	0.319	-4.68	**<0.0001**
Element-C7:Score-1:Segment-NA	-1.29	0.28	0.383	-3.37	**0.00076**
Element-C3-6:Score-1:Segment-NA	-1.18	0.31	0.283	-4.16	**0.00003**
Element-C3-7:Score-1:Segment-NA	-1.3	0.27	0.503	-2.58	**0.00997**
Element-T10:Score-1:Segment-NA	0.32	1.38	0.371	0.87	0.38397
Element-T11:Score-1:Segment-NA	0.26	1.3	0.379	0.7	0.48401
Element-T12:Score-1:Segment-NA	-0.21	0.81	0.348	-0.61	0.53922
Element-T1-9:Score-1:Segment-NA	-0.77	0.46	0.273	-2.82	**0.00474**
Element-T1-12:Score-1:Segment-NA	-0.51	0.6	0.437	-1.16	0.24483
Element-L1:Score-1:Segment-NA	0.08	1.09	0.352	0.23	0.81567
Element-L2:Score-1:Segment-NA	-0.06	0.95	0.351	-0.16	0.87398
Element-L3:Score-1:Segment-NA	0.18	1.19	0.363	0.49	0.62679
Element-L4:Score-1:Segment-NA	0.01	1.01	0.362	0.02	0.98409
Element-L1-5:Score-1:Segment-NA	-0.89	0.41	0.401	-2.21	**0.02718**
Element-C1:Score-3:Segment-NA	-0.64	0.53	1.238	-0.52	0.60591
Element-C2:Score-3:Segment-NA	0.64	1.9	0.966	0.67	0.50604
Element-C7:Score-3:Segment-NA	0.65	1.92	1.263	0.52	0.6045
Element-C3-6:Score--3:Segment-NA	-0.68	0.51	0.587	-1.16	0.24796
Element-C3-7:Score--3:Segment-NA	-1.03	0.36	0.749	-1.38	0.16873

Main effects/Interactions	Estimate	Exp(Estimate)	*SE*	*z*-value	*p*-value
Element-T10:Score-3:Segment-NA	-1.23	0.29	1.246	-0.99	0.32151
Element-T11:Score-3:Segment-NA	0.11	1.11	0.898	0.12	0.90543
Element-T12:Score-3:Segment-NA	-0.76	0.47	0.854	-0.89	0.37118
Element-T1-9:Score-3:Segment-NA	-0.49	0.61	0.582	-0.85	0.39704
Element-T1-12:Score-3:Segment-NA	-0.61	0.54	0.675	-0.9	0.36864
Element-L1:Score-3:Segment-NA	0.21	1.24	0.779	0.27	0.78346
Element-L2:Score-3:Segment-NA	0.43	1.53	0.906	0.47	0.63772
Element-L3:Score-3:Segment-NA	-0.38	0.68	0.86	-0.44	0.65698
Element-L4:Score-3:Segment-NA	-0.98	0.37	0.836	-1.17	0.2402
Element-L1-5:Score-3:Segment-NA	-1.11	0.33	0.728	-1.52	0.12765

* Exponentiated parameter estimate
Significant main effects and interactions are shown in bold font.

Table A 8: Subdivided element frequencies (relative frequencies %) for the humerus, radius, ulna, femur, tibia and fibula stratified by element, side and preservation score (n=2307).

			Score			
Element	Segment	Side	1	2	3	Total
Humerus	PE	L	554 (24.01)	137 (5.94)	16 (0.69)	707 (30.65)
		R	551 (23.88)	121 (5.24)	19 (0.82)	691 (29.95)
	P⅓	L	921 (39.92)	52 (2.25)	10 (0.43)	983 (42.61)
		R	887 (38.45)	54 (2.34)	12 (0.52)	953 (41.31)
	M⅓	L	951 (41.22)	30 (1.3)	6 (0.26)	987 (42.78)
		R	925 (40.1)	28 (1.21)	9 (0.39)	962 (41.7)
	D⅓	L	947 (41.05)	34 (1.47)	11 (0.48)	992 (43)
		R	933 (40.44)	35 (1.52)	11 (0.48)	979 (42.44)
	DE	L	618 (26.79)	93 (4.03)	18 (0.78)	729 (31.6)
		R	590 (25.57)	103 (4.46)	13 (0.56)	706 (30.6)
Radius	PE	L	506 (21.93)	107 (4.64)	9 (0.39)	622 (26.96)
		R	521 (22.58)	93 (4.03)	8 (0.35)	622 (26.96)
	P⅓	L	836 (36.24)	41 (1.78)	11 (0.48)	888 (38.49)
		R	826 (35.8)	46 (1.99)	6 (0.26)	878 (38.06)
	M⅓	L	857 (37.15)	32 (1.39)	3 (0.13)	892 (38.66)
		R	839 (36.37)	31 (1.34)	6 (0.26)	876 (37.97)
	D⅓	L	815 (35.33)	59 (2.56)	14 (0.61)	888 (38.49)
		R	783 (33.94)	73 (3.16)	15 (0.65)	871 (37.75)
	DE	L	582 (25.23)	47 (2.04)	10 (0.43)	639 (27.7)
		R	586 (25.4)	42 (1.82)	13 (0.56)	641 (27.79)
Ulna	PE	L	606 (26.27)	81 (3.51)	9 (0.39)	696 (30.17)
		R	601 (26.05)	66 (2.86)	11 (0.48)	678 (29.39)
	P⅓	L	875 (37.93)	43 (1.86)	7 (0.3)	925 (40.1)
		R	859 (37.23)	39 (1.69)	12 (0.52)	910 (39.45)
	M⅓	L	865 (37.49)	37 (1.6)	12 (0.52)	914 (39.62)
		R	850 (36.84)	29 (1.26)	13 (0.56)	892 (38.66)
	D⅓	L	817 (35.41)	55 (2.38)	16 (0.69)	888 (38.49)
		R	806 (34.94)	49 (2.12)	14 (0.61)	869 (37.67)
	DE	L	476 (20.63)	40 (1.73)	9 (0.39)	525 (22.76)
		R	490 (21.24)	36 (1.56)	8 (0.35)	534 (23.15)
Femur	PE	L	664 (28.78)	154 (6.68)	17 (0.74)	835 (36.19)
		R	671 (29.09)	117 (5.07)	11 (0.48)	799 (34.63)
	P⅓	L	968 (41.96)	63 (2.73)	14 (0.61)	1045 (45.3)
		R	957 (41.48)	46 (1.99)	11 (0.48)	1014 (43.95)
	M⅓	L	988 (42.83)	41 (1.78)	9 (0.39)	1038 (44.99)
		R	954 (41.35)	36 (1.56)	10 (0.43)	1000 (43.35)
	D⅓	L	939 (40.7)	82 (3.55)	16 (0.69)	1037 (44.95)
		R	912 (39.53)	66 (2.86)	19 (0.82)	997 (43.22)
	DE	L	669 (29)	152 (6.59)	21 (0.91)	842 (36.5)
		R	631 (27.35)	162 (7.02)	20 (0.87)	813 (35.24)

Element	Segment	Side	Score 1	Score 2	Score 3	Total
Tibia	PE	L	669 (29)	106 (4.59)	10 (0.43)	785 (34.03)
		R	672 (29.13)	80 (3.47)	13 (0.56)	765 (33.16)
	P⅓	L	867 (37.58)	76 (3.29)	24 (1.04)	967 (41.92)
		R	860 (37.28)	57 (2.47)	23 (1)	940 (40.75)
	M⅓	L	924 (40.05)	31 (1.34)	12 (0.52)	967 (41.92)
		R	912 (39.53)	27 (1.17)	14 (0.61)	953 (41.31)
	D⅓	L	894 (38.75)	58 (2.51)	15 (0.65)	967 (41.92)
		R	877 (38.01)	54 (2.34)	18 (0.78)	949 (41.14)
	DE	L	684 (29.65)	69 (2.99)	9 (0.39)	762 (33.03)
		R	678 (29.39)	59 (2.56)	3 (0.13)	740 (32.08)
Fibula	PE	L	377 (16.34)	64 (2.77)	8 (0.35)	449 (19.46)
		R	348 (15.08)	66 (2.86)	9 (0.39)	423 (18.34)
	P⅓	L	747 (32.38)	48 (2.08)	9 (0.39)	804 (34.85)
		R	717 (31.08)	44 (1.91)	4 (0.17)	765 (33.16)
	M⅓	L	778 (33.72)	25 (1.08)	3 (0.13)	806 (34.94)
		R	753 (32.64)	19 (0.82)	5 (0.22)	777 (33.68)
	D⅓	L	747 (32.38)	50 (2.17)	10 (0.43)	807 (34.98)
		R	725 (31.43)	48 (2.08)	15 (0.65)	788 (34.16)
	DE	L	554 (24.01)	33 (1.43)	5 (0.22)	592 (25.66)
		R	530 (22.97)	40 (1.73)	4 (0.17)	574 (24.88)

Table A 9: Log-linear model fit statistics for sub-divided elements of the humerus, radius, ulna, femur, tibia and fibula. The variables included in the models are element/sub-element (E), preservation score (P) and side/segment (S). The best fitting/selected model is shown in bold font.

Element	Model	G^2	*df*	*p*-value	H_0	ΔG^2	Δdf	Δp-value	AIC	BIC
Humerus	EPS	0	0	1.00000	NA	NA	NA	NA	247.13	289.17
	EP,PS,ES	3.61	8	0.89049	$\lambda_{ijk}^{EPS}=0$	3.61	8	0.890487	234.74	265.57
	EP,PS	3.82	10	0.95510	$\lambda_{ik}^{ES}=0$	0.21	2	0.900325	230.95	258.97
	EP,S	3.92	14	0.99593	$\lambda_{jk}^{PS}=0$	0.1	4	0.998791	223.05	245.47
	E,P,S	417.11	22	0.00000	$\lambda_{ij}^{EP}=0$	413.19	8	0.000000	620.24	631.45
	EP	**5.24**	**15**	**0.98989**	**$\lambda_{k}^{S}=0$**	**1.32**	**1**	**0.250592**	**222.37**	**243.39**
Radius	EPS	0	0	1.00000	NA	NA	NA	NA	242.08	284.12
	EP,PS,ES	6.78	8	0.56054	$\lambda_{ijk}^{EPS}=0$	6.78	8	0.560545	232.87	263.69
	EP,PS	6.81	10	0.74325	$\lambda_{ik}^{ES}=0$	0.03	2	0.985112	228.89	256.92
	EP,S	6.96	14	0.93624	$\lambda_{jk}^{PS}=0$	0.15	4	0.997324	221.05	243.46
	E,P,S	194.33	22	0.00000	$\lambda_{ij}^{EP}=0$	187.37	8	0.000000	392.41	403.62
	EP	**7.18**	**15**	**0.95246**	**$\lambda_{k}^{S}=0$**	**0.22**	**1**	**0.639040**	**219.26**	**240.28**
Ulna	EPS	0	0	1.00000	NA	NA	NA	NA	242.86	284.9
	EP,PS,ES	1.94	8	0.98280	$\lambda_{ijk}^{EPS}=0$	1.94	8	0.982796	228.8	259.62
	EP,PS	4.69	10	0.91090	$\lambda_{ik}^{ES}=0$	2.75	2	0.252840	227.55	255.57
	EP,S	5.06	14	0.98496	$\lambda_{jk}^{PS}=0$	0.37	4	0.984859	219.92	242.34
	E,P,S	84.15	22	0.00000	$\lambda_{ij}^{EP}=0$	79.09	8	0.000000	283.01	294.22
	EP	**5.6**	**15**	**0.98571**	**$\lambda_{k}^{S}=0$**	**0.54**	**1**	**0.462433**	**218.46**	**239.48**
Femur	EPS	0	0	1.00000	NA	NA	NA	NA	252.7	294.74
	EP,PS,ES	8.06	8	0.42763	$\lambda_{ijk}^{EPS}=0$	8.06	8	0.427631	244.76	275.59
	EP,PS	11.03	10	0.35519	$\lambda_{ik}^{ES}=0$	2.97	2	0.226502	243.74	271.76
	EP,S	11.08	14	0.67974	$\lambda_{jk}^{PS}=0$	0.05	4	0.999693	235.78	258.2
	E,P,S	411.55	22	0.00000	$\lambda_{ij}^{EP}=0$	400.47	8	0.000000	620.25	631.46
	EP	**14.3**	**15**	**0.50291**	**$\lambda_{k}^{S}=0$**	**3.22**	**1**	**0.072744**	**237**	**258.02**
Tibia	EPS	0	0	1.00000	NA	NA	NA	NA	247.84	289.88

Element	Model	G^2	*df*	*p*-value	H_0	ΔG^2	*Δdf*	*Δp*-value	AIC	BIC
	EP,PS,ES	5.17	8	0.73926	$\lambda_{ijk}^{EPS}=0$	5.17	8	0.739261	237.02	267.85
	EP,PS	10.62	10	0.38788	$\lambda_{ik}^{ES}=0$	5.45	2	0.065546	238.47	266.49
	EP,S	10.69	14	0.71020	$\lambda_{jk}^{PS}=0$	0.07	4	0.999402	230.54	252.96
	E,P,S	144.4	22	0.00000	$\lambda_{ij}^{EP}=0$	133.71	8	0.000000	348.25	359.45
	EP	**11.85**	**15**	**0.69035**	$\boldsymbol{\lambda_{k}^{S}=0}$	**1.16**	**1**	**0.281466**	**229.7**	**250.72**
Fibula	EPS	0	0	1.00000	NA	NA	NA	NA	235.23	277.27
	EP,PS,ES	5.39	8	0.71519	$\lambda_{ijk}^{EPS}=0$	5.39	8	0.715194	224.62	255.45
	EP,PS	5.66	10	0.84297	$\lambda_{ik}^{ES}=0$	0.27	2	0.873716	220.89	248.91
	EP,S	5.91	14	0.96871	$\lambda_{jk}^{PS}=0$	0.25	4	0.992809	213.14	235.56
	E,P,S	147.36	22	0.00000	$\lambda_{ij}^{EP}=0$	141.45	8	0.000000	338.6	349.81
	EP	**8.44**	**15**	**0.90493**	$\boldsymbol{\lambda_{k}^{S}=0}$	**2.53**	**1**	**0.111700**	**213.67**	**234.69**

Table A 10: Relative frequencies of individuals stratified by sex.

Sex*	Frequency (Relative Frequency %)
F	269 (15.11)
[F]	297 (16.69)
A	251 (14.1)
[M]	370 (20.79)
M	358 (20.11)
ND	235 (13.2)

*F = female, [F] = probably female, A = ambiguous, [M] = probably male, M = male, ND = not determined.

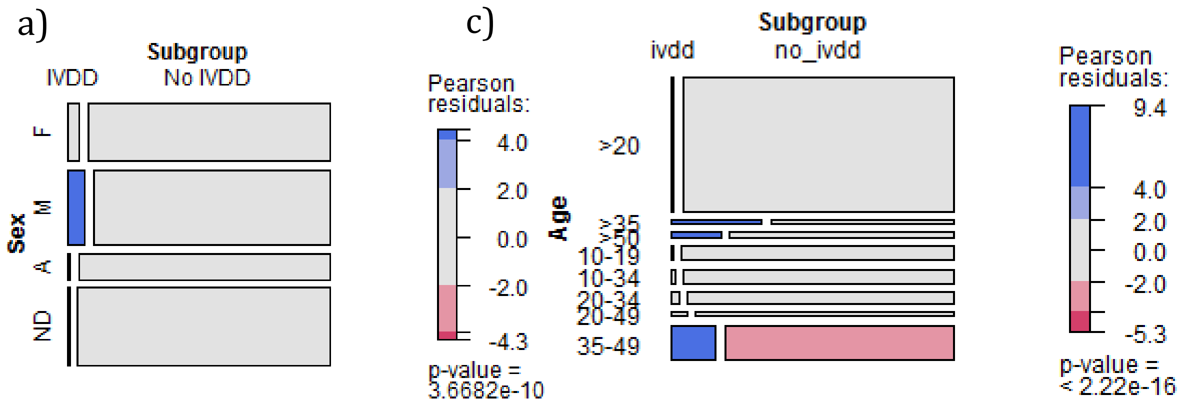
Table A 11: Individuals with multiple fractures

Accession No.	Element	Side	Sub Element	Fracture Type
A 4141	vertebra, lumbar, 4th	bilateral	neural arch	spondylolysis
	vertebra, lumbar, 5th	bilateral	neural arch	spondylolysis
A 4277	vertebra, lumbar, 4th	bilateral	neural arch	spondylolysis
	vertebra, lumbar, 5th	bilateral	neural arch	spondylolysis
ALB 216	vertebra, lumbar, 5th	bilateral	neural arch	spondylolysis
	ulna	R	D1/3	oblique/spiral
ALB 307	metacarpal, 2nd	L	M1/3	oblique/spiral
	metacarpal, 5th	L	P1/3	not determined
MMK 330	ulna	L	M1/3	not determined
	vertebra, Thoracic, 10th	midline	body	compression
	vertebra, thoracic, 12th	midline	body	compression
MMK GLD 31 E1	vertebra, lumbar, 4th	bilateral	neural arch	spondylolysis
	nasal	bilateral	NA	not determined
	zygomatic	L	NA	not determined
	frontal	R	NA	depression
MMK GLD 31 E4	hand phalanx, proximal, 5th	not determined	DE	not determined
	metacarpal, 2nd	R	M1/3	spiral
MMK GLD 34.3	hand phalanx, intermediate, 4th	not determined	M1/3	not determined
	rib, 10th	L	NA	not determined
	frontal	L	NA	depression
MMK GLD 38.9	metatarsal, 4th	R	not determined	not determined
	metatarsal, 5th	R	not determined	oblique/spiral
MMK GLD 74.1	rib, 7th	L	NA	not determined
	rib, 11th	L	NA	not determined
MMK GLD N 100.2	rib, 4th	R	NA	not determined
	clavicle	L	NA	spiral

Accession No.	Element	Side	Sub Element	Fracture Type
MMK GLD NOP 3/4.1	vertebra, lumbar, 2nd	L	transverse process	not determined
	vertebra, lumbar, 5th	bilateral	neural arch	spondylolysis
	frontal	L	NA	depression
MMK GLD S 5.1	nasal	bilateral	NA	not determined
	maxilla	L	frontal process	not determined
MMK GLD SE 11.1	capitate	R	NA	not determined
	metacarpal, 5th	R	DE	physeal
NMB 1202	frontal	L	NA	depression
	parietal	L	NA	depression
	parietal	L	NA	depression
NMB 1327 A and B	radius	L	D1/3	oblique/spiral
	ulna	L	D1/3	oblique/spiral
NMB 1416 A and B	vertebra, lumbar, 5th	bilateral	neural arch	spondylolysis
	ulna	R	D1/3	spiral
NMB 1621 A and B	vertebra, thoracic, [3rd]	midline	body	compression
	vertebra, lumbar, 5th	midline	body	compression
NMB 1640	vertebra, lumbar, 5th	bilateral	neural arch	spondylolysis
	metatarsal, 5th	L	M1/3	oblique/spiral
NMB 1658	radius	L	D1/3	not determined
	fibula	L	DE	not determined
NMB 5	frontal, sphenoid	R, unilateral	NA	penetrating
	frontal, parietal	L, unilateral	NA	penetrating
NMB SS3 / 17M2/96	ribs, [6th-9th?]	L	NA	oblique
	radius	L	D1/3	not determined
	vertebra, thoracic, 12th	midline	body	compression
	vertebra, lumbar, 1st	midline	body	compression
SAM-AP 1146	vertebra, thoracic, 12th	midline	body	compression

Accession No.	Element	Side	Sub Element	Fracture Type
	os coxa	L	ischiopubic ramus	oblique/spiral
SAM-AP 1247 A	ulna	L	D1/3	not determined
	hand phalanx, proximal, 3rd	not determined	not determined	not determined
SAM-AP 1457	metacarpal, 1st	R	not determined	not determined
	metacarpal, 5th	R	P1/3	oblique/spiral
	ulna	L	D1/3	not determined
SAM-AP 1878 A	rib, 4th?	L	NA	not determined
	tibia	L	not determined	transverse
SAM-AP 4322	frontal	R	NA	depression
	metacarpal, 4th	R	not determined	not determined
	metacarpal, 5th	R	not determined	not determined
SAM-AP 4508	vertebra, lumbar, 3rd	bilateral	neural arch	spondylolysis
	vertebra, lumbar, 5th	bilateral	neural arch	spondylolysis
SAM-AP 4701	hand phalanx, proximal, 4th	not determined	not determined	not determined
	fibula	R	not determined	not determined
SAM-AP 4708	metatarsal, 5th	R	not determined	not determined
	femur	L	not determined	not determined
	ulna	L	not determined	not determined
	radius	L	not determined	not determined
SAM-AP 4831	humerus	L	P1/3	not determined
	ulna	L	D1/3	not determined
SAM-AP 4840	vertebra, lumbar, 1st	L	neural arch	transverse split
	vertebra, lumbar, 2nd	L	neural arch	transverse
SAM-AP 5037	rib, 3rd	R	NA	transverse
	tibia	R	M1/3	spiral
SAM-AP 6150	radius	L	D1/3	not determined
	ulna	L	D1/3	not determined

Accession No.	Element	Side	Sub Element	Fracture Type
UP 12	metacarpal, 4th	L	P1/3	not determined
	ulna	L	D1/3	spiral
	vertebra, lumbar, 1st	midline	body	compression
UP 29	metacarpal, 4th	R	M1/3	oblique/spiral
	rib	L	NA	not determined
	rib	R	NA	not determined



b) **Odds Ratio (95% CI)**

	Subgroup
Sex	IVDD:No IVDD
F:M	0.68 (0.42-1.09)
M:A	6.43 (1.99-20.77)
A:ND	1.49 (0.37-6.01)

d) **Odds Ratio (95% CI)**

	Subgroup
Age	IVDD:N IVDD
>20:20-34	0.42 (0.12-1.49)
>35:20-34	16 (3.7-69.18)
>50:20-34	7.27 (1.91-27.74)
10-19:20-34	0.28 (0.03-2.72)
10-34:20-34	0.52 (0.08-3.15)
20-34:20-49	0.52 (0.08-3.22)
20-34:35-49	0.16 (0.05-0.52)

Figure A 1: Mosaic plots and odds ratios for intervertebral disc disease (IVDD) and non-IVDD subgroups. a) Mosaic plot with shaded Pearson residuals comparing sex and data subgroups. b) Odds ratios and 95% confidence intervals comparing sex and data subgroups. c) Mosaic plot with shaded Pearson residuals comparing age-at-death and data subgroups. d) Odds ratios and 95% confidence intervals comparing age-at-death and data subgroups. Confidence intervals that do not contain the null are shown in bold. Sex: F = grouped female and probably female, A = ambiguous, M =grouped male and probably male, ND = not determined.

APPENDIX B

Table B 1: List of accessions and summary of preservation, sex, age-at-death, chronological age and location (n = 2321) for all data presented in this thesis.

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
ALB 2	ALB	3	[M]	mature	>20				Port Elizabeth
ALB 9	ALB	3	M	mature	>20				
ALB 11	ALB	3	ND	immature	6-19				Bethlehem
ALB 13	ALB	3	[F]	mature	>20				
ALB 18	ALB	3	ND	immature	6-19				Port Alfred
ALB 25	ALB	3	A	mature	>20				
ALB 26	ALB	3	[M]	mature	>20				Waterkloof
ALB 28	ALB	3	A	mature	>20				
ALB 29	ALB	3	[M]	mature	>20				
ALB 30	ALB	3	[F]	mature	>20				
ALB 31	ALB	3	[M]	mature	>20				
ALB 33	ALB	3	A	mature	>20				Adelaide
ALB 36	ALB	3	F	mature	>20				
ALB 37	ALB	3	A	mature	>20				Grahamstown
ALB 38	ALB	3	[F]	mature	>20				
ALB 39	ALB	3	[F]	mature	>20				
ALB 40	ALB	3	A	mature	>20				
ALB 42	ALB	3	A	mature	>20				
ALB 49 A	ALB	1	[M]	immature	10-34				Steinkopf
ALB 50	ALB	1	[F]	mature	35-49		2380 ± 45 (Pta-8557)	(Pfeiffer and Sealy, 2006)	Plettenberg Bay
ALB 51	ALB	1	ND	immature	6-19				Plettenberg Bay
ALB 52	ALB	3	[M]	mature	>20				Steinkopf
ALB 53	ALB	3	M	mature	>20				Steinkopf
ALB 54	ALB	1	[M]	mature	>20				Port Alfred

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
ALB 56	ALB	3	A	mature	>20				Port Alfred
ALB 61	ALB	1	[M]	mature	35-49				Port Alfred
ALB 75	ALB	3	F	mature	>20				OlifantsKloof
ALB 76	ALB	3	A	mature	>20				
ALB 78	ALB	3	[F]	mature	>20				Port Elizabeth
ALB 100	ALB	3	A	mature	>20				Grahamstown
ALB 108	ALB	2	ND	immature	2-5		570 ± 60 (TO-11651)	(Silberbauer, 1979)	Port Alfred
ALB 112	ALB	3	A	mature	>20				Riet River Mouth
ALB 114	ALB	3	ND	mature	>20				Port Alfred
ALB 116 [D]	ALB	3	ND	mature	>20				Alicedale
ALB 116 A	ALB	3	ND	immature	2-5				Alicedale
ALB 116 B	ALB	2	ND	immature	birth-5				Alicedale
ALB 118	ALB	2	M	mature	>20				Graaff-Reinet
ALB 119 B	ALB	3	ND	immature	birth-5				Alicedale
ALB 119	ALB	2	ND	immature	10-19		8260 ± 720 (Gak-1541)	(Silberbauer, 1979)	Alicedale
ALB 120	ALB	2	ND	immature	birth-1		4630 ± 70 (TO-10365)	(Silberbauer, 1979)	Alicedale
ALB 121	ALB	2	ND	mature	>20		4680 ± 60 (Pta-8566)	(Silberbauer, 1979)	Alicedale
ALB 122	ALB	2	ND	immature	2-5		4721 ± 31 (OxA-V-2092-2)	(Silberbauer, 1979)	Alicedale
ALB 124	ALB	2	[M]	mature	35-49				Alicedale
ALB 126	ALB	1	M	immature	20-34		540 ± 50 (Pta-8677)	(Silberbauer, 1979)	Port Alfred
ALB 127	ALB	3	ND	mature	>20				
ALB 128 A	ALB	2	F	mature	>50				Spitzkop
ALB 128 B	ALB	2	ND	mature	>20				Spitzkop
ALB 128 C	ALB	3	ND	mature	>20				Spitzkop

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
ALB 128 E	ALB	2	ND	immature	10-19				Spitzkop
ALB 129	ALB	3	[M]	mature	>20				Spitzkop
ALB 131	ALB	1	M	mature	20-49		4700 ± 60 (Pta-5979)	(Silberbauer, 1979)	Spitzkop
ALB 132	ALB	1	ND	immature	6-9		4720 ± 70 (TO-10235)	(Silberbauer, 1979)	Spitzkop
ALB 133	ALB	1	ND	immature	2-5		4860 ± 70 (TO-10366)	(Silberbauer, 1979)	Spitzkop
ALB 135	ALB	2	ND	immature	birth-1		4900 ± 60 (TO-10367)	(Silberbauer, 1979)	Spitzkop
ALB 136	ALB	1	F	mature	>50		4930 ± 70 (Pta-8620)	(Silberbauer, 1979)	Spitzkop
ALB 137	ALB	2	ND	immature	2-5		4750 ± 60 (TO-10236)	(Silberbauer, 1979)	Spitzkop
ALB 138	ALB	2	ND	immature	2-5		4800 ± 80 (TO-10237)	(Silberbauer, 1979)	Spitzkop
ALB 139	ALB	2	F	immature	10-34		5100 ± 70 (Pta-8626)	(Silberbauer, 1979)	Spitzkop
ALB 140	ALB	3	[M]	mature	>20				Rasfonteinpoort
ALB 143	ALB	3	[F]	mature	>20				Sea Field
ALB 148	ALB	3	A	mature	>20				Lichtenburg
ALB 149	ALB	3	A	mature	>20				Jeffrey's Bay
ALB 150	ALB	1	[M]	mature	35-49		1910 ± 60 (TO-10368)	(Silberbauer, 1979)	Kabeljous River
ALB 151 A	ALB	2	ND	immature	10-19		2920 ± 45 (Pta-8570)	(Stynder et al., 2007a)	Kabeljous River
ALB 151 B	ALB	3	ND	immature	2-19				Kabeljous River
ALB 152	ALB	2	ND	immature	10-19		2990 ± 60 (TO-10369)	(Silberbauer, 1979)	Kabeljous River
ALB 153	ALB	3	ND	immature	birth-19				Kabeljous River
ALB 162	ALB	3	[F]	mature	>20				Port Alfred
ALB 163	ALB	3	A	mature	>20				Port Alfred
ALB 165	ALB	1	ND	immature	10-19				Port Alfred

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
ALB 166	ALB	1	[M]	mature	35-49				Port Alfred
ALB 167	ALB	2	A	immature	10-34				Port Alfred
ALB 168	ALB	3	[M]	mature	>20				Port Alfred
ALB 171	ALB	3	[F]	mature	>20				Drivers Bush Farm
ALB 173	ALB	1	[M]	mature	>50				Hole-in-the-Wall
ALB 174	ALB	2	A	immature	10-34		430 ± 50 (Pta-8574)	(Silberbauer, 1979)	Glen Boyd
ALB 175	ALB	1	ND	immature	birth-9		360 ± 50 (TO-10370)	(Silberbauer, 1979)	Glen Boyd
ALB 176	ALB	1	ND	immature	2-5		390 ± 50 (TO-10371)	(Silberbauer, 1979)	
ALB 177	ALB	1	[F]	mature	35-49		390 ± 40 (Pta-8584)	(Silberbauer, 1979)	Glen Boyd
ALB 178	ALB	1	F	mature	35-49		240 ± 45 (Pta-8599)	(Silberbauer, 1979)	Glen Boyd
ALB 179	ALB	1	A	mature	20-34		140 ± 35 (Pta-8563)	(Silberbauer, 1979)	
ALB 180	ALB	1	F	mature	35-49				Uitenhage
ALB 181	ALB	1	ND	immature	2-9		450 ± 50 (TO-10372)	(Silberbauer, 1979)	Glen Boyd
ALB 182	ALB	1	ND	immature	6-19		390 ± 50 (Pta-8939)	(Silberbauer, 1979)	Glen Boyd
ALB 183	ALB	2	ND	immature	2-9		220 ± 50 (TO-10373)	(Silberbauer, 1979)	Dunbrody
ALB 184	ALB	2	[M]	mature	>20		320 ± 50 (TO-10374)	(Silberbauer, 1979)	Dunbrody
ALB 186	ALB	2	[M]	mature	>20		365 ± 20 (Pta-8730)	(Silberbauer, 1979)	
ALB 187	ALB	2	[F]	immature	10-34		380 ± 50 (Pta-8683)	(Silberbauer, 1979)	
ALB 191 [A]	ALB	1	F	mature	35-49				Hole-in-the-Wall
ALB 191 [B]	ALB	3	ND	mature	>20				Hole-in-the-Wall

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
ALB 192	ALB	3	A	mature	>20				Port Alfred
ALB 193	ALB	1	ND	immature	2-5		230 ± 60 (TO-10238)	(Silberbauer, 1979)	Glen Boyd
ALB 195 [A]	ALB	2	[F]	immature	10-34		2870 ± 90 (Pta-706)	(Silberbauer, 1979)	Melkhoutboom
ALB 195 B	ALB	2	ND	immature	prenatal-1				Melkhoutboom
ALB 196	ALB	3	A	mature	>20				
ALB 197	ALB	2	ND	immature	birth-5				Glen Boyd
ALB 198	ALB	1	ND	immature	10-19		5120 ± 70 (Pta-8618)	(Silberbauer, 1979)	Vygeboom; Zuurberg
ALB 199	ALB	2	ND	immature	10-19		5100 ± 70 (TO-10375)	(Silberbauer, 1979)	Vygeboom; Zuurberg
ALB 200	ALB	1	[M]	immature	10-34		5105 ± 20 (Pta-8638)	(Silberbauer, 1979)	Vygeboom; Zuurberg
ALB 201	ALB	3	ND	immature	birth-5				Hlalela
ALB 202	ALB	3	ND	mature	>20				Hlalela
ALB 204	ALB	3	[M]	immature	20-34		3240 ± 60 (Pta-8690)	(Silberbauer, 1979)	Vygeboom; Zuurberg
ALB 205	ALB	2	ND	immature	2-9		3810 ± 70 (TO-10239)	(Silberbauer, 1979)	Vygeboom; Zuurberg
ALB 206	ALB	3	ND	immature	20-34				Vygeboom, Zuurberg
ALB 208	ALB	2	[M]	mature	>50				Alicedale
ALB 209	ALB	2	ND	immature	6-9				Glen Boyd
ALB 210	ALB	2	[M]	mature	>20				Adelaide
ALB 212	ALB	3	ND	immature	6-19				Bowden
ALB 213	ALB	3	ND	mature	>20				Bowden
ALB 214	ALB	3	A	mature	>20				Blouberg
ALB 215	ALB	2	[F]	immature	10-34				Thaba Nchu
ALB 216	ALB	1	M	mature	35-49		490 ± 50 (Pta-8606)	(Silberbauer, 1979)	Bowden

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
ALB 217	ALB	3	ND	immature	10-34		1670 ± 20 (Pta-8613)	(Silberbauer, 1979)	Glen Boyd
ALB 219	ALB	3	A	mature	>20				Saliba Dam
ALB 221	ALB	3	ND	mature	>20		4180 ± 70 (Pta-8736)	(Silberbauer, 1979)	Kleinemonde
ALB 222	ALB	1	M	immature	20-34		2640 ± 60 (Pta-8636)	(Stynder et al., 2007a)	Seal point
ALB 223	ALB	2	[F]	immature	10-34		1650 ± 60 (Pta-8631)	(Silberbauer, 1979)	Seal point
ALB 224	ALB	3	ND	immature	2-5				Seal point
ALB 225	ALB	1	[M]	immature	10-34				Glen Ovis
ALB 226 A	ALB	3	ND	immature	2-5				Sidbury
ALB 226 B	ALB	3	[M]	mature	>20				Spitzkop
ALB 227	ALB	3	F	mature	20-49				Cape Recife
ALB 228	ALB	3	[M]	mature	>20				Grahamstown
ALB 230 [/229]	ALB	3	A	mature	>20				Governors Kop
ALB 230	ALB	3	F	mature	>35				Hermanus
ALB 231	ALB	3	[M]	mature	>20				Grahamstown
ALB 232	ALB	3	A	mature	>20				Grahamstown
ALB 233	ALB	3	A	mature	>20				Port Alfred
ALB 234	ALB	2	[M]	mature	35-49				Green Hills
ALB 235	ALB	1	[M]	mature	35-49		420 ± 50 (Pta-8611)	(Silberbauer, 1979)	Governors Kop
ALB 236	ALB	2	ND	immature	birth-1		2100 ± 60 (TO-10377)	(Silberbauer, 1979)	Midford Park
ALB 237	ALB	1	F	mature	20-34				Plettenberg Bay
ALB 238	ALB	2	ND	immature	6-19		5550 ± 70 (TO-10378)	(Silberbauer, 1979)	Widcombe
ALB 240	ALB	3	A	immature	10-34				

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
ALB 241	ALB	2	[F]	immature	10-34				Aylesbury
ALB 243	ALB	2	[F]	mature	>20				Kommadagga
ALB 244 (3)	ALB	2	F	immature	10-34				Paardefontein
ALB 244 (2)	ALB	2	[M]	immature	10-34				Paardefontein
ALB 244 (1)	ALB	1	F	mature	35-49		1180 ± 50 (Pta-8587)	(Silberbauer, 1979)	Paardefontein
ALB 244 (B)	ALB	3	A	mature	>20				Paardefontein
ALB 244 C	ALB	3	F	mature	>20				Paardefontein
ALB 245	ALB	1	M	mature	>20				Olivewood
ALB 246	ALB	1	ND	immature	10-19				Port Alfred
ALB 247	ALB	3	ND	mature	>20				Olivewood
ALB 248	ALB	3	[M]	mature	>20				
ALB 249	ALB	3	A	mature	>20				
ALB 250	ALB	3	[M]	mature	>20				
ALB 251	ALB	3	ND	immature	birth-5				
ALB 252	ALB	3	ND	mature	>20				Woody Cape
ALB 253	ALB	3	[M]	mature	>20				Greys Gift
ALB 255	ALB	3	[M]	immature	10-19				
ALB 256	ALB	3	[M]	mature	>20				
ALB 258	ALB	3	A	mature	>20				Kwaai Hoek
ALB 259	ALB	2	F	mature	35-49				Addo
ALB 260	ALB	3	[M]	mature	>20				Krans Drift
ALB 261	ALB	1	M	mature	20-34				Port Alfred
ALB 262	ALB	3	F	mature	>20				Rufane river mouth
ALB 263	ALB	2	[M]	mature	35-49				Thusford
ALB 265	ALB	2	ND	immature	prenatal-1		5934 ± 33 (OxA-13569)	(Silberbauer, 1979)	Trappe's Valley
ALB 266	ALB	3	ND	immature	2-9				Trappe's Valley
ALB 267	ALB	1	F	mature	>20				Grahamstown
ALB 268	ALB	3	ND	immature	6-19				Trappe's Valley
ALB 269	ALB	3	[M]	mature	>20				Port Alfred
ALB 270	ALB	1	ND	immature	6-19				Port Alfred

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
ALB 271	ALB	2	[M]	immature	10-34				The Willows
ALB 272	ALB	1	ND	immature	10-19				Teasdale
ALB 273	ALB	1	M	mature	35-49				Teasdale
ALB 274 [A]	ALB	3	ND	immature	2-5				Strowan
ALB 274 [B]	ALB	3	ND	immature	prenatal-1				Strowan
ALB 275 [A]	ALB	3	[F]	mature	>20				Kenton-on-Sea
ALB 275 [B]	ALB	3	[M]	mature	>20				Kenton-on-Sea
ALB 276 [A]	ALB	3	ND	immature	6-19				Cape St Francis
ALB 276 [B]	ALB	3	ND	immature	6-19				Cape St Francis
ALB 277	ALB	1	M	mature	35-49		670 ± 50 (Pta-8685)	(Silberbauer, 1979)	Cape St Francis
ALB 279	ALB	3	ND	mature	>20				Cape St Francis
ALB 280	ALB	3	[F]	mature	>20				Krans Drift
ALB 281	ALB	3	[M]	mature	>20				Salisbury Plain
ALB 282	ALB	2	F	mature	>20		570 ± 50 (Pta-8580)	(Pfeiffer and Sealy, 2006)	Plettenberg Bay
ALB 283	ALB	2	M	mature	35-49				Governors Kop
ALB 284	ALB	2	ND	immature	2-5				Mount Marlow
ALB 285	ALB	2	[F]	mature	>20				Mount Marlow
ALB 286	ALB	3	F	mature	>20				Qora
ALB 287	ALB	3	ND	immature	birth-19				Sea Field
ALB 289	ALB	3	F	mature	>20				Cape St Francis
ALB 290	ALB	3	A	mature	>20				Uitenhage
ALB 291 [B]	ALB	3	ND	immature	6-19				Springmound
ALB 291 A	ALB	3	ND	immature	6-19				Springmound
ALB 292	ALB	3	A	mature	>20				Springmound
ALB 293	ALB	1	F	mature	35-49				Boknes
ALB 294	ALB	2	[M]	mature	>20				Goodhope
ALB 295	ALB	1	ND	immature	birth-1		1860 ± 50 (Pta-4636)	(Pfeiffer and Harrington, 2011)	Klasies River Mouth

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
ALB 296	ALB	2	ND	immature	10-19		2180 ± 50 (Pta-8672)	(Pfeiffer and Harrington, 2011)	Klasies River Mouth
ALB 297	ALB	3	ND	immature	birth-1				Klasies River Mouth
ALB 298	ALB	3	ND	immature	birth-1				Klasies River Mouth
ALB 299	ALB	2	ND	immature	birth-1		3210 ± 70 (TO-10380)	(Pfeiffer and Harrington, 2011)	Klasies River Mouth
ALB 300	ALB	2	ND	immature	birth-1				St Francis Bay
ALB 301	ALB	2	[M]	mature	>20		2570 ± 50 (Pta-8684)	(Stynder et al., 2007a)	St Francis Bay
ALB 302	ALB	2	F	mature	35-49				Goedgeloof
ALB 303	ALB	1	M	mature	20-49				Goedgeloof
ALB 304	ALB	2	[F]	mature	>20				Goedgeloof
ALB 305	ALB	2	F	mature	35-49				Hamburg
ALB 306	ALB	3	[M]	mature	35-49				
ALB 307	ALB	2	M	mature	20-49				
ALB 308	ALB	1	M	mature	35-49				Welgeluk
ALB 309	ALB	2	[M]	mature	>20				Welgeluk
ALB 310	ALB	3	ND	mature	>20				Welgeluk
ALB 311	ALB	1	ND	immature	birth-5		4694 ± 32 (OxA-13570)	(Pfeiffer and Harrington, 2011)	Welgeluk
ALB 312	ALB	2	ND	immature	birth-5				Welgeluk
ALB 313	ALB	3	ND	immature	6-19				Welgeluk
ALB 314	ALB	2	M	mature	20-49				Seafield
ALB 315	ALB	3	[F]	mature	>20				Woody Cape
ALB 316	ALB	1	F	mature	35-49				Groot Kommandokloof
ALB 317	ALB	2	ND	immature	prenatal-1				Groot Kommandokloof
ALB 318	ALB	2	ND	immature	2-9				Kenton-on-Sea
ALB 319 A	ALB	2	[F]	immature	10-34				Joubertina
ALB 319 B	ALB	3	ND	immature	birth-5				Joubertina
ALB 320	ALB	3	[F]	mature	2-9				Springmound

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
ALB 321	ALB	3	ND	mature	>20				Saasveld
ALB 322	ALB	2	ND	immature	birth-1				Klasies River Mouth
ALB 323	ALB	1	F	mature	35-49		1620 ± 35 (Pta-8578)	(Stynder et al., 2007a)	Goedgeloof
ALB 324	ALB	3	[F]	mature	>20				Thysbaai
ALB 325	ALB	2	ND	immature	6-19				Loerie
ALB 326	ALB	3	[F]	mature	>20				Andries Vosloo Kudu Reserve
ALB 327	ALB	3	ND	immature	birth-1				Cambria
ALB 328	ALB	2	M	mature	20-49				Cape St Francis
ALB 329	ALB	2	A	mature	>20				Eersterivier
ALB 331	ALB	3	[F]	mature	>20				Glendower
ALB 332	ALB	3	ND	immature	2-5				
ALB 333	ALB	3	ND	immature	2-9				Boknes
ALB 334	ALB	3	ND	mature	>20				Mbashe River Mouth
ALB 336	ALB	3	M	mature	>20				
ALB 337	ALB	2	ND	immature	prenatal-1				Klasies River Mouth
ALB 338	ALB	1	F	mature	35-49				Oyster Bay
ALB 339	ALB	1	F	mature	35-49				Port Alfred
ALB 340	ALB	3	A	mature	>20				Murraysburg
ALB 341	ALB	2	[F]	mature	>20				Cape St Francis
ALB 342	ALB	3	ND	immature	birth-9				
ALB 343	ALB	1	M	mature	35-49				Queensberry Bay
ALB 344	ALB	2	F	immature	10-34				Gonubie
ALB 345	ALB	3	F	mature	>20				St Francis Bay
ALB 346	ALB	3	[F]	mature	>20				
ALB 347	ALB	1	F	immature	10-34				Bushman's River
ALB 348	ALB	2	[M]	mature	>20				Kei Mouth
ALB 349	ALB	1	M	mature	35-49				Osbosch
ALB 350	ALB	2	ND	immature	6-19				Oyster Bay
ALB 351	ALB	1	ND	immature	10-19				New Papiesfontein

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
ALB 352	ALB	3	[M]	mature	>20				Andries Vosloo Kudu Reserve
ALB 353	ALB	1	M	mature	35-49				Woodbury
ALB 354	ALB	1	F	immature	20-34		3340 ± 60 (Pta-8680)	(Stynder et al., 2007a)	Paradise Beach
ALB 355	ALB	2	ND	immature	prenatal-1				Kulubele
ALB 357	ALB	2	ND	immature	prenatal-1				Goedgeloof
ALB 358	ALB	3	M	mature	>20				Fort Beaufort
ALB 359	ALB	3	ND	mature	>20				Rachels River
ALB 361	ALB	3	A	mature	>20				Gamtoos/Loerie
ALB 362	ALB	3	M	mature	>20				
ALB 363	ALB	1	[F]	mature	35-49				St Francis Bay
ALB 364	ALB	1	[F]	mature	35-49				St Francis Bay?
ALB 365	ALB	2	M	mature	35-49				Hankey
ALB 366 A	ALB	3	[F]	mature	>20				Steytlerville
ALB 366 B	ALB	3	A	mature	>20				Steytlerville
ALB 368	ALB	3	[F]	mature	>20				Cape St Francis?
ALB 369	ALB	3	ND	immature	2-9				Aberdeen
ALB 370	ALB	2	F	mature	20-34				Riet Vlei
ALB 371	ALB	3	ND	immature	6-19				Paradise Beach
ALB 372	ALB	3	[F]	mature	>20				Coega
ALB 373	ALB	3	F	mature	>20				St Francis Bay
ALB 374	ALB	2	A	mature	>20				Cape St Francis
ALB 375	ALB	2	A	mature	>20				Hankey
ALB 376	ALB	1	F	immature	10-34				Hankey
ALB 377	ALB	2	F	mature	35-49				
ALB 378	ALB	2	F	mature	20-49				Blue Horizon Bay
ALB 379	ALB	2	ND	immature	10-19				Port Elizabeth

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
ALB ? (not marked/accessioned and in box with ALB 201-9)	ALB	3	ND	immature	2-19				
ALB [278]	ALB	3	ND	mature	>20				Cape St Francis
ALB [32]	ALB	3	[M]	mature	>20				
ALB 192-7? Not accessioned	ALB	3	ND	immature	prenatal-1				Melkhoutboom
ALB Plettenbery Bay Box2	ALB	1	F	mature	20-49				Plettenberg Bay
ALB (unlabelled) 11.04.03 Army base	ALB	3	ND	mature	>20				
ALB (unlabelled) Cape St Francis	ALB	1	ND	immature	2-9				Cape St Francis
ALB (unmarked) Ugie EC [Could be 380 - there is an entry in the catalogue for this no. for Ugie]	ALB	3	F	mature	>20				Ugie
MMK GLD 8.1	MMK	1	ND	immature	10-34	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD 8.10	MMK	1	M	immature	20-34	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD 8.2	MMK	1	[M]	mature	35-49	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD 8.3	MMK	1	M	mature	35-49	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD 8.4	MMK	1	M	immature	20-34	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD 8.5	MMK	1	M	immature	10-34	1898-1900		(van der Merwe et al., 2010b)	Gladstone

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
MMK GLD 8.6	MMK	1	ND	immature	10-19	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD 8.7	MMK	2	ND	immature	prenatal-1	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD 8.8	MMK	1	M	immature	10-19	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD 8.9	MMK	1	ND	immature	birth-1	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD 31 E1	MMK	1	M	mature	35-49	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD 31 E2	MMK	1	ND	immature	10-19	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD 31 E3	MMK	1	M	mature	35-49	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD 31 E4	MMK	1	M	mature	>35	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD 31 E5	MMK	1	M	mature	35-49	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD 34.1	MMK	3	[M]	mature	>20	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD 34.10	MMK	1	M	immature	20-34	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD 34.11	MMK	1	M	immature	10-34	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD 34.12	MMK	1	M	mature	20-34	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD 34.13	MMK	1	M	immature	10-19	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD 34.14	MMK	1	M	immature	20-34	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD 34.2 [A]	MMK	2	ND	mature	20-34	1898-1900		(van der Merwe et al., 2010b)	Gladstone

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
MMK GLD 34.2 B	MMK	1	[M]	immature	10-34	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD 34.3	MMK	1	M	mature	35-49	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD 34.4	MMK	1	ND	immature	10-19	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD 34.5	MMK	1	M	immature	10-34	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD 34.6	MMK	1	M	immature	20-34	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD 34.7	MMK	1	M	mature	35-49	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD 34.8	MMK	1	M	immature	20-34	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD 34.9	MMK	1	M	immature	10-34	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD 38.1	MMK	1	ND	immature	10-19	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD 38.2	MMK	2	M	immature	20-34	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD 38.4	MMK	1	M	immature	20-34	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD 38.5	MMK	1	M	immature	10-34	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD 38.6	MMK	1	M	mature	20-34	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD 38.7	MMK	1	M	mature	35-49	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD 38.8	MMK	1	M	mature	35-49	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD 38.9	MMK	1	M	mature	35-49	1898-1900		(van der Merwe et al., 2010b)	Gladstone

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
MMK GLD 74.1	MMK	1	M	mature	35-49	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD 74.10	MMK	1	ND	immature	10-19	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD 74.11	MMK	1	ND	immature	10-19	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD 74.12	MMK	1	M	immature	20-34	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD 74.2	MMK	1	M	immature	10-34	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD 74.3	MMK	1	M	immature	10-34	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD 74.4	MMK	1	M	mature	35-49	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD 74.5	MMK	1	M	mature	20-34	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD 74.6	MMK	1	M	mature	35-49	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD 74.7	MMK	1	M	mature	35-49	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD 74.8	MMK	1	ND	immature	10-19	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD 74.9	MMK	1	[M]	immature	20-34	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD N 100.1	MMK	1	M	mature	35-49	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD N 100.2	MMK	1	M	mature	35-49	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD N 100.3	MMK	1	M	mature	35-49	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD N 100.4	MMK	1	M	immature	20-34	1898-1900		(van der Merwe et al., 2010b)	Gladstone

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
MMK GLD N 100.4 [100.5?]	MMK	1	ND	immature	10-19	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD NOP 3/4.1	MMK	1	M	mature	20-34	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD NOP 3/4.2	MMK	1	M	mature	35-49	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD S 1.1	MMK	3	[M]	mature	>20	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD S 1.2	MMK	3	M	mature	>20	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD S 1.3	MMK	2	M	immature	10-34	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD S 1.4	MMK	1	M	immature	10-34	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD S 1.5	MMK	1	M	mature	35-49	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD S 1.6	MMK	1	M	immature	10-34	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD S 2.10	MMK	1	M	immature	20-34	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD S 2.2	MMK	2	M	mature	35-49	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD S 2.3	MMK	2	A	immature	10-34	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD S 2.4	MMK	1	A	mature	35-49	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD S 2.5	MMK	1	[M]	mature	35-49	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD S 2.6	MMK	1	M	immature	20-34	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD S 2.7	MMK	1	[M]	mature	20-34	1898-1900		(van der Merwe et al., 2010b)	Gladstone

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
MMK GLD S 2.8	MMK	1	M	mature	20-34	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD S 2.9	MMK	1	M	mature	20-49	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD S 3.1	MMK	2	M	mature	20-49	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD S 3.2	MMK	1	M	mature	35-49	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD S 3.3	MMK	1	M	immature	20-34	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD S 3.4	MMK	1	M	mature	20-49	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD S 3.5	MMK	1	M	mature	20-49	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD S 3.6	MMK	1	M	immature	10-34	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD S 3.7	MMK	1	M	mature	>50	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD S 4.1	MMK	1	[F]	mature	35-49	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD S 5.1	MMK	1	M	mature	35-49	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD S 2.1?	MMK	2	[M]	mature	>20	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD SE 7	MMK	3	ND	mature	>20	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD SE 7.1	MMK	3	ND	mature	>20	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD SE 7.10	MMK	2	ND	immature	birth-1	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD SE 7.11	MMK	1	F	mature	35-49	1898-1900		(van der Merwe et al., 2010b)	Gladstone

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
MMK GLD SE 7.2	MMK	2	ND	mature	>20	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD SE 7.3	MMK	1	M	mature	20-49	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD SE 7.4	MMK	1	M	mature	35-49	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD SE 7.5	MMK	1	F	mature	35-49	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD SE 7.6	MMK	1	M	mature	20-34	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD SE 7.7	MMK	1	[M]	immature	10-34	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD SE 7.8	MMK	1	M	mature	20-49	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD SE 7.9	MMK	1	[M]	mature	20-34	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD SE 11.1	MMK	2	[M]	mature	>20	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD SE 11.2	MMK	2	ND	mature	>20	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD SE 11.3	MMK	2	M	mature	35-49	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD SE 11.4	MMK	1	M	immature	10-34	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD SE 11.5	MMK	1	[M]	immature	10-34	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD SE 11.6	MMK	1	M	mature	20-49	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD SE 15.2	MMK	2	M	mature	35-49	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD SE 16	MMK	3	[M]	mature	>20	1898-1900		(van der Merwe et al., 2010b)	Gladstone

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
MMK GLD SE 12.1 and 12.2	MMK	2	[M]	mature	>20	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD SE 15.1 [A]	MMK	2	M	mature	35-49	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD SE 15.1 [B]	MMK	2	A	immature	10-34	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK GLD SE 15.1 [C]	MMK	3	ND	immature	10-34	1898-1900		(van der Merwe et al., 2010b)	Gladstone
MMK 128	MMK	3	A	mature	>20				Cape Seal
MMK 130	MMK	3	[M]	mature	>20				
MMK 140	MMK	3	A	mature	>20				Barkly West
MMK 152	MMK	1	M	immature	20-34				
MMK 153	MMK	3	F	immature	10-34				Barkly West
MMK 164	MMK	1	ND	immature	10-19				
MMK 169	MMK	2	M	mature	35-49				Barkly West
MMK 170	MMK	3	[F]	immature	10-34				Riverton
MMK 171	MMK	1	ND	immature	6-19				Herbert
MMK 173	MMK	1	[M]	mature	>20				Douglas
MMK 176	MMK	3	ND	immature	6-19				Kleinsee
MMK 181	MMK	3	[M]	mature	>20				Riverton
MMK 183	MMK	3	ND	immature	birth-1				Hawston
MMK 184	MMK	3	A	mature	>20				Hawston
MMK 185	MMK	1	M	mature	35-49				Hopetown
MMK 186	MMK	1	F	immature	10-34				Hopetown
MMK 187	MMK	3	M	mature	>20				Modder river
MMK 188	MMK	3	ND	immature	6-19				Longlands
MMK 189	MMK	1	M	mature	>35				Koffiefontein
MMK 190	MMK	2	[M]	mature	35-49				Koffiefontein
MMK 191	MMK	1	M	mature	35-49				Prieska
MMK 192	MMK	3	[F]	mature	>20				Koffiefontein
MMK 193	MMK	3	F	mature	>20				Kimberley
MMK 194	MMK	3	[M]	mature	>20				Koffiefontein

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
MMK 195	MMK	2	M	mature	35-49				Koffiefontein
MMK 197	MMK	2	F	mature	>20				Koffiefontein
MMK 198	MMK	3	[M]	mature	>20				Koffiefontein
MMK 199	MMK	3	ND	immature	2-5				Koffiefontein
MMK 200	MMK	3	ND	immature	6-19				Koffiefontein
MMK 201	MMK	2	M	mature	35-49				Koffiefontein
MMK 202	MMK	2	A	mature	>20				Koffiefontein
MMK 203	MMK	1	ND	immature	10-19				Koffiefontein
MMK 204	MMK	1	F	mature	35-49				Koffiefontein
MMK 205	MMK	2	ND	immature	birth-5				Koffiefontein
MMK 206	MMK	1	F	immature	10-34				Koffiefontein
MMK 207	MMK	3	ND	immature	2-5				Koffiefontein
MMK 208	MMK	2	[M]	mature	35-49				Koffiefontein
MMK 209	MMK	1	ND	immature	10-19				Koffiefontein
MMK 210	MMK	3	F	mature	>20				
MMK 211	MMK	3	A	mature	>20				Koffiefontein
MMK 212	MMK	3	A	mature	>20				Koffiefontein
MMK 213	MMK	1	F	mature	35-49				Koffiefontein
MMK 214	MMK	2	ND	immature	2-5				Koffiefontein
MMK 217	MMK	1	F	immature	10-34				Koffiefontein
MMK 218	MMK	2	ND	immature	6-19				Koffiefontein
MMK 219	MMK	3	ND	mature	>20				Koffiefontein
MMK 220	MMK	1	M	mature	35-49				Koffiefontein
MMK 221	MMK	3	[M]	mature	>20				Koffiefontein
MMK 222	MMK	1	ND	immature	10-19				Koffiefontein
MMK 223	MMK	2	ND	immature	2-5				Koffiefontein
MMK 224	MMK	3	F	mature	>20				Koffiefontein
MMK 225	MMK	3	ND	immature	birth-5				Koffiefontein
MMK 226	MMK	3	[M]	mature	>20				Laingsburg
MMK 227	MMK	3	A	mature	>20				Laingsburg
MMK 228	MMK	1	ND	immature	10-19				Koffiefontein
MMK 229	MMK	1	ND	immature	10-19				Koffiefontein

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
MMK 230 A	MMK	3	ND	immature	6-19				Koffiefontein
MMK 230	MMK	2	ND	immature	birth-9				Koffiefontein
MMK 231	MMK	1	M	mature	35-49				Koffiefontein
MMK 232	MMK	3	ND	immature	2-5				Koffiefontein
MMK 233	MMK	2	[F]	mature	>35				Prieska
MMK 234 [A]	MMK	2	[M]	mature	>20				Koffiefontein
MMK 234 [B]	MMK	3	ND	immature	10-19				Koffiefontein
MMK 235	MMK	1	F	mature	35-49		390 ± 50 (Pta-2894)	(Morris, 1992a, 1984)	Koffiefontein
MMK 236	MMK	1	F	mature	35-49				Koffiefontein
MMK 237	MMK	1	F	immature	20-34				Koffiefontein
MMK 238 [A]	MMK	1	ND	immature	2-5				Koffiefontein
MMK 238 [B]	MMK	3	ND	mature	>20				Koffiefontein
MMK 239	MMK	2	[F]	mature	>20				Koffiefontein
MMK 242	MMK	2	M	mature	>20				Douglas
MMK 243	MMK	3	[M]	mature	>20				Douglas
MMK 244	MMK	2	[M]	mature	>20				Douglas
MMK 245	MMK	1	ND	immature	10-19				Koffiefontein
MMK 246	MMK	2	ND	immature	birth-5				Koffiefontein
MMK 247 [A]	MMK	2	ND	immature	10-19				Koffiefontein
MMK 247 [C]	MMK	2	M	mature	20-34				Koffiefontein
MMK 248	MMK	1	ND	immature	10-19				Koffiefontein
MMK 249	MMK	1	ND	immature	10-19				Koffiefontein
MMK 250	MMK	1	F	immature	20-34				Koffiefontein
MMK 253	MMK	3	M	mature	>20				Windsorton
MMK 255	MMK	2	[F]	mature	>20				Koffiefontein
MMK 256	MMK	3	M	mature	>20				
MMK 257	MMK	3	ND	immature	20-34				Douglas
MMK 258	MMK	3	M	mature	>20				Port Nolloth
MMK 260	MMK	3	ND	immature	2-5				Hopetown
MMK 261	MMK	3	M	mature	>20				Griquatown
MMK 263	MMK	3	F	mature	>20				Parys

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
MMK 265	MMK	3	M	mature	>20				Douglas
MMK 266	MMK	3	[F]	mature	>20				Douglas
MMK 267	MMK	3	[M]	mature	>20				Campbell
MMK 268	MMK	3	A	mature	>20				Douglas
MMK 269	MMK	3	[M]	mature	>20				Upington
MMK 270	MMK	3	[M]	mature	>20				Upington
MMK 271	MMK	3	A	mature	>20				
MMK 272	MMK	3	A	mature	>20				Koffiefontein
MMK 273	MMK	3	M	mature	>20				Hopetown
MMK 274	MMK	1	ND	immature	10-19				Longlands
MMK 275	MMK	3	A	mature	>20				Sidney-on-Vaal
MMK 276	MMK	2	A	immature	10-34				Middel Plaats
MMK 277	MMK	2	F	immature	10-34		890 ± 50 (Pta-2898)	(Morris, 1992a, 1984)	Weltevrede
MMK 278	MMK	1	M	mature	35-49				Blaauw Bosch Drift
MMK 279	MMK	3	[F]	mature	>20				Kaaimans River
MMK 281	MMK	1	F	mature	35-49				Reads Drift
MMK 282 [A]	MMK	2	M	mature	35-49				Kirkwood
MMK 282 [B]	MMK	3	F	mature	>20				
MMK 282 [C]	MMK	3	[F]	mature	>20				
MMK 282 [D]	MMK	3	F	mature	>20				
MMK 284	MMK	3	[M]	mature	>20				Upington
MMK 285	MMK	3	F	mature	>20				Douglas
MMK 286	MMK	3	[M]	mature	>20				Douglas
MMK 287	MMK	3	[M]	mature	>20				Koffiefontein
MMK 288	MMK	2	A	mature	>20				Kimberley
MMK 289	MMK	3	M	mature	>20				Louisvale
MMK 290	MMK	3	F	mature	>20				Louisvale
MMK 291	MMK	3	A	mature	>20				Louisvale
MMK 292	MMK	3	A	mature	>20				Koffiefontein
MMK 293	MMK	3	A	mature	>20				Louisvale
MMK 294	MMK	3	A	mature	>20				Springfield?

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
MMK 295	MMK	1	[M]	mature	35-49				Springfield?
MMK 296	MMK	1	M	mature	35-49				Driekopseiland
MMK 297	MMK	3	M	mature	>20				Witput
MMK 298	MMK	3	[F]	mature	>20				Smiths Mine
MMK 299	MMK	3	ND	immature	birth-19				Soutpan
MMK 300	MMK	3	[F]	mature	>20				Soutpan
MMK 301	MMK	3	F	mature	>20				Soutpan
MMK 302	MMK	3	[M]	mature	>20				Soutpan
MMK 303	MMK	3	M	mature	>20				Soutpan
MMK 304	MMK	3	M	mature	>20				Soutpan
MMK 305	MMK	3	[F]	mature	>20				Soutpan
MMK 306	MMK	3	M	mature	>20				Soutpan
MMK 307	MMK	3	F	mature	>20				Soutpan
MMK 308	MMK	1	ND	immature	10-19				Soutpan
MMK 311	MMK	2	A	mature	>20				
MMK 312	MMK	1	[M]	immature	20-34				Vogelfontein
MMK 313	MMK	3	F	mature	>20				Reivilo
MMK 314	MMK	3	ND	immature	10-19				Prieska
MMK 316	MMK	1	ND	immature	10-19				Douglas
MMK 317 [A]	MMK	1	F	mature	35-49				Douglas
MMK 317 [B]	MMK	3	ND	immature	prenatal-1				Douglas
MMK 318	MMK	3	[F]	mature	>20				Bannockburn?
MMK 319	MMK	3	ND	mature	>20				Modder River Station
MMK 321	MMK	1	M	mature	35-49				Douglas
MMK 322	MMK	2	F	mature	>20				Weltevreden
MMK 323	MMK	3	ND	mature	>20				Weltevreden
MMK 324	MMK	3	A	mature	>20				Weltevreden
MMK 325	MMK	2	A	mature	35-49		3360 ± 60 (Pta-681)	(Humphreys, 2007, 1974)	Weltevreden
MMK 326	MMK	2	[M]	mature	>20				Wonderfontein

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
MMK 327	MMK	3	ND	mature	>20				Vleermuisgat Vaal Kop
MMK 328	MMK	3	ND	mature	>20				Pniel Estate
MMK 329	MMK	1	M	mature	35-49		110 ± 50 (Pta-247)	(Maggs, 1971)	Koffiefontein
MMK 330	MMK	2	F	mature	35-49		380 ± 50 (Pta-248)	(Maggs, 1971)	Koffiefontein
MMK 331	MMK	3	[F]	mature	>20				Transvaal Road, Police station
MMK 332	MMK	2	ND	mature	35-49				Reads Drift
MMK 333	MMK	3	ND	immature	2-5				Taung
MMK 334	MMK	2	ND	immature	6-19				Zand Plaats
MMK 335	MMK	1	[F]	immature	20-34				Douglas
MMK 336	MMK	3	ND	immature	6-19				De Hoop
MMK 337	MMK	3	ND	mature	>20				Nooitgedacht
MMK 338	MMK	2	[F]	mature	20-34				
MMK 340	MMK	3	[F]	mature	>20				Spitskop
MMK ? Unaccessioned black box	MMK	1	M	mature	35-49				
MMK Border Cave (Homo)	MMK	3	ND	immature	10-19				Border Cave
MMK Calvinia museum sample	MMK	1	[M]	immature	20-34				
MMK Calvinia washed out skeleton 1	MMK	3	[F]	mature	>20				Calvinia
MMK Calvinia washed out skeleton 2	MMK	2	[F]	mature	>20				Calvinia
MMK Equus Cave (Homo)	MMK	3	ND	mature	>20				Taung
MMK Ex Rolfontein	MMK	3	F	mature	>20				Van Der Kloof Dam
MMK Murton [A]	MMK	1	ND	immature	6-19				
MMK Murton [B]	MMK	1	ND	immature	birth-1				

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
MMK Pofadder East	MMK	1	M	mature	35-49				Pofadder
MMK Rietfontein	MMK	1	[F]	immature	20-34				
MMK Setlegole (SAPS)	MMK	2	[F]	mature	>20				Setlagole
MMK Transvaal Rd B (was grave 3)	MMK	2	A	mature	>20				Kimberley
MMK Transvaal Rd C (was 4)	MMK	3	ND	immature	10-19				Kimberley
MMK Transvaal Rd D (was 2)	MMK	2	[M]	mature	>20				Kimberley
MMK Unmarked	MMK	2	ND	mature	>20				
MMK unprov skull archaeol dept	MMK	3	ND	immature	2-5				
MMK Voetpadsdrift West	MMK	1	M	mature	20-34				Voetpadsdrift West
MMK Windsorton	MMK	3	[F]	mature	>20				Windsorton
NMB 1	NMB	3	A	mature	>20				Douglas
NMB 4	NMB	3	[M]	mature	>20		3940 ± 27 (OxA-V-2064-48)	(Sealy, 2006)	Robberg
NMB 5	NMB	3	[F]	mature	>20		1423 ± 26 (OxA-V-2064-49)	(Sealy, 2006)	Plettenberg Bay
NMB 6	NMB	3	ND	immature	6-19				Matjies River Rock Shelter
NMB 9	NMB	3	ND	mature	>20				Matjies River Rock Shelter
NMB 10	NMB	3	ND	mature	>20				Matjies River Rock Shelter
NMB 11	NMB	2	ND	immature	2-9				Vegkop
NMB 12	NMB	2	F	mature	35-49				Vegkop
NMB 13	NMB	3	F	mature	>20				Doringberg
NMB 14	NMB	3	M	mature	>20				Houdenbek

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
NMB 15	NMB	3	A	mature	>20				Olifantskop
NMB 16	NMB	3	[F]	mature	>20	1900s		(Hoffman, 1942)	Kareeboom
NMB 17	NMB	3	M	mature	>20	1900s		(Hoffman, 1942)	Kareeboom
NMB 18	NMB	3	[M]	mature	>20	1900s		(Hoffman, 1942)	Kareeboom
NMB 19	NMB	3	ND	immature	10-19	1900s		(Hoffman, 1942)	Kareeboom
NMB 21	NMB	3	A	mature	>20	1900s		(Hoffman, 1942)	Kareeboom
NMB 22	NMB	2	[F]	immature	10-34	1900s		(Hoffman, 1942)	Kareeboom
NMB 23 A and B	NMB	2	[M]	mature	>20	1900s		(Hoffman, 1942)	Kareeboom
NMB 24 A and B	NMB	1	ND	immature	10-19	1900s		(Hoffman, 1942)	Kareeboom
NMB 25	NMB	3	ND	immature	6-19				Taaibosspruit, Blanco
NMB 29	NMB	3	M	mature	>20				Brandfort
NMB 52	NMB	3	A	mature	>20				Bethulie
NMB 59	NMB	3	[F]	mature	>20				Goemansberg
NMB 60	NMB	3	[F]	mature	>20				Alice
NMB 61	NMB	3	[M]	mature	>20				Koffiefontein
NMB 62	NMB	3	ND	mature	>20				Leeudoringstad
NMB 63	NMB	3	[F]	mature	35-49				Bloemfontein?
NMB 65	NMB	2	F	mature	>20				Jacobsdal
NMB 66	NMB	2	[F]	mature	35-49				Jacobsdal
NMB 67	NMB	2	A	mature	>20				Jacobsdal
NMB 68 A and B	NMB	1	[M]	mature	>35				Olive Hill, Bloemfontein
NMB 69	NMB	2	[F]	mature	>20				Pelgrimsrus, Paternoster
NMB 70	NMB	3	A	mature	>20				Belvedere, Knysna
NMB 71	NMB	3	A	mature	>20				
NMB 73	NMB	3	ND	immature	10-34				
NMB 74 [A]	NMB	3	M	mature	>20				
NMB 74 [B]	NMB	3	A	mature	35-49				
NMB 75	NMB	3	A	mature	>20				
NMB 76	NMB	3	A	mature	>20				

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
NMB 77	NMB	3	ND	mature	>20				
NMB 78	NMB	3	F	mature	>20				Still Bay
NMB 79	NMB	3	[F]	mature	>20				
NMB 80	NMB	3	ND	immature	6-19				
NMB 81	NMB	3	A	mature	>20				Bethulie
NMB 82	NMB	3	M	mature	>20		2335 ± 40 (GrA-23228)	(Stynder et al., 2007a)	Thysbaai
NMB 83	NMB	3	M	mature	>20		1590 ± 40 (GrA-23227)	(Stynder et al., 2007a)	Cape St Francis
NMB 85	NMB	3	[M]	mature	>20				Cape St Francis
NMB 86	NMB	3	F	mature	>20		2705 ± 40 (GrA-23657)	(Stynder et al., 2007a)	Cape St Francis
NMB 87	NMB	3	ND	immature	2-9				Cape St Francis
NMB 89 [A]	NMB	3	A	mature	>20				Cape St Francis
NMB 89 [B]	NMB	3	A	mature	>20				Cape St Francis
NMB 90	NMB	2	F	mature	35-49				Waterpoort
NMB 91 A and B	NMB	1	[M]	immature	20-34				Wellwood
NMB 92	NMB	1	M	mature	35-49				Fonteintjie
NMB 93	NMB	3	F	mature	>20				Brandfort
NMB 94	NMB	3	[M]	mature	>20				
NMB 105	NMB	3	ND	immature	2-19				Matjies River Rock Shelter
NMB 1064 [A]	NMB	3	[F]	mature	>20				Bainsvlei?
NMB 1064 [B]	NMB	3	M	mature	>20				Bainsvlei?
NMB 1064 [C]	NMB	3	A	mature	>20				Bainsvlei?
NMB 1065 A [A]	NMB	3	M	mature	>20				Bainsvlei?
NMB 1065 A [B]	NMB	3	[F]	mature	>20				Bainsvlei?
NMB 1065 P	NMB	3	A	mature	>20				Bainsvlei?
NMB 1066 A [A]	NMB	3	ND	immature	10-19				Bainsvlei?
NMB 1066 A [B]	NMB	3	M	mature	>20				Bainsvlei?
NMB 1066 A [C]	NMB	3	M	mature	>20				Bainsvlei?
NMB 1066 C	NMB	3	M	mature	>20				Bainsvlei?

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
NMB 1067	NMB	3	M	mature	>20				Bainsvlei?
NMB 1069	NMB	3	M	mature	>20				Bloemfontein?
NMB 1070	NMB	3	ND	immature	2-19				Bloemfontein?
NMB 1071	NMB	3	ND	mature	>20				Bloemfontein?
NMB 1101 A and B	NMB	1	M	mature	35-49	late 1800s		(Peckmann, 2002)	Philippolis
NMB 1102	NMB	2	A	mature	35-49	late 1800s		(Peckmann, 2002)	Philippolis
NMB 1103	NMB	1	ND	immature	10-19				Koffiefontein
NMB 1104	NMB	3	F	mature	>20				Augrabies Falls
NMB 1105	NMB	3	ND	mature	>20				Avalon, Rietrivier?
NMB 1106	NMB	3	[M]	mature	>20				
NMB 1185 A and B	NMB	1	ND	immature	10-34	1900s		(Peckmann, 2002)	Kareeboom
NMB 1186 A and B	NMB	1	M	mature	35-49	1900s		(Peckmann, 2002)	Kareeboom
NMB 1193	NMB	2	M	mature	>20	1900s		(Peckmann, 2002)	Kareeboom
NMB 1194 A and B	NMB	2	M	mature	>20	1900s		(Peckmann, 2002)	Kareeboom
NMB 1195	NMB	2	M	mature	35-49	1900s		(Peckmann, 2002)	Kareeboom
NMB 1196	NMB	3	[F]	mature	>20				Klein Brak River
NMB 1198	NMB	3	[F]	mature	>20				Koffiefontein
NMB 1199	NMB	3	ND	immature	2-19				
NMB 1201	NMB	3	ND	immature	2-19				Kalk Fontein
NMB 1202	NMB	2	F	mature	>20		3140 ± 50 (Pta 8801)	(Stynder et al., 2007a)	Great Brak River
NMB 1203	NMB	2	F	mature	>20		2180 ± 50 (Pta 8783)	(Stynder et al., 2007a)	Great Brak River
NMB 1204	NMB	2	F	mature	>20		2210 ± 35 (Pta 8744)	(Stynder et al., 2007a)	Great Brak River
NMB 1206	NMB	3	M	mature	>20				Mossel Bay
NMB 1207 A and B	NMB	1	M	mature	>50		560 ± 50 (Pta 8755)	(Stynder et al., 2007a)	Hartenbos, Mossel Bay
NMB 1208	NMB	1	F	mature	35-49				Koffiefontein
NMB 1209	NMB	2	[M]	mature	>20				Koffiefontein

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
NMB 1210	NMB	2	M	mature	>20				Koffiefontein
NMB 1211	NMB	3	[M]	mature	>20				Koffiefontein
NMB 1212	NMB	2	M	mature	>20				Koffiefontein
NMB 1213	NMB	2	ND	immature	2-5				Koffiefontein
NMB 1214	NMB	3	ND	mature	>20				Koffiefontein
NMB 1215	NMB	1	F	mature	35-49				Koffiefontein
NMB 1216	NMB	1	F	mature	>50				Koffiefontein
NMB 1217	NMB	3	F	mature	>20				Koffiefontein
NMB 1218	NMB	3	M	mature	>20				Somerset West
NMB 1219	NMB	2	M	mature	>20		650 ± 60 (Pta 8804)	(Stynder et al., 2007a)	Great Brak River
NMB 1220	NMB	2	[M]	mature	>20	1900s		(Peckmann, 2002)	Kareeboom
NMB 1221	NMB	2	M	mature	>20	1900s		(Peckmann, 2002)	Kareeboom
NMB 1222	NMB	3	M	mature	>20				Tsitsikamma
NMB 1223	NMB	3	ND	immature	6-19				Outeniqua Mountains
NMB 1224	NMB	3	ND	immature	2-19				Douglas
NMB 1225	NMB	3	M	mature	>20				Kestell
NMB 1226	NMB	3	A	mature	>20				Pretoria
NMB 1227	NMB	3	M	mature	>20				Harrismith
NMB 1228 A and B	NMB	1	M	mature	35-49	1900s		(Peckmann, 2002)	Kareeboom
NMB 1229 A and B	NMB	1	M	mature	35-49	1900s		(Peckmann, 2002)	Kareeboom
NMB 1230	NMB	3	A	mature	>20	1900s		(Peckmann, 2002)	Kareeboom
NMB 1241 [A]	NMB	2	ND	mature	>20		2970 ± 60 (Pta 6958)	(Sealy et al., 2006)	Matjies River Rock Shelter

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
NMB 1241 [B]	NMB	2	ND	mature	>20		3290 ± 90 (Pta 6950)	(Sealy and Pfeiffer, 2000)	Matjies River Rock Shelter
NMB 1242	NMB	3	F	mature	>20		3030 ± 26 (OxA-V-2064-50)	(Sealy et al., 2006)	Matjies River Rock Shelter
NMB 1244	NMB	3	ND	immature	10-19				Matjies River Rock Shelter
NMB 1246	NMB	3	ND	immature	10-19				Matjies River Rock Shelter
NMB 1247	NMB	3	[F]	mature	>20				Matjies River Rock Shelter
NMB 1248	NMB	3	[F]	mature	>20				Matjies River Rock Shelter
NMB 1249	NMB	3	[M]	mature	>20				Matjies River Rock Shelter
NMB 1250	NMB	3	ND	immature	10-19				Matjies River Rock Shelter
NMB 1252	NMB	3	ND	mature	>20				Matjies River Rock Shelter
NMB 1253	NMB	3	ND	immature	2-9				Matjies River Rock Shelter
NMB 1254	NMB	3	ND	immature	birth-5				Matjies River Rock Shelter
NMB 1255	NMB	3	ND	immature	birth-5				Matjies River Rock Shelter

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
NMB 1256	NMB	3	ND	immature	birth-19				Matjies River Rock Shelter
NMB 1257	NMB	3	ND	immature	birth-5				Matjies River Rock Shelter
NMB 1258	NMB	3	ND	immature	birth-5				Matjies River Rock Shelter
NMB 1259	NMB	3	ND	immature	birth-5				Matjies River Rock Shelter
NMB 1260	NMB	2	ND	immature	2-9				Matjies River Rock Shelter
NMB 1261 [A]	NMB	3	ND	immature	2-5				Matjies River Rock Shelter
NMB 1261 [B]	NMB	3	ND	immature	2-19				Matjies River Rock Shelter
NMB 1263	NMB	3	ND	immature	10-19				Matjies River Rock Shelter
NMB 1264	NMB	3	M	mature	>20				Matjies River Rock Shelter
NMB 1265	NMB	3	[M]	mature	>20				Matjies River Rock Shelter
NMB 1266	NMB	3	ND	immature	birth-5				Matjies River Rock Shelter
NMB 1267	NMB	3	[F]	mature	>20				Matjies River Rock Shelter
NMB 1268 [A]	NMB	3	ND	immature	2-5				Matjies River Rock Shelter

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
NMB 1268 [B]	NMB	3	A	mature	>20				Matjies River Rock Shelter
NMB 1268 [C]	NMB	3	ND	immature	2-9				Matjies River Rock Shelter
NMB 1269 [A]	NMB	3	[F]	mature	>20				Matjies River Rock Shelter
NMB 1269 [B]	NMB	3	[M]	mature	>20				Matjies River Rock Shelter
NMB 1269 [C]	NMB	3	[M]	mature	>20				Matjies River Rock Shelter
NMB 1271	NMB	2	F	immature	10-34		3570 ± 50 (Pta 6957)	(Sealy et al., 2006)	Matjies River Rock Shelter
NMB 1272	NMB	3	[F]	mature	>20		7380 ± 120 (UCLA-1746D)	(Protsch and Oberholzer, 1975)	Matjies River Rock Shelter
NMB 1273	NMB	2	[M]	immature	20-34		3050 ± 60 (Pta 6942)	(Sealy and Pfeiffer, 2000)	Matjies River Rock Shelter

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
NMB 1274	NMB	2	M	mature	>20		5120 ± 50 (Pta 6981)	(Sealy and Pfeiffer, 2000)	Matjies River Rock Shelter
NMB 1275	NMB	2	M	mature	35-49		4850 ± 60 (Pta 6986)	(Sealy and Pfeiffer, 2000)	Matjies River Rock Shelter
NMB 1276	NMB	3	ND	immature	birth-5				Matjies River Rock Shelter
NMB 1277	NMB	3	A	mature	>20				Matjies River Rock Shelter
NMB 1278	NMB	3	A	mature	>20				Matjies River Rock Shelter
NMB 1279	NMB	3	M	mature	>20				Matjies River Rock Shelter
NMB 1280	NMB	3	M	mature	>20				Matjies River Rock Shelter
NMB 1281	NMB	3	[M]	mature	20-34		7420 ± 80 (Pta 6988)	(Sealy and Pfeiffer, 2000)	Matjies River Rock Shelter
NMB 1282	NMB	3	A	mature	>20				Matjies River Rock Shelter
NMB 1283	NMB	3	M	mature	>20				Matjies River Rock Shelter
NMB 1284	NMB	3	ND	immature	birth-19				Matjies River Rock Shelter

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
NMB 1285 [A]	NMB	3	M	mature	>20				Matjies River Rock Shelter
NMB 1285 [B]	NMB	3	A	mature	>20				Matjies River Rock Shelter
NMB 1286	NMB	3	[M]	mature	>20				Matjies River Rock Shelter
NMB 1288 [A]	NMB	3	A	mature	>20				Matjies River Rock Shelter
NMB 1288 [B]	NMB	3	[M]	mature	>20				Matjies River Rock Shelter
NMB 1288 [C]	NMB	3	A	mature	>20				Matjies River Rock Shelter
NMB 1290	NMB	3	ND	immature	birth-19				Matjies River Rock Shelter
NMB 1291	NMB	3	[F]	mature	>20				Matjies River Rock Shelter
NMB 1292	NMB	3	[M]	mature	>20				Matjies River Rock Shelter
NMB 1294	NMB	3	[F]	mature	>20				Matjies River Rock Shelter
NMB 1295	NMB	3	[M]	mature	>20				Matjies River Rock Shelter
NMB 1296	NMB	3	[M]	mature	>20				Matjies River Rock Shelter
NMB 1298	NMB	3	A	mature	>20				Matjies River Rock Shelter
NMB 1299 [A]	NMB	3	[F]	mature	>20				Matjies River Rock Shelter
NMB 1299 [B]	NMB	3	ND	immature	birth-19				Matjies River Rock Shelter
NMB 1299 [C]	NMB	3	[M]	mature	>20				Matjies River Rock Shelter

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
NMB 1300	NMB	3	ND	immature	2-9				Matjies River Rock Shelter
NMB 1311 [A]	NMB	3	[M]	mature	>20				Matjies River Rock Shelter
NMB 1311 [B]	NMB	2	ND	immature	2-5				Matjies River Rock Shelter
NMB 1312	NMB	2	[M]	mature	>20		5980 ± 50 (Pta 7983)	(Sealy, 2006)	Plettenberg Bay
NMB 1313 A, B and C	NMB	1	M	mature	>35	1900s		(Peckmann, 2002)	Kareeboom
NMB 1314	NMB	2	ND	immature	10-34	1900s		(Peckmann, 2002)	Kareeboom
NMB 1316	NMB	2	F	immature	20-34				Bloemfontein
NMB 1317	NMB	1	ND	immature	10-19				Bloemfontein
NMB 1319	NMB	3	M	mature	>20		5251 ± 29 (OxA-V-2064-51)	(Sealy, 2006)	Plettenberg Bay
NMB 1324	NMB	3	A	mature	>20		7245 ± 40 (OxA-V-2055-38)	(Sealy, 2006)	Plettenberg Bay
NMB 1326	NMB	2	F	mature	35-49	1900s		(Peckmann, 2002)	Kareeboom
NMB 1327 A and B	NMB	2	[M]	mature	>20	1900s		(Peckmann, 2002)	Kareeboom
NMB 1328 A and B	NMB	1	M	mature	35-49	1900s		(Peckmann, 2002)	Kareeboom
NMB 1329	NMB	2	M	mature	35-49	1900s		(Peckmann, 2002)	Kareeboom
NMB 1331 A and B	NMB	1	M	mature	35-49	1900s		(Peckmann, 2002)	Kareeboom
NMB 1332	NMB	2	[M]	mature	>20				Upington
NMB 1333	NMB	3	ND	immature	2-9				Upington
NMB 1334	NMB	3	A	mature	>20				Rittel Mago
NMB 1335	NMB	3	ND	immature	birth-19				Jacobsdal
NMB 1336	NMB	3	A	mature	>20				
NMB 1337	NMB	3	[M]	mature	>20				Matjies River Rock Shelter
NMB 1338	NMB	3	M	mature	>20		650 ± 35 (GrA 23711)	(Stynder et al., 2007a)	Wittedrift

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
NMB 1340	NMB	3	ND	immature	prenatal-5				Hanover
NMB 1341 C	NMB	3	ND	mature	>20				Robberg
NMB 1342	NMB	2	[M]	mature	>20		9688 ± 36 (OxA-V-2064-56)	(Sealy, 2006)	Matjies River Rock Shelter
NMB 1343	NMB	3	M	mature	>20				Olifantshoek
NMB 1344	NMB	3	[M]	mature	>20				Swartvlei, Sedgefield
NMB 1345	NMB	2	ND	mature	>20	1900s		(Peckmann, 2002)	Kareeboom
NMB 1346 A and B	NMB	2	M	mature	35-49	1900s		(Peckmann, 2002)	Kareeboom
NMB 1347	NMB	3	A	mature	>20				Saldanha Bay
NMB 1348	NMB	3	[M]	mature	>20				Saldanha Bay
NMB 1349	NMB	3	[F]	mature	>20				Mokkiesdam
NMB 1350	NMB	1	ND	immature	10-19				Cradock/Cookhouse
NMB 1351 A and B	NMB	1	M	mature	>35				Knoffelfontein
NMB 1352	NMB	3	ND	immature	birth-9				
NMB 1363	NMB	2	M	mature	>20				Renosterkop
NMB 1364	NMB	2	M	immature	20-34				Kakamas
NMB 1365	NMB	3	[M]	mature	>20				Kakamas
NMB 1366	NMB	2	[M]	mature	>20				Augrabies Falls
NMB 1367	NMB	2	[M]	mature	35-49				Abrahamsdam
NMB 1368	NMB	3	A	mature	>20				Renosterkop
NMB 1369	NMB	2	ND	immature	10-19		150 ± 40 (Pta 2907)	(Morris, 1984)	Kakamas
NMB 1370	NMB	2	ND	immature	10-34				Kakamas
NMB 1371	NMB	2	M	mature	>20				Kakamas

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
NMB 1372	NMB	2	F	mature	>20		210 ± 40 (Pta 2901)	(Morris, 1984)	Augrabies Falls
NMB 1375	NMB	3	A	mature	>20				Cango Caves
NMB 1376	NMB	3	A	mature	>20				Matjies River Rock Shelter
NMB 1377	NMB	2	[M]	mature	35-49	1900s			Kareeboom
NMB 1378	NMB	2	[M]	mature	>20				Renosterkop
NMB 1379	NMB	2	[M]	immature	20-34				Renosterkop
NMB 1380	NMB	3	[M]	mature	>20				Renosterkop
NMB 1381	NMB	2	ND	immature	10-19				Renosterkop
NMB 1382	NMB	2	M	mature	20-34				Renosterkop
NMB 1383	NMB	2	M	mature	20-49				Renosterkop
NMB 1384	NMB	2	F	mature	>50				Renosterkop
NMB 1385	NMB	3	[M]	mature	>20				Renosterkop
NMB 1386	NMB	2	M	mature	35-49				Kakamas
NMB 1387	NMB	2	[F]	mature	>20				Kakamas
NMB 1388	NMB	3	F	mature	>20				Kakamas
NMB 1389	NMB	3	M	mature	>20				Kakamas
NMB 1390	NMB	3	[M]	mature	>20				Kakamas
NMB 1391	NMB	2	A	mature	>20				Kakamas
NMB 1392	NMB	3	[F]	mature	>20				Kakamas
NMB 1393	NMB	3	ND	immature	6-19				Kakamas
NMB 1394	NMB	2	A	mature	>20				Kakamas
NMB 1395	NMB	2	F	immature	10-34				Kakamas
NMB 1396	NMB	3	A	mature	>20				Kakamas
NMB 1397	NMB	3	ND	immature	10-34				Kakamas
NMB 1398	NMB	2	ND	immature	10-19				Kakamas
NMB 1399	NMB	3	ND	mature	>20				Kakamas
NMB 1400	NMB	1	F	mature	35-49				Niekerkshoop
NMB 1401	NMB	3	A	mature	>20				Niekerkshoop
NMB 1402	NMB	2	M	mature	35-49				Niekerkshoop

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
NMB 1403 A and B	NMB	1	[M]	mature	35-49				Niekerkshoop
NMB 1404	NMB	2	M	mature	>20				Niekerkshoop
NMB 1405 A and B	NMB	2	M	mature	>20				Abrahamsdam
NMB 1406	NMB	2	[M]	mature	>20				Abrahamsdam
NMB 1407	NMB	2	M	mature	>20				Abrahamsdam
NMB 1408	NMB	2	M	mature	35-49				Abrahamsdam
NMB 1410 [A]	NMB	3	[F]	mature	>20				Kakamas
NMB 1410 [B]	NMB	3	A	mature	>20				Kakamas
NMB 1410 [C]	NMB	3	ND	mature	>20				Kakamas
NMB 1411	NMB	2	A	mature	>20				Upington
NMB 1412 A and B	NMB	1	M	mature	35-49				Upington
NMB 1414	NMB	3	[F]	mature	>20				Rooipad
NMB 1415	NMB	3	F	mature	>20				Rooipad
NMB 1416 A and B	NMB	1	M	mature	35-49				Rooipad
NMB 1417	NMB	3	ND	immature	2-9				Rooipad
NMB 1418	NMB	2	M	mature	>20				Rooipad
NMB 1419 [A]	NMB	3	ND	immature	2-19				Rooipad
NMB 1419 [B]	NMB	3	A	mature	>20				Rooipad
NMB 1420 A and B	NMB	1	F	mature	35-49				Augrabies Falls
NMB 1421	NMB	3	[M]	mature	>20				Augrabies Falls
NMB 1422	NMB	2	[M]	mature	>20				Augrabies Falls
NMB 1425	NMB	3	ND	immature	6-9				Augrabies Falls
NMB 1426	NMB	2	[F]	mature	35-49				Augrabies Falls
NMB 1427	NMB	2	[F]	mature	35-49		180 ± 70 (Pta 2908)	(Morris, 1984)	Augrabies Falls
NMB 1428	NMB	3	F	mature	>20				Augrabies Falls
NMB 1429	NMB	2	A	mature	>20				Paarden Eiland
NMB 1430	NMB	3	[F]	mature	>20				Paarden Eiland
NMB 1431	NMB	2	M	mature	>20				Paarden Eiland

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
NMB 1432	NMB	3	A	mature	>20				Swartdraai
NMB 1433	NMB	3	F	mature	>20				Goemansberg
NMB 1438	NMB	3	[F]	mature	>20				Matjies River Rock Shelter
NMB 1440	NMB	2	[M]	mature	>20		3040 ± 60 (Pta 6948)	(Sealy and Pfeiffer, 2000)	Matjies River Rock Shelter
NMB 1441	NMB	3	ND	mature	>20		9230 ± 160 (UCLA-1746B)	(Protsch and Oberholzer, 1975)	Matjies River Rock Shelter
NMB 1442	NMB	2	[M]	mature	>20		9230 ± 160 (UCLA-1746B)	(Protsch and Oberholzer, 1975)	Matjies River Rock Shelter
NMB 1443	NMB	3	A	mature	>20		9230 ± 160 (UCLA-1746B)	(Protsch and Oberholzer, 1975)	Matjies River Rock Shelter
NMB 1444	NMB	2	ND	immature	prenatal-1				Matjies River Rock Shelter
NMB 1445	NMB	3	[F]	mature	>20				Matjies River Rock Shelter
NMB 1446	NMB	3	[F]	mature	>20				Matjies River Rock Shelter
NMB 1447 [A]	NMB	2	A	immature	20-34				Matjies River Rock Shelter
NMB 1447 [B]	NMB	3	ND	immature	birth-5				Matjies River Rock Shelter
NMB 1448 [A]	NMB	3	[M]	mature	>20		7295 ± 32 (OxA-V-2064-52)	(Sealy, 2006; Sealy et al., 2006)	Matjies River Rock Shelter
NMB 1448 [B]	NMB	3	[M]	mature	>20				Matjies River Rock Shelter

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
NMB 1449 [A]	NMB	2	M	mature	>20				Matjies River Rock Shelter
NMB 1449 [B]	NMB	3	F	mature	>20				Matjies River Rock Shelter
NMB 1450	NMB	3	M	mature	>20				Matjies River Rock Shelter
NMB 1451	NMB	1	M	immature	10-34				Matjies River Rock Shelter
NMB 1563	NMB	3	[F]	mature	>20				Rooipan
NMB 1564	NMB	3	M	mature	>20				Tweeling
NMB 1565	NMB	3	A	mature	>20				
NMB 1566	NMB	3	F	mature	>20				Verkeerdevlei
NMB 1567	NMB	2	F	mature	35-49	late 1800s		(Peckmann, 2002)	Philippolis
NMB 1568	NMB	2	M	mature	35-49	late 1800s		(Peckmann, 2002)	Philippolis
NMB 1570	NMB	2	F	mature	35-49	late 1800s		(Peckmann, 2002)	Philippolis
NMB 1583	NMB	3	ND	mature	>20				Toorfontein
NMB 1590	NMB	3	ND	immature	6-19				Buispoort
NMB 1591	NMB	3	[F]	mature	>20				Buispoort
NMB 1592	NMB	3	M	mature	35-49				
NMB 1593	NMB	3	ND	mature	>20				Buispoort
NMB 1594	NMB	3	ND	mature	>20				Buispoort
NMB 1602 [A]	NMB	3	[M]	mature	>20				Matjies River Rock Shelter
NMB 1603	NMB	3	ND	immature	10-19				Matjies River Rock Shelter
NMB 1604	NMB	2	M	mature	35-49	late 1800s		(Peckmann, 2002)	Philippolis
NMB 1605	NMB	2	F	mature	35-49	late 1800s		(Peckmann, 2002)	Philippolis

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
NMB 1606 A and B	NMB	1	[M]	mature	>20	late 1800s		(Peckmann, 2002)	Philippolis
NMB 1607	NMB	2	F	mature	35-49				Bainsvlei
NMB 1608	NMB	2	[F]	mature	>20	late 1800s		(Peckmann, 2002)	Philippolis
NMB 1609	NMB	2	ND	immature	10-19	late 1800s		(Peckmann, 2002)	Philippolis
NMB 1610	NMB	2	F	mature	>20	late 1800s		(Peckmann, 2002)	Philippolis
NMB 1611	NMB	3	[F]	mature	>20	late 1800s		(Peckmann, 2002)	Philippolis
NMB 1612	NMB	2	[F]	mature	>20	late 1800s		(Peckmann, 2002)	Philippolis
NMB 1613	NMB	2	F	mature	35-49	late 1800s		(Peckmann, 2002)	Philippolis
NMB 1616	NMB	3	[M]	mature	>20				Brandfort
NMB 1617 A and B	NMB	1	M	mature	35-49	19th century		(Peckmann, 2002)	Damfontein
NMB 1618 A and B	NMB	1	F	mature	>50	19th century		(Peckmann, 2002)	Damfontein
NMB 1619 A and B	NMB	1	M	mature	35-49	19th century		(Peckmann, 2002)	Damfontein
NMB 1621 A and B	NMB	1	F	mature	35-49	19th century		(Peckmann, 2002)	Damfontein
NMB 1622 A and B	NMB	2	ND	immature	6-19	19th century		(Peckmann, 2002)	Damfontein
NMB 1624	NMB	1	ND	immature	2-5	19th century		(Peckmann, 2002)	Damfontein
NMB 1625	NMB	3	ND	immature	birth-1	19th century		(Peckmann, 2002)	Damfontein
NMB 1627	NMB	3	ND	immature	birth-5	19th century		(Peckmann, 2002)	Damfontein

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
NMB 1628	NMB	2	ND	immature	prenatal-1	19th century		(Peckmann, 2002)	Damfontein
NMB 1629 [A]	NMB	3	ND	immature	birth-1	19th century		(Peckmann, 2002)	Damfontein
NMB 1630	NMB	3	ND	immature	2-9	19th century		(Peckmann, 2002)	Damfontein
NMB 1631	NMB	3	ND	immature	prenatal-1	19th century		(Peckmann, 2002)	Damfontein
NMB 1632	NMB	3	ND	immature	birth-5	19th century		(Peckmann, 2002)	Damfontein
NMB 1633	NMB	3	M	mature	>20				Donkerpoort
NMB 1634	NMB	3	ND	immature	10-19	19th century		(Peckmann, 2002)	Damfontein
NMB 1635	NMB	3	ND	immature	10-34				Sannaspos
NMB 1636	NMB	3	A	mature	>20				Soutpan
NMB 1637	NMB	3	ND	immature	birth-9				Avalon, Rietrivier
NMB 1638 A and B	NMB	2	M	mature	35-49				New Rail Bridge, Bethulie
NMB 1639 A and B	NMB	1	F	mature	20-34		2590 ± 60 (Pta 6965)	(Sealy and Pfeiffer, 2000)	Robberg
NMB 1640	NMB	1	[F]	immature	10-34		4120 ± 60 (Pta 6983)	(Sealy and Pfeiffer, 2000)	Robberg
NMB 1641	NMB	2	ND	immature	2-5				Robberg

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
NMB 1642	NMB	2	ND	immature	2-5				Robberg
NMB 1643	NMB	2	ND	immature	birth-1				Robberg
NMB 1644	NMB	2	ND	immature	birth-1				Robberg
NMB 1654	NMB	3	F	mature	>20				Avalon, Rietrivier
NMB 1655 A and B	NMB	1	F	mature	35-49				Vaalbank
NMB 1656	NMB	3	F	mature	>20				Vlakfontein
NMB 1657 A and B	NMB	1	F	mature	35-49				Vlakfontein
NMB 1658	NMB	1	A	mature	35-49				Vlakfontein
NMB 1659	NMB	1	M	mature	35-49				Vlakfontein
NMB 1660	NMB	3	[F]	mature	>20				Vlakfontein
NMB 1661	NMB	2	ND	immature	birth-5				Vlakfontein
NMB 1662	NMB	3	ND	immature	10-19				Vlakfontein
NMB 1681	NMB	3	[F]	mature	>20				Bloemfontein
NMB 1688	NMB	3	[F]	mature	>20				Bergfontein
NMB 1689	NMB	2	[M]	immature	10-34				Vrede
NMB 1690	NMB	3	[F]	mature	>20				
NMB 1691	NMB	2	ND	immature	birth-1				Bergfontein
NMB 1700	NMB	3	ND	mature	>20	late 1800s			Philippolis
NMB 1701	NMB	2	M	immature	10-34	late 1800s		(Peckmann, 2002)	Philippolis
NMB 1702	NMB	2	F	immature	10-34	late 1800s		(Peckmann, 2002)	Philippolis
NMB 1703	NMB	2	F	mature	>50	late 1800s		(Peckmann, 2002)	Philippolis
NMB 1704	NMB	1	[F]	immature	10-34		760 ± 50 (Pta 6963)	(Sealy and Pfeiffer, 2000)	Plettenberg Bay

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
NMB 1705	NMB	2	F	mature	35-49		2780 ± 60 (A: Pta 6964; B: Pta 6978)	(Sealy and Pfeiffer, 2000)	Plettenberg Bay
NMB 1706	NMB	3	A	mature	>20				Plettenberg Bay
NMB 1707	NMB	3	[M]	mature	>20		1394 ± 24 (OxA-V-2064-53)	(Sealy, 2006; Sealy et al., 2006)	Plettenberg Bay
NMB 1708	NMB	2	M	mature	35-49				Odendaalrus
NMB 1709	NMB	3	ND	immature	birth-5				Bergfontein
NMB 1711	NMB	3	ND	mature	>20				Plettenberg Bay
NMB 1713 (A) [A]	NMB	3	A	mature	>20				Monastery Mine
NMB 1715	NMB	3	A	mature	>20				Winburg
NMB 1717	NMB	3	[F]	mature	>20				
NMB 1767	NMB	2	[M]	mature	>50				Sterkfontein
NMB 1823	NMB	3	F	mature	35-49				Prospect
NMB 1825	NMB	3	M	mature	>20				
NMB 1826	NMB	3	[F]	mature	>20				Gordons Bay
NMB 1827	NMB	3	[F]	mature	>20		2815 ± 40 (GrA-23229)	(Stynder et al., 2007a)	
NMB 1829	NMB	3	A	mature	>20				Alice
NMB 1830	NMB	3	M	mature	>20				Somerset East
NMB 1831	NMB	3	A	mature	>20				Salisbury Island
NMB 1832	NMB	3	[M]	mature	>20				
NMB 1833	NMB	3	[M]	mature	20-34				Alice
NMB 1920 A and B	NMB	2	M	mature	35-49	19th century		(Peckmann, 2002)	Damfontein
NMB ? Matjies River	NMB	3	[M]	mature	>20				Matjies River Rock Shelter
NMB 211? [A]	NMB	2	M	mature	35-49				Matjies River Rock Shelter

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
NMB 211? [B]	NMB	3	ND	immature	birth-5				Matjies River Rock Shelter
NMB 211? [C]	NMB	3	ND	immature	2-5				Matjies River Rock Shelter
NMB 5v7-8/10 / 136	NMB	3	ND	immature	birth-1				Matjies River Rock Shelter
NMB Matjies River Skeleton #1	NMB	2	[F]	immature	10-34		2280 ± 60 (Pta 6952)	(Sealy and Pfeiffer, 2000)	Matjies River Rock Shelter
NMB MR 10 [A]	NMB	3	[F]	mature	>20				Matjies River Rock Shelter
NMB MR 10 [B]	NMB	3	[M]	mature	>20				Matjies River Rock Shelter
NMB MR 10 [C]	NMB	3	[M]	mature	>20				Matjies River Rock Shelter
NMB MR A1	NMB	3	ND	immature	birth-19				Matjies River Rock Shelter
NMB MR Sv0-12/12	NMB	3	[M]	mature	>20				Matjies River Rock Shelter
NMB MR4 (8MZ/96)	NMB	3	A	mature	>20				Matjies River Rock Shelter
NMB MRW WSK 3	NMB	3	ND	mature	>20				Matjies River Rock Shelter
NMB MRWSS2 / WSK2	NMB	3	A	mature	>20				Matjies River Rock Shelter
NMB MSK 2	NMB	2	[F]	mature	35-49		2200 ± 50 (Pta 6944)	(Sealy and Pfeiffer, 2000)	Matjies River Rock Shelter
NMB MSK 4	NMB	3	ND	mature	>20				Matjies River Rock Shelter

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
NMB MSK 3 MR	NMB	3	A	mature	>20				Matjies River Rock Shelter
NMB MSK 5 (MR)	NMB	2	ND	mature	>20		3277 ± 31 (OxA-11939)	(Sealy, 2006; Sealy et al., 2006)	Matjies River Rock Shelter
NMB no accession no. in box with 1715 and 1717	NMB	3	A	mature	>20				
NMB S5-2/13	NMB	3	[M]	mature	>20				Matjies River Rock Shelter
NMB SI Grave II (MR) [A]	NMB	2	ND	immature	6-9				Matjies River Rock Shelter
NMB SI grave II (MR) [B]	NMB	2	ND	immature	prenatal-1				Matjies River Rock Shelter
NMB skeleton #4: Top Mytilus MR	NMB	2	ND	immature	10-19				Matjies River Rock Shelter
NMB skeleton #5	NMB	2	[M]	mature	>20				Matjies River Rock Shelter
NMB Skeleton No.1 Baby MR	NMB	2	ND	immature	prenatal-1				Matjies River Rock Shelter
NMB SS1 MR	NMB	2	M	mature	35-49				Matjies River Rock Shelter
NMB SS2	NMB	2	F	immature	10-34		5370 ± 70 (Pta 6976)	(Sealy and Pfeiffer, 2000)	Matjies River Rock Shelter

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
NMB SS3 / 17M2/96	NMB	2	M	mature	35-49		5390 ± 70 (Pta 6975)	(Sealy and Pfeiffer, 2000)	Matjies River Rock Shelter
NMB SS3 MR	NMB	3	[M]	mature	>20				Matjies River Rock Shelter
NMB SS4 MR	NMB	3	ND	mature	>20				Matjies River Rock Shelter
NMB SS5 MR	NMB	3	ND	immature	birth-1				Matjies River Rock Shelter
NMB unmarked MR / 150.1 / 11M2/96 / ? MR#4	NMB	3	ND	immature	2-19				Matjies River Rock Shelter
NMB Unmarked MR / 151.1 / 25M2/96	NMB	3	[M]	mature	>20				Matjies River Rock Shelter
NMB Unmarked MR? Wilton grave no. 4 Sii-iv (A)	NMB	2	F	immature	10-34		271 ± 20 (OxA-11965)	(Sealy, 2006; Sealy et al., 2006)	Matjies River Rock Shelter
NMB Unmarked MR? Wilton grave no. 4 Sii-iv (B)	NMB	3	ND	immature	6-19				Matjies River Rock Shelter
NMB UV (unaccessioned in box with 80-82)	NMB	3	A	mature	>20				
NMB WSK 2 / 162.1	NMB	3	ND	mature	>20				Matjies River Rock Shelter
NMB WSK1 (2M11/95)	NMB	3	ND	immature	birth-1				Matjies River Rock Shelter
NMB WSK1 (36M2/96)	NMB	2	ND	immature	prenatal-1				Matjies River Rock Shelter

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
NMB WSK3 MR / 41M2/96	NMB	3	[M]	mature	>20				Matjies River Rock Shelter
SAM-AP 1	SAM	3	[F]	mature	>20				
SAM-AP 2	SAM	3	[F]	mature	>20				Somerset West
SAM-AP 4	SAM	3	A	mature	>20				
SAM-AP 6	SAM	3	[M]	mature	>20				Port Nolloth
SAM-AP 7	SAM	3	A	mature	>20				Knysna
SAM-AP 10	SAM	3	[M]	mature	>20				Melkbosstrand
SAM-AP 11	SAM	3	[M]	mature	>20				Kowie River
SAM-AP 15	SAM	3	[F]	mature	>20				Paternoster
SAM-AP 17	SAM	3	A	mature	>20				
SAM-AP 18	SAM	3	M	mature	>20				
SAM-AP 20	SAM	3	F	mature	>20				
SAM-AP 24	SAM	3	F	mature	>20				Cape Peninsula
SAM-AP 26	SAM	3	[F]	mature	>20		270 ± 100 (OxA-458)	(Sealy et al., 2000)	Clanwilliam
SAM-AP 27	SAM	3	M	mature	>20		250 ± 90 (OxA-452)	(Sealy et al., 2000)	Clanwilliam
SAM-AP 30	SAM	3	[F]	mature	>20				Barbierskraal
SAM-AP 32	SAM	1	[F]	mature	>50		3754 ± 35 (OxA-V-2055-47)	(Stynder et al., 2007a)	Bloukrans River
SAM-AP 33 A	SAM	2	M	mature	>50				
SAM-AP 34	SAM	1	A	mature	35-49		2310 ± 25 (Pta 6599)	(Sealy and Pfeiffer, 2000)	Touws River Mouth

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
SAM-AP 36	SAM	3	[M]	mature	>20				Bloubergstrand
SAM-AP 37	SAM	3	ND	mature	>20		6120 ± 70 (Pta 4353)	(Sealy and van der Merwe, 1988)	Bloubergstrand
SAM-AP 39 A	SAM	3	[F]	mature	>20				Gordons Bay
SAM-AP 39 B	SAM	3	ND	ND	ND				Gordons Bay
SAM-AP 39	SAM	3	ND	immature	birth-19		2448 ± 29 (OxA-V-2055-43)	(Stynder et al., 2007a)	Gordons Bay
SAM-AP 42	SAM	3	A	mature	>20				Lake District
SAM-AP 43	SAM	3	[M]	mature	>20				Ezeljagt
SAM-AP 47	SAM	3	[M]	mature	>20				Hawston
SAM-AP 48	SAM	3	A	mature	>20				Hawston
SAM-AP 82 Box 1 and 2	SAM	1	ND	immature	birth-5				
SAM-AP 168 G	SAM	3	M	mature	>20				
SAM-AP 267 G	SAM	3	[M]	mature	>20				
SAM-AP 278 G	SAM	3	[M]	mature	>20		2158 ± 28 (OxA-V-2065-43)	(Stynder et al., 2007a)	Klein Brakrivier
SAM-AP 320 G	SAM	3	A	mature	>20		1707 ± 27 (OxA-V-2056-24)	(Stynder et al., 2007a)	Klein Brakrivier
SAM-AP 354 G	SAM	3	A	mature	>20				Riet River, Bethany
SAM-AP 1128/9	SAM	2	ND	immature	2-19				Robberg?
SAM-AP 1128	SAM	3	[F]	mature	>20		3156 ± 33 (OxA-V-2055-49)	(Stynder et al., 2007a)	Robberg
SAM-AP 1129	SAM	3	[M]	mature	>20		5379 ± 34 (OxA-V-2053-47)	(Sealy, 2006)	Robberg
SAM-AP 1130	SAM	3	ND	immature	2-9				Robberg

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
SAM-AP 1131	SAM	1	F	mature	>50		4030 ± 110 (TO-8401)	(Pfeiffer and Sealy, 2006)	Robberg
SAM-AP 1132	SAM	1	ND	immature	2-9				Robberg
SAM-AP 1142	SAM	3	[F]	mature	>20		2090 ± 27 (OxA-V-2056-32)	(Stynder et al., 2007a)	Somerset West
SAM-AP 1143	SAM	3	[F]	mature	>20				Port Elizabeth
SAM-AP 1144	SAM	3	[M]	mature	>20				Pearston?
SAM-AP 1145	SAM	1	A	mature	>50		3210 ± 70 (Pta 2284)	(Sealy and Pfeiffer, 2000)	Robberg
SAM-AP 1146	SAM	1	[M]	mature	35-49		2240 ± 20 (Pta-6646)	(Sealy and Pfeiffer, 2000)	Robberg
SAM-AP 1149	SAM	2	ND	mature	>20		3970 ± 50 (Pta 4690)	(Sealy, 1989)	Bloubergstrand
SAM-AP 1150	SAM	3	F	mature	>20				Bloubergstrand
SAM-AP 1152	SAM	3	[M]	mature	>20				Cape Point
SAM-AP 1153	SAM	2	F	mature	35-49		440 ± 50 (Pta-4197)	(Sealy and van der Merwe, 1988)	Cape Peninsula
SAM-AP 1154	SAM	3	ND	immature	6-9				
SAM-AP 1156	SAM	3	ND	mature	>20				Pletternberg Bay

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
SAM-AP 1157	SAM	1	F	mature	35-49		2420 ± 60 (Pta 4217)	(Sealy and van der Merwe, 1988)	Bloubergstrand
SAM-AP 1158	SAM	3	[M]	mature	>20				
SAM-AP 1161	SAM	3	[F]	mature	>20				Cold Stream
SAM-AP 1162	SAM	3	A	mature	>20				Cold Stream
SAM-AP 1239	SAM	3	ND	immature	birth-5				Barrydale
SAM-AP 1240	SAM	3	F	mature	>20				Richtersveld
SAM-AP 1247 A	SAM	1	F	mature	>50		1180 ± 50 (Pta 4281)	(Sealy and van der Merwe, 1988)	Bloubergstrand
SAM-AP 1247 B	SAM	3	[M]	mature	>35		1280 ± 50 (Pta 4341)	(Sealy and van der Merwe, 1988)	Bloubergstrand
SAM-AP 1254	SAM	2	M	mature	>20				Sakrivier
SAM-AP 1257	SAM	3	A	mature	>20				Amsterdam Battery, Cape Town
SAM-AP 1260	SAM	3	M	mature	>20		1137 ± 27 (OxA-V-2066-28)	(Stynder et al., 2007a)	Oudtshoorn
SAM-AP 1267	SAM	1	[M]	mature	>20				Jakkelsfontein
SAM-AP 1268	SAM	1	M	mature	20-49				Orange River, Namaqualand
SAM-AP 1269	SAM	1	M	mature	35-49				
SAM-AP 1270	SAM	1	M	mature	35-49				
SAM-AP 1271	SAM	2	F	mature	>50				Namaqualand
SAM-AP 1272	SAM	1	F	mature	35-49				

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
SAM-AP 1273	SAM	1	ND	immature	10-19				Jakkelswater, Namaqualand
SAM-AP 1274	SAM	1	ND	immature	10-19				Matjies River, Northern Cape
SAM-AP 1275	SAM	1	ND	immature	2-9				Matjies River, Northern Cape
SAM-AP 1276	SAM	1	[M]	mature	>20				
SAM-AP 1277	SAM	1	M	mature	20-34				
SAM-AP 1278	SAM	2	F	mature	>50				
SAM-AP 1440	SAM	2	M	mature	>20				
SAM-AP 1441	SAM	1	M	mature	>50		2170 ± 60 (Pta 4201)	(Sealy and van der Merwe, 1988)	Melkbosstrand
SAM-AP 1442	SAM	3	F	mature	>20				Gordons Bay
SAM-AP 1443	SAM	1	M	mature	>20		2050 ± 50 (Pta 2309)	(Sealy and van der Merwe, 1988)	Gordons Bay
SAM-AP 1444	SAM	1	[M]	mature	35-49		1290 ± 50 (Pta 4398)	(Sealy and van der Merwe, 1988)	Gordons Bay
SAM-AP 1445 [A]	SAM	3	ND	mature	>20				Mossel Bay
SAM-AP 1445 [B]	SAM	3	ND	mature	>20				Mossel Bay
SAM-AP 1446	SAM	1	A	mature	35-49		740 ± 30 (Pta 9085)	(Stynder et al., 2007a)	Port Nolloth
SAM-AP 1448	SAM	1	ND	immature	2-9				Knysna
SAM-AP 1449	SAM	2	[F]	mature	20-34		2230 ± 100 (OxA 453)	(Sealy et al., 1986)	Cederberg

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
SAM-AP 1450	SAM	1	ND	immature	6-9				Knysna
SAM-AP 1451	SAM	3	[F]	mature	>20				Knysna
SAM-AP 1452	SAM	3	M	mature	>20				Knysna
SAM-AP 1453	SAM	3	ND	immature	2-9				
SAM-AP 1454	SAM	1	A	immature	10-34				Richtersveld
SAM-AP 1455	SAM	1	M	mature	35-49				Orange River Mouth
SAM-AP 1456	SAM	3	ND	mature	>20				
SAM-AP 1457	SAM	1	F	mature	>50		910 ± 35 (Pta-2149)	(Stynder, 2009)	Klein Brakrivier
SAM-AP 1458	SAM	3	M	mature	>20				Mossel Bay
SAM-AP 1459	SAM	3	[F]	mature	>20				Outeniqua
SAM-AP 1460	SAM	3	ND	immature	6-19				Gordons Bay
SAM-AP 1462	SAM	1	[M]	mature	>20				Kruidfontein?
SAM-AP 1471	SAM	3	M	mature	>20				
SAM-AP 1473	SAM	1	[F]	mature	>50		1880 ± 60 (Pta 8773)	(Stynder et al., 2007a)	Onrus
SAM-AP 1860	SAM	2	ND	immature	2-19				Namaqualand
SAM-AP 1861	SAM	2	ND	mature	>20				
SAM-AP 1862 [A]	SAM	3	ND	immature	6-19				Namaqualand
SAM-AP 1862 [B]	SAM	2	[M]	mature	>20				Namaqualand
SAM-AP 1863	SAM	2	ND	mature	20-34		800 ± 50 (Pta 4708)	(Sealy, 1989)	Cape Point
SAM-AP 1864	SAM	3	[F]	mature	>20				
SAM-AP 1865	SAM	3	A	mature	>20				
SAM-AP 1871	SAM	1	A	mature	>50		3310 ± 60 (Pta 2273)	(Sealy and Pfeiffer, 2000)	Robberg
SAM-AP 1872 [A]	SAM	2	ND	immature	prenatal-5				
SAM-AP 1872 [B]	SAM	2	ND	immature	prenatal-5				
SAM-AP 1873	SAM	2	ND	immature	2-5				Robberg

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
SAM-AP 1874	SAM	2	ND	immature	prenatal-5				
SAM-AP 1875	SAM	2	ND	immature	prenatal-1				
SAM-AP 1876 Bis	SAM	2	ND	immature	prenatal-1				Robberg
SAM-AP 1877	SAM	3	M	mature	>20				Paardeberg
SAM-AP 1878 A	SAM	1	M	mature	>35		2170 ± 20 (Pta 6592)	(Sealy and Pfeiffer, 2000)	Robberg
SAM-AP 1878 B	SAM	2	F	mature	>50		2620 ± 35 (Pta 2145)	(Sealy and Pfeiffer, 2000)	Robberg
SAM-AP 1879	SAM	1	[M]	mature	35-49		3440 ± 60 (Pta 2283)	(Sealy and Pfeiffer, 2000)	Robberg
SAM-AP 1880	SAM	3	[M]	mature	>20				
SAM-AP 1881	SAM	3	ND	immature	6-19				Ottoshoop
SAM-AP 1885	SAM	2	[M]	mature	>50				Kruidfontein
SAM-AP 1886	SAM	3	[M]	mature	>20				Kruidfontein
SAM-AP 1887	SAM	1	ND	immature	2-5				Kruidfontein
SAM-AP 1888	SAM	1	[M]	mature	>20				Kruidfontein
SAM-AP 1889	SAM	2	[M]	mature	>50		2310 ± 50 (Pta 6594)	(Sealy and Pfeiffer, 2000)	Robberg
SAM-AP 1891	SAM	2	ND	immature	birth-5				
SAM-AP 1892	SAM	2	ND	immature	2-9				
SAM-AP 1893	SAM	2	M	mature	35-49		2360 ± 20 (Pta 6613)	(Sealy and Pfeiffer, 2000)	Robberg
SAM-AP 1894	SAM	3	A	mature	>20		3511 ± 30 (OxA-V-2053-43)	(Sealy, 2006)	Robberg
SAM-AP 1895	SAM	3	[M]	mature	>20				

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
SAM-AP 1898	SAM	3	ND	immature	2-19				Groot River
SAM-AP 3021	SAM	2	[F]	mature	35-49		4030 ± 60 (Pta 6595)	(Sealy and Pfeiffer, 2000)	Robberg
SAM-AP 3022	SAM	3	ND	immature	2-9				
SAM-AP 3023	SAM	2	ND	immature	2-19				
SAM-AP 3024	SAM	1	ND	immature	2-5				Robberg
SAM-AP 3025	SAM	1	ND	immature	2-9				
SAM-AP 3026 [A]	SAM	2	ND	immature	10-19		3980 ± 60 (Pta 7925)	(Muller, 2002)	Robberg
SAM-AP 3026 [B]	SAM	3	ND	immature	10-19				Robberg
SAM-AP 3027	SAM	1	ND	immature	10-19				Robberg
SAM-AP 3028	SAM	2	ND	immature	birth-5				
SAM-AP 3029	SAM	2	ND	immature	prenatal-5				
SAM-AP 3030	SAM	3	ND	mature	>20		2570 ± 50 (Pta 7940)	(Sealy, 2006)	Robberg
SAM-AP 3031	SAM	2	ND	immature	prenatal-5				
SAM-AP 3033	SAM	2	ND	immature	birth-1				
SAM-AP 3034 A and B	SAM	3	ND	immature	2-5				Robberg
SAM-AP 3042	SAM	2	ND	immature	10-19				
SAM-AP 3043	SAM	3	ND	mature	>20				Port St Johns
SAM-AP 3044	SAM	3	[M]	mature	>20				Bloubergstrand
SAM-AP 3045	SAM	3	[M]	mature	>20				Cape Town
SAM-AP 3046	SAM	3	ND	mature	>20				Cape Town
SAM-AP 3047	SAM	3	ND	mature	>20				
SAM-AP 3048	SAM	3	[M]	mature	>20				Groot River
SAM-AP 3049	SAM	3	F	mature	>20				Groot River
SAM-AP 3052	SAM	3	M	mature	>20				

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
SAM-AP 3053	SAM	2	[M]	mature	>20		1990 ± 50 (Pta 4411)	(Sealy and van der Merwe, 1988)	Strand
SAM-AP 3054	SAM	3	[M]	mature	>20				Vleesbaai
SAM-AP 3057	SAM	3	ND	immature	2-9				
SAM-AP 3058	SAM	3	ND	immature	2-9				Kruidfontein
SAM-AP 3059 [A]	SAM	3	ND	immature	2-9				Kruidfontein
SAM-AP 3059 [B]	SAM	3	A	mature	>20				Kruidfontein
SAM-AP 3457	SAM	1	[F]	mature	20-34				Kruidfontein
SAM-AP 3458	SAM	2	[F]	mature	>20				Bloubergstrand
SAM-AP 3462	SAM	1	M	mature	20-34				
SAM-AP 3495	SAM	3	M	mature	>20				
SAM-AP 3691	SAM	1	ND	immature	6-19				Upington
SAM-AP 3692	SAM	3	[F]	mature	>20				
SAM-AP 3693	SAM	3	ND	immature	2-9				
SAM-AP 3695	SAM	3	M	mature	>20				
SAM-AP 3696	SAM	2	[F]	mature	10-34				Rooiels Cave
SAM-AP 3697	SAM	2	M	mature	35-49				Rooiels Cave
SAM-AP 3698	SAM	2	ND	immature	6-19				
SAM-AP 3699	SAM	3	A	mature	>20				
SAM-AP 3700	SAM	3	[M]	mature	>20				Welgemoed Farm
SAM-AP 3701 bis	SAM	3	F	mature	>20				Knysna
SAM-AP 3701	SAM	3	M	mature	>20				Knysna
SAM-AP 3702	SAM	3	[M]	mature	>20				
SAM-AP 3703	SAM	1	F	mature	>20				Graaff Reinet
SAM-AP 3704	SAM	3	[F]	mature	>20				Rooiels Cave
SAM-AP 3729	SAM	3	A	mature	>20				
SAM-AP 3734	SAM	3	[M]	mature	>20				
SAM-AP 3735	SAM	3	ND	immature	2-9				
SAM-AP 3737	SAM	2	ND	immature	6-19		1370 ± 45 (Pta 4217)	(Sealy and van der Merwe, 1988)	Hout Bay
SAM-AP 3738	SAM	1	M	mature	>20				Greenpoint

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
SAM-AP 3779	SAM	3	[F]	mature	>20				
SAM-AP 4178	SAM	3	[F]	mature	>20				Gouritz
SAM-AP 4179	SAM	3	[F]	mature	>20				Thys Bay
SAM-AP 4180	SAM	3	A	mature	>20		688 ± 27 (OxA-V-2056-23)	(Stynder et al., 2007a)	Thys Bay
SAM-AP 4181	SAM	3	ND	mature	>20		688 ± 27 (OxA-V-2056-23)	(Stynder et al., 2007a)	
SAM-AP 4182	SAM	3	[M]	mature	>20		6811 ± 36 (OxA-V-2056-26)	(Stynder et al., 2007a)	Cold Stream
SAM-AP 4183	SAM	3	F	mature	>20				van der Walt's Cave
SAM-AP 4184	SAM	3	ND	mature	>20				
SAM-AP 4186	SAM	3	M	mature	>20				Stormberg
SAM-AP 4187	SAM	3	ND	immature	birth-19				
SAM-AP 4188 A and B	SAM	1	A	mature	35-49				Kruidfontein
SAM-AP 4190	SAM	3	F	mature	>20				
SAM-AP 4192	SAM	3	[M]	mature	>20				Melkbosstrand
SAM-AP 4193	SAM	3	F	mature	>20				
SAM-AP 4194	SAM	3	ND	immature	2-9				
SAM-AP 4195	SAM	1	[M]	mature	20-49				
SAM-AP 4196	SAM	1	[M]	mature	20-34				
SAM-AP 4197	SAM	1	ND	immature	6-19				
SAM-AP 4198	SAM	1	M	mature	35-49				
SAM-AP 4199	SAM	1	M	mature	20-49				
SAM-AP 4201	SAM	1	A	immature	10-34				
SAM-AP 4202	SAM	3	F	mature	>20		2673 ± 29 (OxA-V-2056-25)	(Stynder et al., 2007a)	
SAM-AP 4204	SAM	3	A	mature	>20				Great Brak River
SAM-AP 4205	SAM	3	ND	mature	>20				

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
SAM-AP 4206 BIS	SAM	2	[F]	mature	>50				van der Walt's Cave
SAM-AP 4206	SAM	1	[F]	mature	>50				van der Walt's Cave
SAM-AP 4207	SAM	2	ND	immature	birth-5				Cold Stream
SAM-AP 4208 A	SAM	2	[M]	mature	>50		9720 ± 100 (Pta 6615)	(Sealy and Pfeiffer, 2000)	Cold Stream
SAM-AP 4209	SAM	2	ND	mature	>20				
SAM-AP 4210	SAM	2	A	immature	10-19		3760 ± 60 (Pta 6654)	(Sealy and Pfeiffer, 2000)	Cold Stream
SAM-AP 4211	SAM	2	ND	immature	2-19				Cold Stream
SAM-AP 4212	SAM	2	[F]	mature	>50		1710 ± 70 (Pta 6642)	(Sealy and Pfeiffer, 2000)	Cold Stream
SAM-AP 4292	SAM	2	ND	mature	>50		2120 ± 50 (Pta 6617)	(Sealy and Pfeiffer, 2000)	van der Walt's Cave
SAM-AP 4293	SAM	3	[M]	mature	>50				van der Walt's Cave
SAM-AP 4294	SAM	3	ND	ND	ND				van der Walt's Cave
SAM-AP 4295	SAM	3	ND	mature	>20				van der Walt's Cave
SAM-AP 4296	SAM	3	[M]	mature	>20				van der Walt's Cave
SAM-AP 4297 [A]	SAM	3	ND	mature	>20				Kommetjie
SAM-AP 4297 [B]	SAM	3	ND	immature	2-9				Kommetjie
SAM-AP 4298	SAM	3	[F]	mature	>20		3380 ± 33 (OxA-V-2055-41)	(Stynder et al., 2007a)	Kommetjie
SAM-AP 4299	SAM	3	A	mature	>20		2294 ± 29 (OxA-V-2065-46)	(Stynder et al., 2007a)	Noordhoek
SAM-AP 4300	SAM	3	[M]	mature	>20		2304 ± 29 (OxA-V-2065-37)	(Stynder et al., 2007a)	Noordhoek
SAM-AP 4301*	SAM	3	[F]	mature	>20		2250 ± 30 (OxA-V-2055-40)	(Stynder et al., 2007a)	Noordhoek

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
SAM-AP 4301	SAM	3	ND	mature	>20				North Bay; Saldanha
SAM-AP 4302	SAM	3	[F]	mature	>20				Noordhoek
SAM-AP 4303	SAM	3	[M]	mature	>20				Noordhoek
SAM-AP 4304 [A]	SAM	3	ND	mature	>20		2220 ± 50 (Pta 4656)	(Sealy and van der Merwe, 1988)	Noordhoek
SAM-AP 4304 [B]	SAM	2	ND	immature	2-19		2070 ± 50 (Pta 4391)	(Sealy and van der Merwe, 1988)	Noordhoek
SAM-AP 4305	SAM	2	[M]	mature	35-49		2100 ± 45 (Pta 4660)	(Sealy, 1989)	Noordhoek
SAM-AP 4306	SAM	2	ND	mature	>20		2210 ± 50 (Pta 4350)	(Sealy and van der Merwe, 1988)	Noordhoek
SAM-AP 4308	SAM	2	[M]	mature	>20		2170 ± 60 (Pta 4404)	(Sealy and van der Merwe, 1988)	Noordhoek
SAM-AP 4309 [A]	SAM	2	[F]	mature	>20		2120 ± 45 (Pta 4385)	(Sealy and van der Merwe, 1988)	Noordhoek
SAM-AP 4309 [B]	SAM	3	ND	immature	birth-5				Noordhoek
SAM-AP 4310	SAM	3	[M]	mature	>20				
SAM-AP 4312	SAM	2	[F]	mature	>20		2260 ± 170 (Pta 2164)	(Morris, 1992a)	Mossel Bay?
SAM-AP 4313	SAM	3	ND	immature	birth-5				Mossel Bay
SAM-AP 4314	SAM	3	A	mature	>20		1319 ± 25 (OxA-V-2066-26)	(Stynder et al., 2007a)	
SAM-AP 4315	SAM	3	[F]	mature	>20				Cold Stream
SAM-AP 4316	SAM	1	ND	immature	10-19	1866		(Botha and Steyn, 2015; Drennan, 1929; Peckmann, 2002; Slome, 1929)	Colesberg

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
SAM-AP 4317	SAM	1	[M]	mature	35-49	1866		(Botha and Steyn, 2015; Drennan, 1929; Peckmann, 2002; Slome, 1929)	Colesberg
SAM-AP 4320	SAM	1	M	mature	35-49	1866		(Botha and Steyn, 2015; Drennan, 1929; Peckmann, 2002; Slome, 1929)	Colesberg
SAM-AP 4322	SAM	1	[M]	mature	>20	1866		(Botha and Steyn, 2015; Drennan, 1929; Peckmann, 2002; Slome, 1929)	Colesberg
SAM-AP 4326	SAM	2	[F]	mature	35-49	1866		(Botha and Steyn, 2015; Drennan, 1929; Peckmann, 2002; Slome, 1929)	Colesberg
SAM-AP 4329	SAM	1	[M]	mature	35-49	1866		(Botha and Steyn, 2015; Drennan, 1929; Peckmann, 2002; Slome, 1929)	Colesberg
SAM-AP 4330	SAM	1	[F]	mature	>20	1866		(Botha and Steyn, 2015; Drennan, 1929; Peckmann, 2002; Slome, 1929)	Colesberg

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
SAM-AP 4331	SAM	1	[M]	mature	35-49	1866		(Botha and Steyn, 2015; Drennan, 1929; Peckmann, 2002; Slome, 1929)	Colesberg
SAM-AP 4488	SAM	1	[F]	mature	35-49	1866		(Botha and Steyn, 2015; Drennan, 1929; Peckmann, 2002; Slome, 1929)	Colesberg
SAM-AP 4489	SAM	1	A	mature	>50	1866		(Botha and Steyn, 2015; Drennan, 1929; Peckmann, 2002; Slome, 1929)	Colesberg
SAM-AP 4490	SAM	1	M	mature	20-34	1866		(Botha and Steyn, 2015; Drennan, 1929; Peckmann, 2002; Slome, 1929)	Colesberg
SAM-AP 4491	SAM	1	A	mature	35-49	1866		(Botha and Steyn, 2015; Drennan, 1929; Peckmann, 2002; Slome, 1929)	Colesberg
SAM-AP 4493	SAM	1	M	mature	>20	1866		Sloan, 1929; Drennan, 1929; Peckmann, 2003; Botha and Steyn, 2015	Colesberg

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
SAM-AP 4499	SAM	1	[F]	mature	>20	1866		(Botha and Steyn, 2015; Drennan, 1929; Peckmann, 2002; Slome, 1929)	Colesberg
SAM-AP 4501	SAM	2	[M]	mature	>20	1866		(Botha and Steyn, 2015; Drennan, 1929; Peckmann, 2002; Slome, 1929)	Colesberg
SAM-AP 4502	SAM	1	[M]	mature	35-49	1866		(Botha and Steyn, 2015; Drennan, 1929; Peckmann, 2002; Slome, 1929)	Colesberg
SAM-AP 4504	SAM	1	M	mature	35-49	1866		(Botha and Steyn, 2015; Drennan, 1929; Peckmann, 2002; Slome, 1929)	Colesberg
SAM-AP 4505	SAM	1	F	mature	35-49	1866		(Botha and Steyn, 2015; Drennan, 1929; Peckmann, 2002; Slome, 1929)	Colesberg
SAM-AP 4507	SAM	1	A	mature	>20	1866		(Botha and Steyn, 2015; Drennan, 1929; Peckmann, 2002; Slome, 1929)	Colesberg

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
SAM-AP 4508	SAM	1	[F]	mature	20-34	1866		(Botha and Steyn, 2015; Drennan, 1929; Peckmann, 2002; Slome, 1929)	Colesberg
SAM-AP 4509	SAM	1	[F]	mature	>20	1866		(Botha and Steyn, 2015; Drennan, 1929; Peckmann, 2002; Slome, 1929)	Colesberg
SAM-AP 4511	SAM	1	F	mature	>20	1866		(Botha and Steyn, 2015; Drennan, 1929; Peckmann, 2002; Slome, 1929)	Colesberg
SAM-AP 4512	SAM	1	M	mature	20-34	1866		(Botha and Steyn, 2015; Drennan, 1929; Peckmann, 2002; Slome, 1929)	Colesberg
SAM-AP 4515	SAM	1	[F]	mature	>20	1866		(Botha and Steyn, 2015; Drennan, 1929; Peckmann, 2002; Slome, 1929)	Colesberg
SAM-AP 4516	SAM	1	ND	immature	10-19	1866		(Botha and Steyn, 2015; Drennan, 1929; Peckmann, 2002; Slome, 1929)	Colesberg

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
SAM-AP 4518	SAM	1	ND	immature	10-19	1866		(Botha and Steyn, 2015; Drennan, 1929; Peckmann, 2002; Slome, 1929)	Colesberg
SAM-AP 4520	SAM	1	A	mature	35-49	1866		(Botha and Steyn, 2015; Drennan, 1929; Peckmann, 2002; Slome, 1929)	Colesberg
SAM-AP 4521	SAM	1	F	immature	10-34	1866		(Botha and Steyn, 2015; Drennan, 1929; Peckmann, 2002; Slome, 1929)	Colesberg
SAM-AP 4524	SAM	2	ND	mature	>20				
SAM-AP 4527	SAM	1	M	mature	>50				
SAM-AP 4589	SAM	3	[M]	mature	>20				Hermanus
SAM-AP 4620	SAM	3	A	mature	>20				Goodwood
SAM-AP 4621	SAM	3	[M]	mature	>20				
SAM-AP 4623	SAM	3	M	mature	>20				
SAM-AP 4625	SAM	3	F	mature	>20				
SAM-AP 4626	SAM	3	M	mature	>20				
SAM-AP 4627	SAM	3	[F]	mature	>20		2665 ± 27 (OxA-V-2056-34)	(Stynder et al., 2007a)	
SAM-AP 4629	SAM	3	F	mature	>20				Piet se Bank, Plettenberg Bay
SAM-AP 4630	SAM	2	M	mature	>20		1775 ± 80 (Gx 13178)	(Sealy and van der Merwe, 1988)	Sandy Bay
SAM-AP 4631	SAM	3	A	mature	>20				Hout Bay

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
SAM-AP 4632	SAM	3	[M]	mature	>20		2183 ± 26 (OxA-V-2053-42)	(Sealy, 2006)	Peit se Bank, Plettenberg Bay
SAM-AP 4633	SAM	3	[M]	mature	>20				
SAM-AP 4634	SAM	3	[M]	mature	>20				Struisbaai?
SAM-AP 4635	SAM	3	ND	immature	2-19				Port Nolloth
SAM-AP 4636	SAM	2	[M]	mature	>20		2130 ± 45 (Pta 4379)	(Sealy and van der Merwe, 1988)	Bloubergstrand
SAM-AP 4637	SAM	2	F	mature	>50		3880 ± 50 (Pta 4803)	(Sealy, 1989; Sealy et al., 2006)	Gordons Bay
SAM-AP 4659	SAM	3	F	mature	>20		1815 ± 29 (OxA-V-2056-43)	(Stynder et al., 2007a)	Melkbosstrand
SAM-AP 4660	SAM	3	ND	immature	birth-19		440 ± 80 (OxA 454)	(Sealy et al., 2000)	Clanwilliam
SAM-AP 4661	SAM	3	[F]	mature	>20		1310 ± 30 (OxA-V-2055-34)	(Sealy, 2006)	Peit se Bank, Plettenberg Bay
SAM-AP 4662	SAM	3	M	mature	>20				
SAM-AP 4663	SAM	3	ND	immature	birth-9				Upington?
SAM-AP 4664	SAM	3	[F]	mature	>20				Upington
SAM-AP 4665	SAM	3	ND	mature	2-19				
SAM-AP 4666	SAM	3	A	mature	>20				
SAM-AP 4667	SAM	3	[M]	mature	>20				Table Mountain
SAM-AP 4668	SAM	3	M	mature	>20				
SAM-AP 4669	SAM	3	F	mature	>20		1333 ± 25 (OxA-V-2056-28)	(Stynder et al., 2007a)	Gordons Bay
SAM-AP 4670	SAM	3	A	mature	>20				
SAM-AP 4678	SAM	3	ND	mature	>20				Mossel Bay

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
SAM-AP 4679	SAM	3	[F]	mature	>20				
SAM-AP 4680	SAM	3	ND	immature	2-9				
SAM-AP 4681	SAM	3	F	mature	>20				
SAM-AP 4682*	SAM	3	ND	mature	>20				
SAM-AP 4682	SAM	3	ND	mature	>20				Oakhurst Shelter?
SAM-AP 4683	SAM	3	ND	immature	2-19				
SAM-AP 4684 [A]	SAM	3	ND	mature	>20				
SAM-AP 4684 [B]	SAM	3	ND	mature	>20				
SAM-AP 4685	SAM	3	[M]	mature	>20				
SAM-AP 4686	SAM	3	ND	immature	birth-9				
SAM-AP 4687	SAM	2	ND	immature	prenatal-1				
SAM-AP 4688	SAM	3	[F]	mature	>20				Port Nolloth?
SAM-AP 4689	SAM	3	[F]	mature	>20				
SAM-AP 4690	SAM	3	[M]	mature	>20				
SAM-AP 4691*	SAM	3	ND	immature	birth-19				
SAM-AP 4691	SAM	1	[F]	mature	>20				
SAM-AP 4693	SAM	2	ND	immature	2-19				
SAM-AP 4695	SAM	2	M	mature	35-49				
SAM-AP 4696	SAM	3	[M]	mature	>20				
SAM-AP 4697	SAM	2	ND	immature	2-5				
SAM-AP 4698	SAM	2	ND	immature	prenatal-1				
SAM-AP 4699	SAM	2	[M]	mature	>20				
SAM-AP 4700	SAM	2	A	immature	10-34				
SAM-AP 4701	SAM	1	M	mature	>20				Namaqualand
SAM-AP 4702	SAM	2	ND	immature	10-19				
SAM-AP 4703	SAM	1	M	mature	35-49				
SAM-AP 4704	SAM	2	F	mature	>50				
SAM-AP 4707	SAM	3	ND	mature	20-49				
SAM-AP 4708	SAM	1	F	immature	10-34				
SAM-AP 4709	SAM	2	F	mature	35-49				

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
SAM-AP 4713	SAM	2	[M]	mature	>20		3030 ± 60 (Pta 6525)	(Sealy and Pfeiffer, 2000)	Glentyre Shelter
SAM-AP 4716 [A]	SAM	2	ND	immature	2-9				
SAM-AP 4716 [B]	SAM	2	ND	immature	10-19				
SAM-AP 4719	SAM	2	A	mature	>20				
SAM-AP 4720	SAM	2	[M]	mature	>50		2195 ± 80 (GX-13179)	(Sealy and van der Merwe, 1988)	Kommetjie
SAM-AP 4721	SAM	3	F	mature	>50				
SAM-AP 4724	SAM	2	ND	mature	>20				
SAM-AP 4725	SAM	3	ND	mature	>20				
SAM-AP 4726	SAM	3	[F]	mature	35-49				
SAM-AP 4727 B	SAM	2	ND	immature	2-9				
SAM-AP 4727	SAM	2	A	mature	20-34				
SAM-AP 4728 Box 1	SAM	3	ND	mature	>20				Cold Stream
SAM-AP 4729	SAM	3	ND	mature	>20				
SAM-AP 4730	SAM	2	ND	immature	prenatal-1				
SAM-AP 4731	SAM	2	ND	immature	prenatal-1				
SAM-AP 4732	SAM	2	ND	immature	prenatal-5				
SAM-AP 4733 A	SAM	3	ND	mature	>20				
SAM-AP 4733 B	SAM	3	ND	immature	birth-5				
SAM-AP 4733	SAM	2	ND	immature	birth-5				
SAM-AP 4734 A	SAM	3	ND	mature	>20		7440 ± 60 (Pta-5959)	(Sealy and Pfeiffer, 2000)	Cold Stream
SAM-AP 4734 B	SAM	3	ND	mature	>20		7050 ± 80 (Pta 6632)	(Sealy and Pfeiffer, 2000)	Cold Stream
SAM-AP 4734 C	SAM	3	ND	immature	birth-5				Cold Stream
SAM-AP 4734	SAM	3	ND	mature	>20				Cold Stream
SAM-AP 4735	SAM	3	[F]	mature	20-34				

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
SAM-AP 4738	SAM	2	ND	immature	birth-1				
SAM-AP 4739	SAM	3	ND	mature	>20				
SAM-AP 4740	SAM	3	ND	mature	>20				
SAM-AP 4741 A	SAM	3	[M]	mature	>20				Greenpoint
SAM-AP 4741 B	SAM	3	[F]	mature	>20				
SAM-AP 4742 B	SAM	2	F	mature	>50				
SAM-AP 4743	SAM	2	A	mature	>20				Greenpoint
SAM-AP 4746	SAM	2	M	mature	35-49				Greenpoint
SAM-AP 4747	SAM	3	ND	mature	>20				Greenpoint
SAM-AP 4749	SAM	2	ND	mature	>20				Greenpoint
SAM-AP 4752 A	SAM	3	[M]	mature	>20				Greenpoint
SAM-AP 4753	SAM	3	F	mature	>20				Greenpoint
SAM-AP 4754	SAM	3	F	mature	>20				Greenpoint
SAM-AP 4755	SAM	3	[F]	mature	>20				Greenpoint
SAM-AP 4756	SAM	3	A	mature	>20				Greenpoint
SAM-AP 4758	SAM	3	[F]	mature	>20				Greenpoint
SAM-AP 4759	SAM	3	[M]	mature	>20				Greenpoint
SAM-AP 4760	SAM	3	A	mature	>20				
SAM-AP 4761	SAM	3	M	mature	>20				Greenpoint
SAM-AP 4762	SAM	2	ND	mature	>20	1818		(Cox and Sealy, 1997)	Fort Knokke
SAM-AP 4763	SAM	1	M	mature	35-49	1818		(Cox and Sealy, 1997)	Fort Knokke
SAM-AP 4764	SAM	3	[F]	mature	>20			(Cox and Sealy, 1997)	Greenpoint?
SAM-AP 4765	SAM	3	ND	immature	10-19			(Cox and Sealy, 1997)	Greenpoint?
SAM-AP 4766	SAM	3	M	mature	>20				Greenpoint

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
SAM-AP 4767	SAM	3	ND	immature	10-19				
SAM-AP 4774	SAM	2	ND	immature	6-19	1818		(Cox and Sealy, 1997)	Fort Knokke
SAM-AP 4775	SAM	2	A	immature	10-34	1818		(Cox and Sealy, 1997)	Fort Knokke
SAM-AP 4778	SAM	2	M	mature	20-34				
SAM-AP 4781	SAM	2	[M]	immature	10-34	1818		(Cox and Sealy, 1997)	Fort Knokke
SAM-AP 4782	SAM	2	[M]	mature	35-49	1818		(Cox and Sealy, 1997)	Fort Knokke
SAM-AP 4784 A	SAM	3	[F]	mature	>20				
SAM-AP 4784 B	SAM	3	ND	immature	2-9				
SAM-AP 4784 C	SAM	3	ND	immature	birth-19				
SAM-AP 4784	SAM	2	ND	mature	>20				
SAM-AP 4785	SAM	2	A	mature	>20				Kroonstad
SAM-AP 4786	SAM	2	[M]	mature	20-34				Bellville
SAM-AP 4788	SAM	3	ND	immature	6-19	1818		(Cox and Sealy, 1997)	Fort Knokke
SAM-AP 4790	SAM	1	M	mature	20-34		1610 ± 150 (Pta 2163)	(Morris, 1992a)	Hermanus
SAM-AP 4791	SAM	3	ND	mature	>20				North Bay, Saldanha

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
SAM-AP 4793	SAM	2	[M]	mature	>20		4110 ± 60 (Pta 4694)	(Dewar, 2010)	North Bay, Saldanha
SAM-AP 4794	SAM	3	[F]	mature	>20				North Bay, Saldanha
SAM-AP 4795	SAM	3	A	mature	>20				Calypso Bay, Langebaan
SAM-AP 4796	SAM	3	[M]	mature	>20				Grotto Bay / Modder River
SAM-AP 4797	SAM	3	ND	mature	>20				Bokbaai
SAM-AP 4798	SAM	2	ND	immature	2-9				Grotto Bay / Modder River
SAM-AP 4799	SAM	3	[M]	mature	>20				Hout Bay
SAM-AP 4800	SAM	2	F	mature	35-49				Quoin Point
SAM-AP 4802	SAM	3	ND	immature	2-19				Yzerfontein
SAM-AP 4803	SAM	3	ND	mature	>20				Yzerfontein
SAM-AP 4804	SAM	3	[M]	mature	>20				Noordhoek
SAM-AP 4805	SAM	3	ND	mature	>20				St Helena Bay
SAM-AP 4806	SAM	3	ND	immature	birth-5				Bloubergstrand
SAM-AP 4807	SAM	3	ND	mature	>20				St Helena Bay
SAM-AP 4808	SAM	3	ND	immature	6-19				Vleesbaai
SAM-AP 4810	SAM	3	ND	mature	>20				Grotto Bay / Modder River
SAM-AP 4811	SAM	3	[F]	mature	>20				Jeffreys Bay
SAM-AP 4812	SAM	3	[F]	mature	>20				Stompneusbaai
SAM-AP 4813	SAM	2	F	mature	35-49		2140 ± 45 (Pta 4204)	(Sealy and van der Merwe, 1988)	Bokbaai
SAM-AP 4815	SAM	3	[M]	mature	>20				
SAM-AP 4816	SAM	3	[M]	mature	>20				Struisbaai?
SAM-AP 4817	SAM	3	ND	mature	>20				Langebaan Lagoon

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
SAM-AP 4818	SAM	3	[M]	mature	>20				Lamberts Bay
SAM-AP 4819	SAM	3	ND	mature	>20				
SAM-AP 4821	SAM	3	ND	immature	2-9				
SAM-AP 4822	SAM	2	ND	mature	>20				
SAM-AP 4825	SAM	2	A	mature	20-34		2060 ± 50 (Pta 6607)	(Sealy and Pfeiffer, 2000)	Cold Stream
SAM-AP 4826	SAM	2	ND	immature	2-5				
SAM-AP 4827	SAM	3	ND	mature	>20		9720 ± 140 (Pta 6636)	(Sealy and Pfeiffer, 2000)	Tucker's and Van Bonde's Caves
SAM-AP 4828	SAM	3	[M]	mature	>20		9830 ± 80 (Pta 6605)	(Sealy and Pfeiffer, 2000)	Tucker’s Cave
SAM-AP 4829	SAM	2	ND	immature	birth-1			(Pfeiffer and Harrington, 2011)	Tucker's and Van Bonde's Caves
SAM-AP 4830	SAM	3	ND	immature	birth-5				
SAM-AP 4831	SAM	2	ND	mature	>20				
SAM-AP 4832	SAM	3	ND	mature	>20				
SAM-AP 4833	SAM	3	ND	mature	>20				
SAM-AP 4834	SAM	3	[M]	mature	35-49		9270 ± 140 (Pta 6616)	(Sealy and Pfeiffer, 2000)	van Bonde's Cave
SAM-AP 4835	SAM	2	ND	immature	birth-5				Cold Stream
SAM-AP 4836	SAM	3	ND	immature	birth-19				
SAM-AP 4837	SAM	3	[M]	mature	>20				
SAM-AP 4838*	SAM	2	[M]	mature	20-34				Peers Cave
SAM-AP 4838**	SAM	2	F	mature	35-49				Peers Cave
SAM-AP 4838	SAM	3	[M]	mature	>20				Peers Cave
SAM-AP 4839	SAM	1	ND	immature	6-9				Peers Cave
SAM-AP 4840	SAM	1	[F]	immature	10-34				Peers Cave
SAM-AP 4842	SAM	3	ND	mature	>20				

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
SAM-AP 4843	SAM	3	[M]	mature	35-49		3900 ± 25 (Pta 6606)	(Sealy and Pfeiffer, 2000)	van Bonde's Cave
SAM-AP 4844	SAM	1	F	mature	35-49				Peers Cave
SAM-AP 4845	SAM	2	[M]	mature	>20				
SAM-AP 4866	SAM	3	[M]	mature	>20				
SAM-AP 4867	SAM	2	[M]	mature	>20		590 ± 45 (Pta 4407)	(Morris, 1992a)	Vredenburg
SAM-AP 4870	SAM	3	ND	immature	20-34	1818		(Cox and Sealy, 1997)	Fort Knokke
SAM-AP 4871	SAM	3	[F]	mature	>20	1818		(Cox and Sealy, 1997)	Fort Knokke
SAM-AP 4872	SAM	3	A	mature	>20	1818		(Cox and Sealy, 1997)	Fort Knokke
SAM-AP 4874	SAM	3	[M]	mature	>20		1426 ± 29 (OxA-V-2056-45)	(Stynder et al., 2007a)	Cape St Francis
SAM-AP 4875	SAM	2	F	mature	35-49				
SAM-AP 4876	SAM	3	ND	immature	birth-1				
SAM-AP 4877	SAM	3	[F]	mature	>20				
SAM-AP 4878	SAM	3	ND	mature	>20				Norvalspont
SAM-AP 4879	SAM	3	[M]	mature	>20				
SAM-AP 4880	SAM	3	M	mature	>20				
SAM-AP 4881	SAM	3	[M]	mature	>20				Steytlerville
SAM-AP 4882	SAM	3	ND	mature	>20				Norvalspont
SAM-AP 4883	SAM	3	[M]	mature	>20				Hopetown
SAM-AP 4884	SAM	1	ND	immature	10-19				
SAM-AP 4885	SAM	2	ND	mature	>20				
SAM-AP 4886	SAM	3	[M]	mature	>20				Alexandria
SAM-AP 4887	SAM	3	ND	immature	10-19				
SAM-AP 4888	SAM	3	[F]	mature	>20				Swartkops River Mouth

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
SAM-AP 4889	SAM	3	[M]	mature	>20				Bakens River Mouth , PE
SAM-AP 4890	SAM	1	F	mature	35-49				
SAM-AP 4891	SAM	3	ND	mature	>20				
SAM-AP 4892	SAM	1	F	mature	35-49				
SAM-AP 4894	SAM	3	A	mature	>20				New Brighton
SAM-AP 4895	SAM	3	[F]	mature	>20				Douglas
SAM-AP 4896	SAM	3	ND	mature	>20				
SAM-AP 4897	SAM	1	[F]	mature	35-49				
SAM-AP 4898	SAM	3	A	mature	>20		1226 ± 26 (OxA-V-2053-49)	(Sealy, 2006)	Robberg
SAM-AP 4899	SAM	1	[M]	mature	20-49		2440 ± 60 (Pta 4149)	(Sealy and van der Merwe, 1988)	Saldanha
SAM-AP 4900	SAM	2	M	mature	35-49				
SAM-AP 4901	SAM	2	M	mature	35-49		1892 ± 28 (OxA-V-2065-40)	(Stynder et al., 2007a)	Pearly Beach
SAM-AP 4902	SAM	3	ND	mature	>20				
SAM-AP 4904	SAM	3	[M]	mature	>20				Springbok
SAM-AP 4905	SAM	2	[M]	mature	20-34		1210 ± 50 (Pta 4349)	(Sealy and van der Merwe, 1988)	Bloubergstrand
SAM-AP 4906 A	SAM	3	[F]	mature	>20		2635 ± 29 (OxA-V-2065-35)	(Stynder et al., 2007a)	Bloubergstrand
SAM-AP 4906 B	SAM	3	[F]	mature	>20		2977 ± 33 (OxA-V-2056-48)	(Stynder et al., 2007a)	Bloubergstrand
SAM-AP 4906	SAM	3	ND	mature	>20				Bloubergstrand
SAM-AP 4908	SAM	3	ND	mature	>20				

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
SAM-AP 4913	SAM	1	[M]	mature	>50				
SAM-AP 4914	SAM	3	ND	mature	>20				
SAM-AP 4915	SAM	3	ND	mature	>20				Upington
SAM-AP 4917	SAM	1	M	mature	35-49				
SAM-AP 4918 A	SAM	3	[M]	mature	>20				
SAM-AP 4918 B	SAM	3	A	mature	>20				
SAM-AP 4918 C	SAM	3	ND	mature	>20				
SAM-AP 4920 A	SAM	3	[F]	mature	>20		1364 ± 32 (OxA-V-2059-17)	(Stynder et al., 2007a)	Melkbosstrand
SAM-AP 4920 B	SAM	3	[M]	mature	>20				Melkbosstrand
SAM-AP 4920 C	SAM	3	[F]	mature	>20				Melkbosstrand
SAM-AP 4923	SAM	3	[F]	mature	>20				Cape Town
SAM-AP 4924	SAM	3	[F]	mature	>20				Cape Town
SAM-AP 4925	SAM	3	[M]	mature	>20				Cape Town
SAM-AP 4926	SAM	3	A	mature	>20				
SAM-AP 4927	SAM	2	[M]	mature	35-49				
SAM-AP 4928	SAM	1	M	immature	10-34				Melkbosstrand
SAM-AP 4929	SAM	2	ND	mature	>20		1040 ± 50 (Pta 4823)	(Pfeiffer and Sealy, 2006)	Greenpoint
SAM-AP 4930	SAM	3	F	mature	35-49		1130 ± 40 (Pta 4827)	(Black, 2014)?	Greenpoint
SAM-AP 4931	SAM	1	[M]	mature	35-49		3750 ± 60 (Pta 4827)	(Morris, 1992a)	Kleinsee
SAM-AP 4932	SAM	2	ND	immature	10-34				
SAM-AP 4933	SAM	3	[F]	mature	>20				Stompneusbaai
SAM-AP 4934	SAM	2	[M]	mature	20-34		1220 ± 50 (Pta 4210)	(Sealy and van der Merwe, 1988)	Saldanha
SAM-AP 4935	SAM	2	[F]	mature	>20		2540 ± 50 (Pta 4275)	(Sealy and van der Merwe, 1988)	Stompneusbaai

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
SAM-AP 4936	SAM	3	ND	mature	>20				Humansdorp
SAM-AP 4937	SAM	2	F	immature	10-34				
SAM-AP 4938	SAM	3	[M]	mature	>20				Buchuberg Settlement
SAM-AP 4939	SAM	3	ND	immature	10-19				
SAM-AP 4940	SAM	3	A	mature	>20				Augrabies Falls
SAM-AP 4941	SAM	3	[F]	mature	>20				
SAM-AP 4942	SAM	2	A	mature	>20		2220 ± 45 (Pta 4829)	(Sealy, 1989; Stynder et al., 2007a)	Kommetjie
SAM-AP 4943	SAM	2	F	mature	35-49		2610 ± 50 (Pta 4821)	(Pfeiffer and Sealy, 2006; Sealy, 1989)	Kommetjie
SAM-AP 4945	SAM	1	M	mature	20-34				Cape Town
SAM-AP 4946	SAM	1	[F]	mature	>20				Campbell?
SAM-AP 4947	SAM	3	[F]	mature	>20				Diep River
SAM-AP 4948	SAM	3	F	mature	>20				
SAM-AP 4949	SAM	3	[M]	mature	>20				Simon's Town
SAM-AP 4950 A	SAM	3	[F]	mature	>20				
SAM-AP 4950 B	SAM	2	[M]	mature	>20				Pearly beach
SAM-AP 4951	SAM	3	[M]	mature	2-9				
SAM-AP 4952	SAM	3	[M]	mature	>20				
SAM-AP 4953	SAM	3	[M]	mature	>20				
SAM-AP 4954	SAM	3	M	mature	>20				
SAM-AP 4955	SAM	3	[F]	mature	>20				
SAM-AP 4956	SAM	3	[F]	mature	>20				
SAM-AP 4957	SAM	3	[F]	mature	>20				
SAM-AP 4958	SAM	3	ND	immature	2-5				
SAM-AP 4959	SAM	3	[F]	mature	>20				Noordhoek
SAM-AP 4960	SAM	3	ND	mature	>20				
SAM-AP 4962	SAM	1	[F]	mature	>50				O'Kiep

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
SAM-AP 4964	SAM	2	A	immature	10-34		2190 ± 60 (Pta-8756)	(Sealy, 2010)	Die Dam
SAM-AP 4965 A	SAM	3	ND	immature	birth-19				
SAM-AP 4965 B	SAM	2	ND	mature	>20				
SAM-AP 4966	SAM	2	F	mature	>35				
SAM-AP 4967	SAM	2	F	mature	35-49				
SAM-AP 4968	SAM	3	ND	mature	>20				
SAM-AP 4969	SAM	2	[F]	mature	35-49				
SAM-AP 4972	SAM	3	A	mature	>20				Tulbagh
SAM-AP 4974 A	SAM	3	[F]	mature	>20				Gansbaai
SAM-AP 4974	SAM	3	[F]	mature	>20		3363 ± 34 (OxA-V-2055-48)	(Stynder et al., 2007a)	Gansbaai
SAM-AP 4976	SAM	3	[F]	mature	>20				
SAM-AP 4979 A	SAM	3	ND	immature	birth-19				Kalk Bay
SAM-AP 4979 B	SAM	2	ND	immature	prenatal-1				Kalk Bay
SAM-AP 4981	SAM	3	A	mature	>20				
SAM-AP 5009	SAM	1	[M]	immature	10-34				
SAM-AP 5010	SAM	3	[F]	mature	>20				
SAM-AP 5012	SAM	3	[F]	mature	>20		812 ± 26 (OxA-V-2065-36)	(Stynder et al., 2007a)	Langebaan Lagoon
SAM-AP 5013	SAM	3	[F]	mature	>20				
SAM-AP 5014	SAM	3	[F]	mature	>20				
SAM-AP 5029	SAM	1	M	mature	>35				
SAM-AP 5030	SAM	3	[M]	mature	>20				
SAM-AP 5031	SAM	3	A	mature	>20				
SAM-AP 5032	SAM	3	[M]	mature	>20		765 ± 25 (OxA-V-2056-35)	(Stynder et al., 2007a)	Milnerton
SAM-AP 5033	SAM	3	A	mature	>20				

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
SAM-AP 5034	SAM	2	[F]	mature	35-49		1390 ± 40 (Pta 4771)	(Sealy, 1989)	Hout Bay
SAM-AP 5035 A	SAM	1	M	mature	>35		620 ± 35 (Pta 4401)	(Sealy, 1989)	Melkbosstrand
SAM-AP 5035 B	SAM	3	M	mature	>20		2011 ± 30 (OxA-V-2055-46)	(Stynder et al., 2007a)	Melkbosstrand
SAM-AP 5035 C	SAM	3	A	mature	>20				Melkbosstrand
SAM-AP 5035 D	SAM	3	A	mature	>20				Melkbosstrand
SAM-AP 5035 E [A]	SAM	2	ND	mature	>20				Melkbosstrand
SAM-AP 5035 E [B]	SAM	2	ND	mature	>20				Melkbosstrand
SAM-AP 5035 E [C]	SAM	3	ND	mature	>20				Melkbosstrand
SAM-AP 5036	SAM	1	M	mature	20-49				Melkbosstrand
SAM-AP 5037	SAM	1	M	mature	35-49				
SAM-AP 5038	SAM	1	[F]	immature	10-34				Bellville
SAM-AP 5039	SAM	3	[F]	mature	>20				
SAM-AP 5040	SAM	2	F	mature	>50		3570 ± 60 (Pta 4225)	(Sealy and van der Merwe, 1988)	Bokbaai
SAM-AP 5041	SAM	1	[F]	mature	20-49		2010 ± 50 (Pta 4376)	(Sealy and van der Merwe, 1988)	Melkbosstrand
SAM-AP 5044	SAM	1	ND	immature	6-19				Robberg
SAM-AP 5045	SAM	3	[F]	mature	>20				Robberg
SAM-AP 5047	SAM	2	[F]	mature	35-49				Britstown
SAM-AP 5048	SAM	2	A	immature	10-34		2780 ± 60 (Pta 7924)	(Sealy, 2006)	Robberg

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
SAM-AP 5049	SAM	2	A	mature	>20		2740 ± 50 (Pta 7934)	(Sealy, 2006)	Robberg
SAM-AP 5050	SAM	2	[F]	immature	10-34		2580 ± 60 (Pta 7927)	(Sealy, 2006)	Robberg
SAM-AP 5051	SAM	3	M	mature	>20		207 ± 25 (OxA-V-2053-48)	(Sealy, 2006)	Robberg
SAM-AP 5052	SAM	3	ND	immature	10-19		2416 ± 27 (OxA-V-2053-46)	(Sealy, 2006)	Robberg
SAM-AP 5053	SAM	3	[M]	mature	>20		370 ± 27 (OxA-V-2065-41)	(Sealy, 2006)	Robberg
SAM-AP 5054	SAM	3	[M]	mature	>20				Robberg
SAM-AP 5055	SAM	3	M	mature	>20		6995 ± 50 (OxA-V-2065-42)	Sealy, 2006; (Stynder et al., 2007a)	Robberg
SAM-AP 5056	SAM	3	[F]	mature	>20				
SAM-AP 5058	SAM	3	ND	immature	birth-19				
SAM-AP 5059	SAM	3	[M]	mature	>20				Greenpoint
SAM-AP 5060	SAM	3	[M]	mature	>20				Greenpoint
SAM-AP 5061	SAM	3	[F]	mature	>20				Greenpoint
SAM-AP 5062	SAM	2	A	immature	10-34				Greenpoint
SAM-AP 5063	SAM	3	M	mature	>20				Greenpoint
SAM-AP 5064	SAM	3	A	mature	>20				
SAM-AP 5065	SAM	3	A	mature	>20				Greenpoint
SAM-AP 5068	SAM	2	A	mature	35-49		5680 ± 70 (Pta 4370)	(Sealy and van der Merwe, 1988)	Yzerfontein
SAM-AP 5069	SAM	3	[F]	mature	>20		2634 ± 28 (OxA-V-2066-34)	(Stynder et al., 2007a)	Doringbaai

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
SAM-AP 5070	SAM	3	[F]	mature	>20		2573 ± 31 (OxA-V-2056-46)	(Stynder et al., 2007a)	Melkbosstrand
SAM-AP 5071	SAM	3	ND	mature	>20				
SAM-AP 5072	SAM	3	A	mature	>20				Hopefield
SAM-AP 5073	SAM	2	ND	immature	10-19				Groot Hagelkraal
SAM-AP 5074	SAM	2	A	immature	10-34				
SAM-AP 5075	SAM	2	[M]	mature	35-49		2530 ± 60 (Pta 4669)	(Sealy, 1989)	Cape Point
SAM-AP 5076	SAM	3	M	mature	35-49		1510 ± 40 (Pta-4738)	(Sealy, 1989)	Hout Bay?
SAM-AP 5077 (4)	SAM	2	[M]	mature	35-49				Cape Agulhas
SAM-AP 5077 (3)	SAM	2	ND	immature	6-19				Cape Agulhas
SAM-AP 5077 (2)	SAM	2	ND	mature	20-34				Cape Agulhas
SAM-AP 5080	SAM	3	ND	mature	>20				
SAM-AP 5081	SAM	1	[M]	mature	35-49				Faure
SAM-AP 5082	SAM	1	[M]	mature	>20		2150 ± 60 (Pta 4199)	(Sealy and van der Merwe, 1988)	Hout Bay
SAM-AP 5083	SAM	1	[F]	mature	>50		1490 ± 50 (Pta 926)	(Sealy and van der Merwe, 1988)	Yzerfontein
SAM-AP 5084	SAM	3	A	mature	>20				Yzerfontein
SAM-AP 5085	SAM	3	ND	mature	>20				
SAM-AP 5086	SAM	3	ND	mature	>20				Oakhurst Shelter
SAM-AP 5087	SAM	3	ND	immature	2-5				Kommetjie
SAM-AP 5088	SAM	3	A	mature	>20				
SAM-AP 5089	SAM	3	ND	mature	>20				
SAM-AP 5090	SAM	2	M	mature	35-49				Cape Town

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
SAM-AP 5091	SAM	1	[M]	mature	>50		2830 ± 50 (Pta 4724)	(Sealy, 1989)	Yzerfontein
SAM-AP 5092	SAM	3	[M]	mature	>20				Britstown
SAM-AP 5094	SAM	3	ND	immature	2-19				
SAM-AP 5095	SAM	1	[F]	mature	>20		2660 ± 70 (Pta 4674)	(Sealy, 1989)	Saldanha
SAM-AP 5096	SAM	3	ND	mature	>20				
SAM-AP 6012	SAM	3	F	mature	>20				
SAM-AP 6013	SAM	3	ND	mature	>20				
SAM-AP 6014 [A]	SAM	2	[M]	mature	>20				
SAM-AP 6014 [B]	SAM	3	ND	immature	birth-19				
SAM-AP 6015	SAM	3	[F]	mature	>20				
SAM-AP 6016	SAM	3	A	mature	>20		2813 ± 29 (OxA-V-2053-45)	(Sealy, 2006)	Robberg
SAM-AP 6017	SAM	3	ND	mature	>20		2490 ± 50 (Pta 4293)	(Sealy and van der Merwe, 1988)	Melkbosstrand
SAM-AP 6018	SAM	2	[F]	mature	>20				
SAM-AP 6020	SAM	1	M	mature	35-49		620 ± 30 (Pta 4189)	(Sealy and van der Merwe, 1988)	Saldanha
SAM-AP 6021	SAM	3	ND	mature	>20				
SAM-AP 6022	SAM	1	[M]	mature	35-49				Pearly Beach
SAM-AP 6023	SAM	2	[M]	mature	>50		2355 ± 85 (Gx-13180)	(Sealy, 1989)	Melkbosstrand
SAM-AP 6024	SAM	3	F	mature	>20				Hout Bay
SAM-AP 6025	SAM	3	M	mature	>20				
SAM-AP 6026	SAM	3	[M]	mature	>20				Greenpoint
SAM-AP 6027	SAM	3	[F]	mature	>20				Greenpoint
SAM-AP 6028 G	SAM	3	A	mature	>20				Greenpoint
SAM-AP 6028 H Individual A	SAM	3	A	mature	>20				Greenpoint

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
SAM-AP 6028 H Individual B	SAM	3	[M]	mature	>20				Greenpoint
SAM-AP 6028 I	SAM	3	[M]	mature	>20				Greenpoint
SAM-AP 6028 J	SAM	3	A	mature	>20				Greenpoint
SAM-AP 6029	SAM	1	M	mature	35-49				Victoria West
SAM-AP 6030	SAM	2	[M]	mature	35-49				Langebaan Lagoon
SAM-AP 6031	SAM	2	[M]	immature	10-34		2560 ± 50 (Pta 4814)	(Sealy, 1989)	Langebaan Lagoon
SAM-AP 6032	SAM	2	[M]	mature	35-49		5180 ± 65 (Pta-1089)	(de Villiers, 1974)	Cape St Francis
SAM-AP 6033	SAM	1	[M]	immature	10-34				
SAM-AP 6039	SAM	2	[M]	mature	>20				
SAM-AP 6040	SAM	2	[F]	mature	35-49				Heidelberg
SAM-AP 6041 A	SAM	3	ND	immature	10-19		1800 ± 50 (Pta 4722) 1824 ± 27 (OxA-V-2056-27)	(Sealy, 1989; Stynder et al., 2007a)	Milnerton
SAM-AP 6041 B	SAM	3	M	mature	35-49		2010 ± 45 (Pta 4768)	(Sealy, 1989)	Milnerton
SAM-AP 6041 C	SAM	3	ND	mature	>20				Ysterplaat
SAM-AP 6042	SAM	3	M	mature	>20				
SAM-AP 6043	SAM	2	[M]	mature	>20		2295 ± 28 (OxA-V-2056-40)	(Stynder et al., 2007a)	Melkbosstrand
SAM-AP 6044	SAM	3	[F]	mature	>20				Riversdale
SAM-AP 6047 A	SAM	3	M	mature	>20				Saldanha
SAM-AP 6047 B	SAM	2	A	mature	>20				Saldanha
SAM-AP 6048 [A]	SAM	2	ND	immature	6-19				De Hoop Nature Reserve

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
SAM-AP 6048 [B]	SAM	2	[M]	mature	>20				De Hoop Nature Reserve
SAM-AP 6049	SAM	1	[M]	mature	>50				Byneskranskop
SAM-AP 6050	SAM	2	A	mature	>50		1480 ± 50 (Pta 2855)	(de Villiers and Wilson, 1982)	Byneskranskop
SAM-AP 6051	SAM	2	F	mature	20-34		3190 ± 50 (Pta 2969)	(de Villiers and Wilson, 1982)	Byneskranskop
SAM-AP 6052	SAM	1	ND	immature	6-9		1780 ± 50 (Pta 2869)	(de Villiers and Wilson, 1982)	Byneskranskop
SAM-AP 6053	SAM	2	ND	immature	birth-9				Byneskranskop
SAM-AP 6054 [A]	SAM	2	ND	immature	2-9		2780 ± 45 (Pta 4211)	(Sealy and van der Merwe, 1988)	Modder River
SAM-AP 6054 [B]	SAM	2	ND	immature	birth-1		2530 ± 60 (Pta 4151)	(Sealy and van der Merwe, 1988)	Modder River
SAM-AP 6054 [C]	SAM	2	ND	immature	birth-5	ca. 2600 BP		(Pfeiffer and Harrington, 2011)	Modder River
SAM-AP 6056	SAM	2	ND	immature	2-9				
SAM-AP 6057	SAM	3	[M]	mature	>20				
SAM-AP 6058	SAM	3	M	mature	>50				Byneskranskop
SAM-AP 6061	SAM	2	ND	mature	>20				
SAM-AP 6062	SAM	3	[F]	mature	>20				Betty's Bay
SAM-AP 6063	SAM	2	[F]	mature	35-49		1170 ± 30 (Pta 4279)	(Sealy and van der Merwe, 1988)	Saldanha
SAM-AP 6064	SAM	2	M	mature	>20				
SAM-AP 6066	SAM	3	M	mature	>20				
SAM-AP 6067	SAM	1	A	immature	10-34				Greenpoint/De Waterkant
SAM-AP 6068	SAM	2	M	mature	35-49				Greenpoint/De Waterkant

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
SAM-AP 6070a	SAM	3	A	mature	>20				Greenpoint/De Waterkant
SAM-AP 6070	SAM	2	[M]	mature	20-34				Greenpoint/De Waterkant
SAM-AP 6071	SAM	3	[M]	mature	>20		2935 ± 32 (OxA-V-2055-42)	(Stynder et al., 2007a)	Vredenburg
SAM-AP 6072	SAM	2	M	mature	20-34				Vredenburg
SAM-AP 6073	SAM	3	[M]	mature	>20				
SAM-AP 6074	SAM	1	ND	immature	10-19		1360 ± 40 (Pta 4148)	(Sealy and van der Merwe, 1988)	Saldanha
SAM-AP 6075	SAM	1	F	mature	35-49		1330 ± 40 (Pta 4186)	(Sealy and van der Merwe, 1988)	Saldanha
SAM-AP 6076	SAM	3	[F]	mature	>20				
SAM-AP 6078	SAM	3	[F]	mature	>20				Saldanha
SAM-AP 6079b	SAM	3	ND	immature	birth-1				
SAM-AP 6079	SAM	3	ND	immature	10-34				
SAM-AP 6082	SAM	3	ND	mature	>20				Oakhurst Shelter
SAM-AP 6083	SAM	2	F	mature	35-49		2000 ± 50 (Pta 4358)	(Sealy and van der Merwe, 1988)	Milnerton
SAM-AP 6084	SAM	1	[F]	mature	>20				
SAM-AP 6085	SAM	2	ND	immature	10-19		1850 ± 60 (Pta 4224)	(Sealy and van der Merwe, 1988)	Milnerton
SAM-AP 6086	SAM	2	ND	immature	2-9				
SAM-AP 6087	SAM	3	ND	immature	2-19				Buffels River Beach
SAM-AP 6088	SAM	3	ND	immature	birth-1				
SAM-AP 6089	SAM	3	ND	mature	>20				
SAM-AP 6091a	SAM	3	ND	immature	birth-19				
SAM-AP 6091b	SAM	3	ND	immature	birth-1				
SAM-AP 6114	SAM	1	[F]	mature	35-49	1837 - 1920		(February, 1996)	Vredendal

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SAM-AP 6115	SAM	1	ND	immature	birth-1	1837 - 1920		(February, 1996)	Vredendal
SAM-AP 6116	SAM	2	ND	immature	birth-1	1837 - 1920		(February, 1996)	Vredendal
SAM-AP 6117	SAM	1	M	mature	35-49	1837 - 1920		(February, 1996)	Vredendal
SAM-AP 6118	SAM	3	[M]	mature	>20			(February, 1996)	Vredendal
SAM-AP 6119	SAM	2	ND	immature	prenatal-1	1837 - 1920		(February, 1996)	Vredendal
SAM-AP 6120	SAM	1	ND	immature	birth-1	1837 - 1920		(February, 1996)	Vredendal
SAM-AP 6121	SAM	1	M	mature	35-49	1837 - 1920		(February, 1996)	Vredendal
SAM-AP 6122	SAM	2	ND	immature	prenatal-1	1837 - 1920		(February, 1996)	Vredendal
SAM-AP 6123	SAM	1	ND	immature	birth-5	1837 - 1920		(February, 1996)	Vredendal
SAM-AP 6124	SAM	2	ND	immature	prenatal-1	1837 - 1920		(February, 1996)	Vredendal
SAM-AP 6125	SAM	2	ND	immature	birth-1	1837 - 1920		(February, 1996)	Vredendal
SAM-AP 6126*	SAM	3	ND	immature	prenatal-1	1837 - 1920		(February, 1996)	Vredendal
SAM-AP 6126	SAM	2	ND	mature	>20	1837 - 1920		(February, 1996)	Vredendal
SAM-AP 6127	SAM	1	ND	immature	birth-1	1837 - 1920		(February, 1996)	Vredendal
SAM-AP 6128	SAM	2	ND	immature	prenatal-1	1837 - 1920		(February, 1996)	Vredendal
SAM-AP 6129	SAM	1	ND	immature	birth-1	1837 - 1920		(February, 1996)	Vredendal

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SAM-AP 6130	SAM	1	ND	immature	birth-1	1837 - 1920		(February, 1996)	Vredendal
SAM-AP 6131	SAM	2	ND	immature	prenatal-1	1837 - 1920		(February, 1996)	Vredendal
SAM-AP 6132	SAM	1	ND	immature	prenatal-1	1837 - 1920		(February, 1996)	Vredendal
SAM-AP 6133	SAM	2	ND	immature	birth-5	1837 - 1920		(February, 1996)	Vredendal
SAM-AP 6134	SAM	2	ND	immature	prenatal-1	1837 - 1920		(February, 1996)	Vredendal
SAM-AP 6135	SAM	2	ND	immature	prenatal-1	1837 - 1920		(February, 1996)	Vredendal
SAM-AP 6136	SAM	2	ND	immature	prenatal-1	1837 - 1920		(February, 1996)	Vredendal
SAM-AP 6137	SAM	2	ND	immature	prenatal-1	1837 - 1920		(February, 1996)	Vredendal
SAM-AP 6138	SAM	2	ND	immature	prenatal-1	1837 - 1920		(February, 1996)	Vredendal
SAM-AP 6139	SAM	2	ND	immature	prenatal-1	1837 - 1920		(February, 1996)	Vredendal
SAM-AP 6140	SAM	2	ND	immature	birth-1	1837 - 1920		(February, 1996)	Vredendal
SAM-AP 6141	SAM	1	F	mature	>50	1837 - 1920		(February, 1996)	Vredendal
SAM-AP 6142	SAM	1	M	mature	35-49	1837 - 1920		(February, 1996)	Vredendal
SAM-AP 6143	SAM	1	ND	immature	birth-1	1837 - 1920		(February, 1996)	Vredendal
SAM-AP 6144	SAM	2	ND	immature	prenatal-1	1837 - 1920		(February, 1996)	Vredendal
SAM-AP 6145	SAM	2	M	mature	35-49	1837 - 1920		(February, 1996)	Vredendal

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SAM-AP 6146	SAM	1	ND	immature	2-9	1837 - 1920		(February, 1996)	Vredendal
SAM-AP 6147	SAM	1	[M]	mature	20-34		2920 ± 60 (Pta 8774)	(Stynder et al., 2007a)	Vredendal
SAM-AP 6148	SAM	1	[F]	immature	10-34				Koingnaas
SAM-AP 6150	SAM	1	F	mature	35-49				Koingnaas
SAM-AP 6151	SAM	2	M	mature	35-49				Koingnaas
SAM-AP 6152	SAM	3	[M]	mature	20-34				
SAM-AP 6153	SAM	3	ND	immature	6-19				Gouritz
SAM-AP 6154	SAM	1	ND	immature	birth-9	1837 - 1920		(February, 1996)	Vredendal
SAM-AP 6155	SAM	2	ND	immature	birth-1	1837 - 1920		(February, 1996)	Vredendal
SAM-AP 6156	SAM	1	ND	immature	birth-5	1837 - 1920		(February, 1996)	Vredendal
SAM-AP 6157	SAM	3	[M]	mature	20-34	1837 - 1920		(February, 1996)	Vredendal
SAM-AP 6158	SAM	2	ND	immature	prenatal-1	1837 - 1920		(February, 1996)	Vredendal
SAM-AP 6159	SAM	1	ND	mature	35-49	1837 - 1920		(February, 1996)	Vredendal
SAM-AP 6161	SAM	1	ND	immature	10-19	1837 - 1920		(February, 1996)	Vredendal
SAM-AP 6162	SAM	1	M	mature	20-49	1837 - 1920		(February, 1996)	Vredendal
SAM-AP 6163	SAM	1	M	mature	35-49	1837 - 1920		(February, 1996)	Vredendal
SAM-AP 6164	SAM	1	ND	immature	birth-5	1837 - 1920		(February, 1996)	Vredendal
SAM-AP 6165	SAM	1	M	mature	35-49	1837 - 1920		(February, 1996)	Vredendal

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SAM-AP 6166	SAM	3	ND	immature	prenatal-1				Koingnaas
SAM-AP 6211	SAM	3	[F]	mature	20-34				Sedgefield
SAM-AP 6212	SAM	3	M	mature	20-34				Sedgefield
SAM-AP 6213	SAM	3	M	mature	>20		1558 ± 27 (OxA-V-2065-39)	(Stynder et al., 2007a)	Sedgefield
SAM-AP 6214	SAM	3	ND	immature	10-19				
SAM-AP 6215	SAM	3	ND	immature	2-9				
SAM-AP 6217	SAM	3	[F]	mature	>20				
SAM-AP 6220	SAM	3	ND	mature	>20				
SAM-AP 6221	SAM	3	ND	mature	>20		880 ± 50 (Pta 4356)	(Sealy and van der Merwe, 1988)	Yzerfontein
SAM-AP 6231	SAM	2	M	mature	>20				Kleinbaai
SAM-AP 6232	SAM	2	[F]	mature	35-49		560 ± 50 (Pta 6611)	(Sealy and Pfeiffer, 2000)	Sedgefield
SAM-AP 6233	SAM	2	M	mature	35-49				
SAM-AP 6234	SAM	3	[M]	mature	20-34				Paternoster
SAM-AP 6235 A	SAM	3	ND	immature	birth-1				Arniston
SAM-AP 6235 B	SAM	3	A	mature	>20				Arniston
SAM-AP 6235 C	SAM	3	[M]	mature	>20				
SAM-AP 6240	SAM	3	ND	mature	>20				
SAM-AP 6241	SAM	3	M	mature	>20				Port Nolloth
SAM-AP 6251	SAM	1	M	mature	35-49				Droerivier, Calvinia
SAM-AP 6252 A	SAM	2	F	immature	10-34				Klein Sleutelfontein
SAM-AP 6252 B	SAM	3	ND	immature	prenatal-5				
SAM-AP 6253	SAM	2	F	mature	>20				
SAM-AP 6254	SAM	3	[F]	mature	2-19				
SAM-AP 6259	SAM	3	[F]	mature	>20				
SAM-AP 6260 B	SAM	2	ND	immature	2-5				

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SAM-AP 6260 C	SAM	2	ND	immature	prenatal-1				
SAM-AP 6262	SAM	3	[M]	mature	>20				
SAM-AP 6263	SAM	3	ND	immature	6-19				
SAM-AP 6266	SAM	2	ND	immature	2-5				
SAM-AP 6270	SAM	3	ND	mature	>20				Grotto Bay/Modder River
SAM-AP 6271	SAM	3	M	mature	>20				
SAM-AP 6273	SAM	3	[M]	mature	>20				
SAM-AP 6274	SAM	3	A	mature	>20				
SAM-AP 6283	SAM	1	F	mature	35-49				
SAM-AP 6284	SAM	3	ND	immature	prenatal-1				
SAM-AP 6285	SAM	1	M	mature	35-49				
SAM-AP 6298	SAM	3	ND	mature	>20				Melkbosstrand
SAM-AP 6299	SAM	3	ND	mature	>20				
SAM-AP 6307	SAM	3	[F]	mature	>20				
SAM-AP 6308	SAM	2	ND	immature	birth-1				
SAM-AP 6309	SAM	2	ND	immature	6-19				Melkbosstrand
SAM-AP 6310	SAM	2	[F]	immature	10-34				Melkbosstrand
SAM-AP 6311	SAM	3	ND	mature	>20				
SAM-AP 6312	SAM	3	ND	immature	prenatal-1				Vredenburg
SAM-AP 6313 C	SAM	2	[M]	mature	>20				Melkbosstrand
SAM-AP 6314	SAM	2	ND	immature	prenatal-1				Lamberts Bay
SAM-AP 6315	SAM	2	ND	immature	birth-5		1985 ± 50 (OxA 6218)	(Sealy et al., 2000)	Watervalsrivier, Clanwilliam
SAM-AP 6316	SAM	3	[M]	mature	>20				Saldanha
SAM-AP 6317	SAM	1	M	mature	35-49		2970 ± 60 (Pta 8807)	(Stynder et al., 2007a)	Melkbosstrand

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SAM-AP 6318	SAM	1	M	mature	35-49		3310 ± 60 (Pta 8741)	(Stynder et al., 2007a)	Melkbosstrand
SAM-AP 6319	SAM	2	F	mature	>20		3200 ± 35 (Pta 8752)	(Stynder et al., 2007a)	Melkbosstrand
SAM-AP 6323	SAM	2	M	mature	35-49				
SAM-AP 6324	SAM	2	ND	immature	6-19				
SAM-AP 6329	SAM	1	F	mature	35-49				Yzerfontein
SAM-AP 6330	SAM	2	F	mature	35-49				Melkbosstrand
SAM-AP 6331	SAM	2	F	mature	>50		790 ± 90 (TO 8953)	(Pfeiffer and Sealy, 2006)	St Helena Bay
SAM-AP 6332	SAM	1	[M]	immature	10-34		980 ± 50 (Pta 8767)	(Stynder et al., 2007a)	Melkbosstrand
SAM-AP 6333	SAM	1	F	mature	>20				Hospitaalpunt
SAM-AP 6335	SAM	1	ND	immature	2-9				Blombos
SAM-AP 6336	SAM	3	ND	mature	>20				Riversdale
SAM-AP 6337*	SAM	3	ND	immature	6-19				Tabakbaai, Saldanha
SAM-AP 6337	SAM	3	ND	mature	>20				Tabakbaai, Saldanha
SAM-AP 6338	SAM	3	ND	mature	>20				Britannia Bay
SAM-AP 6339	SAM	3	ND	mature	>20				Herbertsdale
SAM-AP 6340	SAM	3	ND	immature	2-9				Kreeftebaai
SAM-AP 6341	SAM	2	ND	mature	>20				Igoda River Mouth
SAM-AP 6342	SAM	3	A	mature	>20				
SAM-AP 6343	SAM	2	ND	immature	2-5				
SAM-AP 6344	SAM	3	[M]	mature	>20				
SAM-AP 6345	SAM	3	ND	immature	birth-1				Ganzekraal
SAM-AP 6347	SAM	2	ND	immature	prenatal-5				Peers Cave
SAM-AP 6348 A	SAM	1	ND	immature	10-19		2490 ± 50 (Gx-23871)	(Pfeiffer et al., 1999)	Melkbosstrand
SAM-AP 6348 B	SAM	1	F	mature	>35				Melkbosstrand
SAM-AP 6349	SAM	2	M	mature	>20				Godegevonden, Wolseley

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SAM-AP 6350 A	SAM	3	ND	mature	>20				
SAM-AP 6351	SAM	3	[M]	mature	>20				
SAM-AP 6352	SAM	3	ND	immature	2-5				North Bay, Saldanha
SAM-AP 6353	SAM	3	[M]	mature	>20				
SAM-AP 6354	SAM	3	[M]	mature	>20				
SAM-AP 6355	SAM	3	[M]	mature	>20				
SAM-AP 6356 A	SAM	1	ND	immature	10-19				Jakkalsfontein
SAM-AP 6358	SAM	2	F	mature	>50				Calypso Bay, Langebaan
SAM-AP 6359	SAM	1	M	mature	20-49				De Kelders
SAM-AP 6360	SAM	3	ND	immature	2-5				Melkbosstrand
SAM-AP 6361	SAM	2	F	mature	>50				
SAM-AP 6363 A	SAM	1	M	mature	>20				Yzerfontein
SAM-AP 6363 B	SAM	2	M	mature	35-49				Yzerfontein
SAM-AP 6364	SAM	1	ND	immature	2-9				Melkbosstrand
SAM-AP 6367 A	SAM	1	F	mature	>50				Paternoster
SAM-AP 6367 B	SAM	3	ND	mature	>20				Paternoster
SAM-AP 6367 C	SAM	2	ND	immature	2-5				Paternoster
SAM-AP 4711/12	SAM	3	ND	mature	>20				Glentyre Shelter
SAM-AP 4714/5	SAM	2	M	mature	>20				Glentyre Shelter
SAM-AP 6026/7	SAM	3	ND	mature	>20				Greenpoint
SAM-AP 6071/2	SAM	3	ND	mature	>20				
UCT 552	UCT	1	M	mature	35-49	1755-1827 AD		(Apollonio, 1998)	Greenpoint
UP 1	UP	2	M	mature	35-49		1310 ± 50 (Pta 2692)	(Steyn et al., 1994)	Happy Rest
UP 2	UP	2	F	mature	20-49				Happy Rest
UP 4	UP	3	[M]	mature	>20				Vrede
UP 5	UP	3	ND	mature	>20				Barberton Nature Reserve

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UP 7	UP	3	ND	immature	6-19		300 ± 60 (Pta 5894)	(Steyn and Broekhutzen, 1993)	Rooiberg
UP 8 (A84)	UP	3	[F]	mature	birth-9				Derdepoort
UP 8	UP	2	M	mature	35-49		240 ± 50 (Pta 548)	(Voigt and de Villiers, 1972)	Welgegund
UP 9	UP	2	F	mature	>35		100 ± 40 (Pta 5273)	(Morris, 1984)	Suurbergspruit
UP 11	UP	2	[F]	mature	>20				Vryheid
UP 12	UP	1	F	mature	>35				Derdepoort
UP 13	UP	3	A	mature	>20				Hole-in-the-Wall
UP 14	UP	3	A	mature	>20				Louis Trichardt
UP 16	UP	3	[F]	mature	>20				Still Bay
UP 17 [A]	UP	3	[M]	mature	>20				Echo Caves
UP 17 [B]	UP	3	[F]	mature	>20				Echo Caves
UP 26	UP	3	[M]	mature	>20				Polokwane
UP 27	UP	2	ND	immature	2-5				Gluckstadt
UP 28	UP	1	[M]	immature	10-19		130 ± 30 (Pta 6667)	(Pistorius and Steyn, 1995)	Brits
UP 29	UP	2	M	mature	35-49				Skukuza
UP 32	UP	3	ND	mature	>20				Schuinsdraai
UP 33 [A]	UP	2	F	mature	>20				Phalaborwa
UP 33 [B]	UP	3	ND	mature	>20				Phalaborwa
UP 34	UP	2	[M]	mature	20-49				
UP 35	UP	2	F	mature	>50				
UP 36?? [A]	UP	3	F	mature	>20				Brits
UP 36?? [B]	UP	3	F	mature	>20				Brits
UP 39	UP	3	ND	immature	10-19				Nelspruit
UP 41? / 42?	UP	2	[F]	immature	10-34				Pilanesberg?
UP 45 (A44)	UP	3	ND	immature	10-19				Schuinsdraai

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UP 47 (A46)	UP	3	ND	mature	>20				Schuinsdraai
UP 48 (A47)	UP	1	M	mature	35-49	AD 1677 (1693,1727) 1768; 1802 (1816) 1893	(Pta-7380)	(Pistorius et al., 1998)	Makgope
UP 49 (A48)	UP	2	A	mature	>20	AD 1670 (1682) 1702; 1716 (1745) 1784; 1792 (1807) 1822	(Pta-7379)	(Pistorius et al., 1998)	Makgope
UP 50	UP	3	ND	mature	>20	LSA		(Korsman and Plug, 1994)?	Honingklip
UP 51	UP	2	[F]	immature	10-34		890 ± 50 (Pta 7331)	(L'Abbé et al., 2008)	Pilanesberg
UP 52	UP	2	ND	mature	>20				Krugersdorp
UP 54 [A]	UP	2	[M]	mature	>20				Krugersdorp
UP 54 [B]	UP	3	ND	immature	10-34				Krugersdorp
UP 54 [C]	UP	3	ND	immature	10-34				Krugersdorp
UP 55 (A56)	UP	2	[M]	mature	>20				Krugersdorp
UP 56 (A56)	UP	2	ND	immature	birth-19				Krugersdorp
UP 60	UP	2	ND	immature	10-19				
UP 61	UP	2	ND	immature	prenatal-1				
UP 62 (A62)	UP	2	ND	mature	>20				Krugersdorp
UP 63 (A64)	UP	2	ND	immature	10-19				
UP 64 (A64)	UP	2	M	mature	35-49				
UP 66 (A66)	UP	3	ND	mature	>20				Krugersdorp
UP 67 (A66)	UP	3	ND	mature	>20				
UP 68 (A66)	UP	3	ND	mature	>20				
UP 69 (A66)	UP	3	ND	mature	>20				
UP 70 (A67)	UP	3	ND	immature	6-19				Ongelukskraal
UP 71 (A67)	UP	2	[F]	mature	>20				Willowglen

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UP 72 (A68)	UP	1	F	immature	10-34				Willowglen
UP 73 (A69)	UP	1	M	mature	35-49		40 ± 45 (Pta 7549)	(Pistorius et al., 2002)	Malle
UP 74 (A70)	UP	2	ND	immature	birth-1				Malle
UP 75 (A71)	UP	3	ND	mature	2-9				Uitval
UP 76 (A63)	UP	3	[F]	mature	2-9				Ben Lavin
UP 77 (A72)	UP	3	ND	mature	>20		910 ± 50 (Pta 4328)	(Steyn, 2003)	Stayt
UP 78 (A72)	UP	3	ND	immature	prenatal-1		910 ± 50 (Pta 4328)	(Steyn, 2003)	Stayt
UP 80 (A74)	UP	2	[M]	mature	>20				Cyferkuil
UP 81 (A75)	UP	3	[F]	mature	birth-9				Mayville
UP 88 (A80)	UP	2	F	mature	35-49				Phalaborwa
UP 89 (A80)	UP	2	[F]	mature	>20				Phalaborwa
UP 91 (A81)	UP	2	ND	immature	2-5	Post-1830		(Steyn, 2003)	Nandoni Dam
UP 92 (A82)	UP	3	ND	mature	6-19	Pre-1830		(Steyn, 2003)	Phalaborwa
UP 94 (A83)	UP	1	[F]	mature	35-49				Cyferkuil
UP 95 (A79)	UP	1	F	mature	35-49		160 ± 40 (Pta 8029)	(L'Abbé et al., 2008)	Pilanesberg
UP 96 (A87)	UP	3	ND	mature	>20				
UP 97 (A88)	UP	2	[M]	mature	35-49				
UP 98 (A89)	UP	3	M	mature	35-49				
UP 99 (A90)	UP	2	[F]	mature	>20				
UP 100 (A91)	UP	2	A	immature	10-34				
UP 101 (A92)	UP	2	A	mature	35-49				
UP 103 (A94)	UP	3	ND	immature	birth-5				
UP 104 (A94) [A]	UP	3	M	mature	>20				
UP 105 (A95)	UP	3	ND	immature	10-34				
UP 127 (A93)	UP	2	F	immature	10-34				
UP 130 (A98)	UP	2	M	mature	10-19				Nwanedzi

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
UP 131 (A99)	UP	3	[F]	mature	>20				Ben Alberts Nature Reserve
UP 132 (A100)	UP	2	F	mature	35-49				Ben Alberts Nature Reserve
UP 139 (A103)	UP	1	A	immature	10-34		460 ± 50 (Pta 8659)	(L'Abbé et al., 2008)	Pilanesberg
UP 141 (A105)	UP	2	ND	immature	prenatal-1	Early 19th century		(Boeyens et al., 2009)	Melora Saddle
UP 142 (A106)	UP	2	ND	immature	10-19				
UP 143	UP	1	M	mature	35-49		300 ± 35 (Pta 8944)	(L'Abbé et al., 2008)	Pilanesberg
UP 146 (A110)	UP	1	ND	immature	10-19				
UP 147	UP	3	ND	mature	>20		620 ± 40 (Pta 9304)	(L'Abbé et al., 2008)	Malatse Dam
UP 148 (A38) and (A42)	UP	2	[F]	mature	35-49				Pilanesberg
UP 149 (A38)	UP	3	ND	immature	2-5				Pilanesberg
UP 150 (in A22)	UP	3	ND	mature	>20		60 ± 40 (Pta 9458)	(L'Abbé et al., 2008)	Malatse Dam
UP 151 (A38)	UP	3	[M]	mature	>20				Pilanesberg
UP 152 (A38)	UP	3	[M]	mature	>20		670 ± 50 (Pta 9475)	(L'Abbé et al., 2008)	Malatse Dam
UP 152 (in A22)	UP	3	[M]	mature	>20		670 ± 50 (Pta 9475)	(L'Abbé et al., 2008)	Malatse Dam
UP 153 (A38) and (A42)	UP	2	[M]	mature	>20				Pilanesberg
UP ? (A108) [UP 155?]	UP	2	ND	immature	2-9				Malatse Dam
UP ? (A84)	UP	3	[M]	mature	>20				
UP ? (A85) [A]	UP	3	F	mature	>20				
UP ? (A85) [B]	UP	3	[M]	mature	>20				
UP ? (A85) [C]	UP	3	ND	mature	>20				
UP ? (A96) [A]	UP	2	F	mature	35-49				

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
UP ? (A96) [B]	UP	3	ND	immature	6-19				
UP ? (A97)	UP	2	M	mature	35-49				
UP HS 69	UP	3	ND	immature	birth-19				
UP MST /2(i) 406	UP	3	ND	mature	>20				
UP TP1 A/1 SK11	UP	3	F	mature	>20				
A 32	WITS	3	[F]	mature	>20				False Bay
A 34	WITS	3	F	mature	>20				Schweizer-Reneke
A 35	WITS	3	[F]	mature	>20				Leeudoringstad
A 37	WITS	3	A	mature	>20				
A 41	WITS	3	[F]	mature	>20				
A 47	WITS	3	[F]	mature	>20				Modder River
A 48	WITS	2	[F]	mature	>20				Gatkop Cave
A 116	WITS	3	F	mature	>20				
A 121	WITS	2	ND	mature	>20				Vegkop
A 123	WITS	2	[M]	immature	10-34				Koffiefontein
A 124	WITS	2	ND	immature	birth-5				Brits
A 125	WITS	3	ND	immature	10-19				Hole-in-the-Wall, Mqanduli
A 131	WITS	3	[F]	mature	>20				
A 133	WITS	2	A	mature	>20				Booysens
A 135	WITS	3	[M]	mature	>20				Mistkraal
A 173	WITS	1	ND	immature	10-19				Hole-in-the-Wall, Mqanduli
A 180	WITS	3	A	mature	>20				Waterloo Bay
A 189	WITS	3	ND	immature	10-19				Doornvlei
A 197	WITS	3	[F]	mature	>20				Weenen
A 198	WITS	3	F	mature	>20				Longlands
A 216	WITS	3	[M]	mature	>20				Germiston
A 222	WITS	3	[F]	mature	>20				

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
A 224	WITS	3	F	mature	>20				Dundee
A 225	WITS	3	F	mature	>20				Biggarsgat
A 233	WITS	2	ND	mature	>20		1000 ± 60 (Pta-5218)	(Lee-Thorp et al., 1993)	Heilbron
A 240	WITS	1	ND	immature	10-19				Koffiefontein
A 246	WITS	3	A	mature	>20				Heilbron
A 247	WITS	3	ND	mature	>20				Umgababa
A 254	WITS	3	F	mature	>20				
A 255	WITS	3	ND	immature	2-5				Norvalspont
A 268 (5)	WITS	3	ND	immature	2-5				Blaauwheuwel
A 268 (4)	WITS	3	ND	immature	birth-5				Blaauwheuwel
A 268 (3)	WITS	2	ND	immature	prenatal-1				Blaauwheuwel
A 268 (2)	WITS	2	ND	immature	prenatal-1				Blaauwheuwel
A 268 (1)	WITS	2	ND	immature	prenatal-1				Blaauwheuwel
A 268 (B1)	WITS	1	M	mature	>20				Blaauwheuwel
A 269	WITS	1	M	mature	35-49				Blaauwheuwel
A 272	WITS	1	F	mature	35-49				
A 274	WITS	3	A	mature	>20				
A 275	WITS	3	F	mature	>20				Ypres
A 276	WITS	3	A	mature	>20				Theunissen
A 277	WITS	3	[M]	mature	>20				Port St Johns
A 279	WITS	2	ND	immature	2-5				Groot Marico
A 294	WITS	1	A	immature	20-34				Cathkin Park

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
A 295	WITS	2	M	mature	35-49				Cathkin Park
A 298	WITS	2	ND	immature	6-19				Cathkin Park
A 299	WITS	2	M	mature	35-49				Cathkin Park
A 300	WITS	2	[M]	immature	10-34				Cathkin Park
A 301	WITS	3	ND	immature	birth-1				Cathkin Park
A 302	WITS	1	M	mature	35-49				Cathkin Park
A 303	WITS	3	ND	mature	>20				Vegkop
A 304	WITS	3	ND	mature	>20				Willowglen
A 305	WITS	3	ND	immature	2-9				Willowglen
A 306	WITS	3	ND	mature	>20				Willowglen
A 307	WITS	3	ND	immature	birth-5				Willowglen
A 312	WITS	2	ND	immature	6-19				Krugerskraal
A 319	WITS	1	M	immature	20-34				Bedford
A 320	WITS	1	F	mature	35-49				Bedford
A 321	WITS	3	F	mature	>20				
A 322	WITS	3	ND	immature	10-19				
A 326	WITS	1	F	mature	35-49				Oppermansgronde
A 327	WITS	1	F	immature	10-34				Oppermansgronde
A 328	WITS	3	ND	mature	>20				
A 330	WITS	2	[M]	mature	35-49				Jacobsdal
A 331	WITS	1	[M]	mature	20-49				Jacobsdal
A 332	WITS	2	A	mature	>20				Jacobsdal
A 333	WITS	1	[F]	mature	>20				Jacobsdal
A 334	WITS	1	A	mature	20-49				Jacobsdal
A 336	WITS	3	ND	immature	birth-19				

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
A 349	WITS	3	[M]	mature	>20	1860-1870		(Morris, 1992a)	East London
A 350	WITS	3	F	mature	>20	1860-1870		(Morris, 1992a)	East London
A 351	WITS	2	F	mature	>20				Signal Hill, East London
A 352	WITS	2	F	immature	20-34				Signal Hill, East London
A 354	WITS	3	ND	immature	2-9				Signal Hill, East London
A 355	WITS	1	F	mature	35-49				Signal Hill, East London
A 357	WITS	2	ND	immature	10-19				Signal Hill, East London
A 358 [A]	WITS	3	F	mature	>20				Signal Hill, East London
A 359	WITS	3	[F]	mature	>20				Signal Hill, East London
A 360	WITS	3	ND	immature	10-19				Signal Hill, East London
A 361	WITS	3	ND	mature	>20				East London
A 362	WITS	3	ND	mature	>20				East London
A 363	WITS	3	ND	mature	>20				East London
A 364	WITS	2	F	mature	35-49				Buffalo River, East London
A 365	WITS	3	ND	immature	2-9				East London
A 366	WITS	1	[M]	mature	>35	1840-1870		(Morris, 1992a)	East London

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
A 369	WITS	3	ND	immature	10-19		150 ± 50 (Pta 5217)	(Lee-Thorp et al., 1993)	Irene Cave
A 370	WITS	3	ND	mature	>20				Irene Cave
A 372	WITS	3	[M]	mature	>20				Irene Cave
A 373	WITS	3	[F]	mature	>20				
A 374	WITS	3	A	mature	>20				
A 376 [box says 371]	WITS	2	ND	mature	>20				
A 377	WITS	3	A	mature	>20				
A 378	WITS	3	F	mature	>20				
A 379	WITS	1	M	mature	>35	1880		(Morris, 1992a)	Florisbad
A 385	WITS	3	ND	immature	birth-1				Makapansgat Valley
A 386	WITS	2	F	mature	35-49				Karridene
A 389	WITS	2	[M]	mature	>20	1865			Ficksburg
A 390	WITS	3	[F]	mature	>20				
A 391	WITS	3	ND	mature	>20				Tsitsikamma
A 392	WITS	3	M	mature	>20				Rustenburg
A 403 [A]	WITS	2	M	mature	20-34				Lindequesdrif
A 407	WITS	3	A	mature	>20				Lindequesdrif
A 410 [B]	WITS	3	[F]	mature	>20				Nuwe Smitsdorp
A 410 [C]	WITS	3	F	mature	>20				Nuwe Smitsdorp
A 414 [B]	WITS	3	F	mature	>20		600 ± 50 (Pta 4973)	(Morris, 1992a)	Ellerton Mine
A 415 [B]	WITS	3	[F]	mature	>20				Koppie Enkel
A 416	WITS	2	[F]	immature	10-34				Ellerton Mine
A 417	WITS	3	ND	immature	birth-1				Umhloti
A 418	WITS	2	ND	immature	2-9				Leydsdorp
A 419	WITS	3	A	mature	>20				
A 426	WITS	3	A	mature	>20				Swartvlei
A 575	WITS	3	[F]	mature	>20				Kuruman

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
A 606	WITS	3	F	mature	>20				Illovo
A 609 [A]	WITS	2	ND	mature	>20				Tinley Manor
A 609 [B]	WITS	3	ND	immature	2-9				Tinley Manor
A 610	WITS	3	ND	mature	>20				
A 611 [A]	WITS	3	[F]	mature	>20				Molteno
A 611 [B]	WITS	3	A	mature	2-9				Molteno
A 612	WITS	3	ND	immature	10-19				
A 613	WITS	3	F	mature	>20				Buispoort
A 616 [A]	WITS	2	ND	mature	>20				
A 616 [B]	WITS	1	M	mature	35-49				
A 616 [C]	WITS	2	[F]	mature	>20				
A 616 A	WITS	3	[F]	mature	>20				Bekkersfontein
A 616 B	WITS	3	A	mature	>20				Fauresmith
A 616 C	WITS	3	A	mature	>20				Philippolis
A 616 D	WITS	3	[F]	mature	>20				Kroonstad
A 627	WITS	1	M	mature	35-49				Windsorton
A 628	WITS	3	ND	immature	6-19				Ventersdorp
A 629	WITS	2	[M]	mature	>20				Bloemhof
A 630 A	WITS	3	A	mature	>20				Umhlali
A 630 B	WITS	3	[F]	mature	>20				Umhlali
A 630 C	WITS	3	[F]	mature	>20				Umhlali
A 631	WITS	3	ND	immature	birth-19				Pacaltsdorp
A 635	WITS	1	F	immature	10-34		2532 ± 26 (OxA-V-2064-55)	(Ribot et al., 2010)	Richards Bay
A 653	WITS	3	[F]	mature	>20				Tsitsikamma
A 654	WITS	3	A	mature	>20				Pretoria North
A 671	WITS	3	ND	immature	birth-5				Victoria West
A 672	WITS	3	F	mature	>20				Middelkraal
A 813	WITS	3	[F]	mature	>20				Reitz
A 870	WITS	3	[F]	mature	>20				Ingwavuma

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
A 910	WITS	1	ND	immature	10-19				Port St Johns
A 912	WITS	3	F	mature	>20				Gansbaai
A 914	WITS	3	A	mature	>20				Blaauwkrantz
A 915	WITS	3	ND	immature	10-19				
A 996	WITS	2	[M]	mature	35-49	1904-1910		(Dart, 1952; Gibbon et al., 2010; Meyer et al., 2013)	Witwatersrand Deep Mine
A 997	WITS	1	M	immature	20-34	1904-1910		(Dart, 1952; Gibbon et al., 2010; Meyer et al., 2013)	Witwatersrand Deep Mine
A 998	WITS	2	M	mature	>20	1904-1910		(Dart, 1952; Gibbon et al., 2010; Meyer et al., 2013)	Witwatersrand Deep Mine
A 999	WITS	2	M	immature	10-34	1904-1910		(Dart, 1952; Gibbon et al., 2010; Meyer et al., 2013)	Witwatersrand Deep Mine
A 1000	WITS	2	M	immature	10-34	1904-1910		(Dart, 1952; Gibbon et al., 2010; Meyer et al., 2013)	Witwatersrand Deep Mine
A 1001	WITS	1	M	mature	10-34	1904-1910		(Dart, 1952; Gibbon et al., 2010; Meyer et al., 2013)	Witwatersrand Deep Mine
A 1002	WITS	2	[M]	mature	>20	1904-1910		(Dart, 1952; Gibbon et al., 2010; Meyer et al., 2013)	Witwatersrand Deep Mine

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
A 1003	WITS	1	M	mature	35-49	1904-1910		(Dart, 1952; Gibbon et al., 2010; Meyer et al., 2013)	Witwatersrand Deep Mine
A 1004	WITS	1	[M]	immature	10-34	1904-1910		(Dart, 1952; Gibbon et al., 2010; Meyer et al., 2013)	Witwatersrand Deep Mine
A 1005	WITS	1	A	immature	10-34	1904-1910		(Dart, 1952; Gibbon et al., 2010; Meyer et al., 2013)	Witwatersrand Deep Mine
A 1006	WITS	2	[M]	mature	20-49	1904-1910		(Dart, 1952; Gibbon et al., 2010; Meyer et al., 2013)	Witwatersrand Deep Mine
A 1007	WITS	1	M	mature	35-49	1904-1910		(Dart, 1952; Gibbon et al., 2010; Meyer et al., 2013)	Witwatersrand Deep Mine
A 1008	WITS	1	[M]	mature	35-49	1904-1910		(Dart, 1952; Gibbon et al., 2010; Meyer et al., 2013)	Witwatersrand Deep Mine
A 1009	WITS	1	[M]	immature	10-34	1904-1910		(Dart, 1952; Gibbon et al., 2010; Meyer et al., 2013)	Witwatersrand Deep Mine
A 1010	WITS	2	[M]	mature	10-34	1904-1910		(Dart, 1952; Gibbon et al., 2010; Meyer et al., 2013)	Witwatersrand Deep Mine

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
A 1011	WITS	1	[M]	immature	20-34	1904-1910		(Dart, 1952; Gibbon et al., 2010; Meyer et al., 2013)	Witwatersrand Deep Mine
A 1012	WITS	2	A	mature	>20	1904-1910		(Dart, 1952; Gibbon et al., 2010; Meyer et al., 2013)	Witwatersrand Deep Mine
A 1013	WITS	1	[M]	mature	20-34	1904-1910		(Dart, 1952; Gibbon et al., 2010; Meyer et al., 2013)	Witwatersrand Deep Mine
A 1014	WITS	2	[M]	mature	20-34	1904-1910		(Dart, 1952; Gibbon et al., 2010; Meyer et al., 2013)	Witwatersrand Deep Mine
A 1015	WITS	1	[M]	immature	10-34	1904-1910		(Dart, 1952; Gibbon et al., 2010; Meyer et al., 2013)	Witwatersrand Deep Mine
A 1016	WITS	2	[M]	mature	>20	1904-1910		(Dart, 1952; Gibbon et al., 2010; Meyer et al., 2013)	Witwatersrand Deep Mine
A 1017	WITS	2	[M]	mature	>20	1904-1910		(Dart, 1952; Gibbon et al., 2010; Meyer et al., 2013)	Witwatersrand Deep Mine
A 1018	WITS	2	[M]	mature	20-34	1904-1910		(Dart, 1952; Gibbon et al., 2010; Meyer et al., 2013)	Witwatersrand Deep Mine

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
A 1019	WITS	2	[M]	immature	10-34	1904-1910		(Dart, 1952; Gibbon et al., 2010; Meyer et al., 2013)	Witwatersrand Deep Mine
A 1020	WITS	2	ND	immature	10-19	1904-1910		(Dart, 1952; Gibbon et al., 2010; Meyer et al., 2013)	Witwatersrand Deep Mine
A 1021	WITS	2	M	mature	>20	1904-1910		(Dart, 1952; Gibbon et al., 2010; Meyer et al., 2013)	Witwatersrand Deep Mine
A 1022	WITS	1	M	immature	10-34	1904-1910		(Dart, 1952; Gibbon et al., 2010; Meyer et al., 2013)	Witwatersrand Deep Mine
A 1023	WITS	1	[M]	mature	35-49	1904-1910		(Dart, 1952; Gibbon et al., 2010; Meyer et al., 2013)	Witwatersrand Deep Mine
A 1024	WITS	3	ND	mature	>20	1904-1910		(Dart, 1952; Gibbon et al., 2010; Meyer et al., 2013)	Witwatersrand Deep Mine
A 1025	WITS	3	[M]	immature	10-34	1904-1910		(Dart, 1952; Gibbon et al., 2010; Meyer et al., 2013)	Witwatersrand Deep Mine
A 1026	WITS	3	A	immature	10-34	1904-1910		(Dart, 1952; Gibbon et al., 2010; Meyer et al., 2013)	Witwatersrand Deep Mine

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
A 1027	WITS	2	A	mature	20-34	1904-1910		(Dart, 1952; Gibbon et al., 2010; Meyer et al., 2013)	Witwatersrand Deep Mine
A 1028	WITS	2	[M]	mature	20-34	1904-1910		(Dart, 1952; Gibbon et al., 2010; Meyer et al., 2013)	Witwatersrand Deep Mine
A 1029	WITS	2	A	immature	10-34	1904-1910		(Dart, 1952; Gibbon et al., 2010; Meyer et al., 2013)	Witwatersrand Deep Mine
A 1030	WITS	1	[M]	immature	10-34	1904-1910		(Dart, 1952; Gibbon et al., 2010; Meyer et al., 2013)	Witwatersrand Deep Mine
A 1031	WITS	2	A	immature	10-34	1904-1910		(Dart, 1952; Gibbon et al., 2010; Meyer et al., 2013)	Witwatersrand Deep Mine
A 1032	WITS	3	F	mature	>20				Cape Peninsula
A 1048	WITS	3	F	mature	>20				Graaff-Reinet
A 1049	WITS	3	F	mature	>20				Ladysmith
A 1050	WITS	3	ND	immature	10-19				
A 1051	WITS	3	F	mature	>20				
A 1052	WITS	2	ND	immature	10-19				Holley Shelter
A 1053	WITS	2	F	immature	10-34				Holley Shelter
A 1054	WITS	1	[F]	mature	>35				Makapansgat Valley
A 1055	WITS	1	M	immature	20-34		1080 ± 40 (Pta 6955)	(Sealy and Pfeiffer, 2000)	Keurbooms River

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
A 1058	WITS	3	ND	immature	10-19				Margate
A 1059	WITS	3	ND	mature	>20				Bloemfontein
A 1060	WITS	3	ND	immature	10-19		1025 ± 35 (OxA-6220)	(Ribot et al., 2010)	Umgababa
A 1061	WITS	3	[M]	mature	>20				
A 1062	WITS	3	F	mature	>20				
A 1066	WITS	3	[M]	mature	>20				
A 1069	WITS	1	F	mature	35-49				De Aar
A 1071	WITS	2	[M]	mature	>20		1320 ± 50 (Pta-6997)	(Ribot et al., 2010)	Mahlongwana River
A 1072	WITS	2	[M]	mature	>20				
A 1075	WITS	3	M	mature	>20				Mapochs Mine
A 1076	WITS	3	[M]	mature	>20				Roossenekal
A 1077	WITS	3	F	immature	10-34				Pilgrimsrest
A 1078	WITS	3	[M]	mature	>20				Pilgrimsrest
A 1079	WITS	3	F	mature	>20				Pilgrimsrest
A 1080	WITS	3	[M]	mature	>20				Pilgrimsrest
A 1084	WITS	2	ND	immature	birth-5				Heilbron
A 1085	WITS	3	ND	mature	>20				Umgababa
A 1086	WITS	1	ND	immature	6-19				Polokwane
A 1093 [A]	WITS	2	M	mature	35-49				
A 1093 [B]	WITS	2	ND	immature	2-5				
A 1095 [A]	WITS	2	F	mature	35-49				Rietspruit
A 1095 [B]	WITS	2	ND	immature	2-9				Rietspruit
A 1101 [A]	WITS	2	A	mature	35-49				eNtokozweni
A 1101 [B]	WITS	3	A	mature	>20				East London
A 1103	WITS	2	[F]	mature	>20				Wellington Estates
A 1104	WITS	2	ND	mature	>20				Wellington Estates
A 1105	WITS	3	ND	mature	>20				
A 1107	WITS	2	M	mature	20-49				Port St Johns

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
A 1110 [A]	WITS	3	[F]	mature	>20				Zuurberg
A 1110 [B]	WITS	3	[M]	mature	>20				Zuurberg
A 1110 [C]	WITS	3	F	mature	>20				Zuurberg
A 1112 [A]	WITS	3	F	mature	>20				Lottering River, Tsitsikamma
A 1112 [B]	WITS	3	A	mature	>20		3355 ± 45 (GrA-23232)	(Stynder et al., 2007a)	Lottering River, Tsitsikamma
A 1112 [C]	WITS	3	M	mature	>20				
A 1112 [D]	WITS	2	F	mature	>50		2160 ± 50 (Pta 6949)	(Sealy and Pfeiffer, 2000)	Lottering River, Tsitsikamma
A 1113	WITS	3	[M]	mature	>20				Knysna
A 1114	WITS	3	F	mature	>20		2271 ± 33 (OxA-V-2055-51)	(Stynder et al., 2007a)	Knysna
A 1115	WITS	3	A	mature	>20				Knysna
A 1117	WITS	1	F	mature	20-34		1060 ± 50 (Pta 8727)	(Stynder et al., 2007a)	Limebank, Loerie
A 1119	WITS	1	ND	immature	10-19				Blue Cliff
A 1121	WITS	3	F	mature	>20				Don Pedro Jetty
A 1122	WITS	3	A	mature	>20				Don Pedro Jetty
A 1123	WITS	3	[F]	mature	>20				Keurbooms River
A 1124	WITS	3	[M]	mature	>20		4320 ± 32 (OxA-V-2056-42)	(Stynder et al., 2007a)	Port Elizabeth
A 1125	WITS	3	F	mature	>20				Port Elizabeth
A 1126	WITS	3	[F]	mature	>20				Port Elizabeth
A 1127	WITS	3	F	mature	>20		1891 ± 29 (OxA-V-2066-36)	(Stynder et al., 2007a)	Jeffreys Bay
A 1128	WITS	3	ND	immature	6-19				Port Elizabeth
A 1129	WITS	3	[F]	mature	>20				Port Elizabeth

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
A 1130	WITS	3	M	mature	>20				Port Elizabeth
A 1131	WITS	3	[M]	mature	>20				Humewood
A 1132	WITS	2	F	mature	>20				Port Elizabeth
A 1133	WITS	2	ND	immature	2-9				Lottering River, Tsitsikamma
A 1134	WITS	3	[M]	mature	>20				Port Elizabeth
A 1135	WITS	3	ND	immature	6-19				Korsten
A 1136	WITS	3	M	mature	>20				Dunbrody
A 1137	WITS	3	M	mature	>20				Baakens River
A 1138	WITS	2	A	immature	10-34				Seaview
A 1139	WITS	2	[F]	immature	10-34		4800 ± 50 (Pta 8816)	(Stynder et al., 2007a)	Kenkelbosch
A 1140	WITS	3	ND	mature	>20				Townlands, Pietermaritzburg
A 1141 [B]	WITS	3	[F]	mature	>20				Lottering River, Tsitsikamma
A 1141 [D]	WITS	3	[F]	mature	>20				Lottering River, Tsitsikamma
A 1141 [E]	WITS	3	[F]	mature	>20				Lottering River, Tsitsikamma
A 1141 [F]	WITS	3	A	mature	>20				Lottering River, Tsitsikamma
A 1143	WITS	3	[F]	mature	>20				Lottering River, Tsitsikamma
A 1144	WITS	3	ND	immature	2-5				Lottering River, Tsitsikamma
A 1145	WITS	3	ND	immature	2-5				Lottering River, Tsitsikamma

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
A 1146	WITS	3	F	mature	>20				Lottering River, Tsitsikamma
A 1147	WITS	3	ND	immature	10-19				Lottering River, Tsitsikamma
A 1148	WITS	3	ND	immature	6-19				Lottering River, Tsitsikamma
A 1149	WITS	3	F	mature	>20				Lottering River, Tsitsikamma
A 1150	WITS	3	ND	immature	birth-19				Boesmansrivier
A 1151	WITS	3	ND	immature	6-19				Steytlerville
A 1152	WITS	2	F	mature	>20		1850 ± 35 (Pta 8757)	(Stynder et al., 2007a)	Amsterdam Hoek
A 1153	WITS	3	F	mature	>20		636 ± 26 (OxA-V-2065-47)	(Stynder et al., 2007a)	Steytlerville
A 1154	WITS	3	A	mature	>20		905 ± 25 (OxA-V-2066-33)	(Stynder et al., 2007a)	Steytlerville
A 1155	WITS	3	[M]	mature	>20		190 ± 45 (Pta 6980)	(Sealy and Pfeiffer, 2000)	Tsitsikamma
A 1156	WITS	3	A	mature	>20				Happy Valley
A 1157	WITS	3	M	mature	>20				New Brighton
A 1158	WITS	3	F	mature	>20				
A 1159	WITS	1	F	mature	20-34				Klipplaat
A 1160	WITS	3	A	mature	>20				Schoenmakerskop
A 1161	WITS	3	F	mature	>20				
A 1162	WITS	3	ND	immature	birth-9				Port Elizabeth
A 1163	WITS	3	ND	immature	10-19				Port Elizabeth

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
A 1164	WITS	3	ND	immature	birth-19				Port Elizabeth
A 1165	WITS	3	M	mature	>20				Port Elizabeth
A 1166	WITS	3	F	mature	>20		1818 ± 27 (OxA-V-2056-33 A)	(Stynder et al., 2007a)	Humewood
A 1167	WITS	3	A	mature	>20				Selborne
A 1168	WITS	3	[F]	mature	>20				Port Elizabeth
A 1169	WITS	3	ND	immature	6-19				Whitcher's Cave
A 1170	WITS	3	ND	immature	6-9				Whitcher's Cave
A 1171	WITS	3	[F]	mature	>20				Whitcher's Cave
A 1172 [A]	WITS	1	ND	immature	6-19				Whitcher's Cave
A 1172 [B]	WITS	3	[F]	mature	>20		2950 ± 40 (GrA-23647)	(Stynder et al., 2007a)	Whitcher's Cave
A 1173	WITS	3	F	mature	>20				Whitcher's Cave
A 1174	WITS	3	A	mature	>20				Whitcher's Cave
A 1175	WITS	3	[M]	mature	>20				Whitcher's Cave
A 1176	WITS	3	F	mature	>20				Whitcher's Cave
A 1177	WITS	3	[M]	mature	>20				Whitcher's Cave
A 1178	WITS	3	ND	immature	birth-19				
A 1179	WITS	3	F	mature	>20				
A 1180	WITS	3	[F]	mature	>20				
A 1181	WITS	3	[F]	mature	>20				
A 1182	WITS	3	A	mature	>20				
A 1183	WITS	3	[M]	mature	>20				
A 1186	WITS	2	F	mature	20-49		2880 ± 60 (Pta 6953)	(Sealy and Pfeiffer, 2000)	Whitcher's Cave
A 1194	WITS	2	M	mature	35-49				
A 1195	WITS	3	A	mature	>20				London
A 1217	WITS	3	A	mature	>20				Keurbooms River

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
A 1648	WITS	3	F	mature	35-49				
A 1649	WITS	3	ND	mature	>20				
A 1654	WITS	3	[F]	mature	>20				Mossel Bay
A 1655	WITS	3	ND	immature	2-5				Mossel Bay
A 1679	WITS	3	[M]	mature	>20				
A 1756	WITS	3	[F]	mature	>20				
A 1778 [A]	WITS	3	ND	immature	birth-19				Elliotdale
A 1778 [B]	WITS	3	[F]	mature	>20				Elliotdale
A 1835	WITS	2	[F]	mature	35-49				Makapansgat Farm
A 1836	WITS	1	F	mature	35-49				Wellington Estates
A 1837	WITS	2	A	mature	>20				Wellington Estates
A 1838 [A]	WITS	2	A	immature	10-19				Wellington Estates
A 1839	WITS	3	[F]	mature	>20				Makapansgat Valley
A 1920	WITS	3	F	mature	>20				
A 1956	WITS	3	ND	mature	>20				Glory Hill
A 1957	WITS	3	A	mature	>20				
A 1968	WITS	3	ND	immature	2-9				
A 1969	WITS	3	ND	immature	2-9				Wellington Estates
A 1983	WITS	3	ND	immature	2-19				
A 1984	WITS	3	ND	immature	6-19				
A 2002	WITS	3	F	mature	2-9				
A 2003	WITS	3	M	mature	35-49				Ferreira Town, Jeffreys Bay
A 2004	WITS	3	ND	immature	6-19				Barrage
A 2005	WITS	2	A	mature	35-49				
A 2034	WITS	3	ND	immature	6-9				Durban
A 2036	WITS	3	A	mature	>20				Amanzimtoti
A 2041	WITS	3	ND	mature	>20				Northcliff
A 2065	WITS	1	M	mature	20-34				
A 2083	WITS	2	ND	immature	birth-5				

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
A 2120	WITS	2	F	immature	10-34		940 ± 20 (Pta 6108)	(Ribot et al., 2010)	Ballito Bay
A 2121	WITS	2	ND	immature	2-5				Ballito Bay
A 2123	WITS	3	ND	immature	6-19				Makapansgat Valley
A 2124	WITS	2	[F]	mature	>20				Makapansgat Valley
A 2142 [A 2042]	WITS	3	ND	mature	>20				
A 2190	WITS	3	[F]	mature	>20				Kinross
A 2246	WITS	2	A	mature	>20				Bedfordview
A 2260	WITS	3	ND	immature	2-19				Makapansgat Valley
A 2274	WITS	3	[F]	mature	35-49				Ohrigstad
A 2275	WITS	3	ND	immature	10-19				Ohrigstad
A 2380	WITS	3	ND	mature	>20				Makapansgat Valley
A 2745	WITS	2	[F]	immature	10-34				Phalaborwa
A 2814	WITS	3	ND	mature	>20				
A 2836 (H852 / 805)	WITS	2	ND	immature	2-5	early to mid 19th century		(Morris, 1992a, 1984)	Campbell
A 2844	WITS	1	[M]	mature	20-49				Rietpan
A 2852	WITS	2	ND	immature	2-9				Olifantspoort
A 2853	WITS	3	ND	mature	>20				Olifantspoort
A 2854	WITS	2	F	mature	>35				Olifantspoort
A 2855	WITS	2	ND	immature	6-19				Olifantspoort
A 2856	WITS	2	F	mature	35-49	AD 1500		(Mason, 1986)	Olifantspoort
A 2857	WITS	3	ND	mature	>20				Olifantspoort
A 2859	WITS	2	ND	immature	2-9				Rietpan

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
A 2904	WITS	3	ND	mature	>20				Galpin's farm / Mosdene
A 4141	WITS	2	F	mature	20-49				Bambo Hills
A 4143	WITS	2	A	mature	>20				Broederstroom
A 4146	WITS	1	M	mature	35-49	1818-1835 AD		(Beaumont, 1967)	Brotherton Shelter
A 4151	WITS	3	A	mature	>20				Eiland
A 4152	WITS	1	F	mature	20-49	?15th Century [Midden above dung and skeleton dated to 1490±50 AD, grave dug into dung layer]		(Moore, 1981)	Makapansgat Valley
A 4157	WITS	3	ND	mature	>20		650 ± 40 (Pta-1343)	(Mason, 1981)	Broederstroom
A 4208	WITS	2	[M]	mature	35-49	18th-19th century AD		(Mason, 1986, 1968)	Klipriviersberg
A 4209	WITS	2	ND	immature	10-19	18th-19th century AD		(Mason, 1986, 1968)	Klipriviersberg
A 4212	WITS	2	A	mature	>20				
A 4213	WITS	2	ND	immature	10-19				
A 4214	WITS	2	[F]	mature	35-49				
A 4220	WITS	1	A	immature	20-34				Nylsvley

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
A 4221	WITS	2	F	mature	>20	19th century AD		(Henneberg and Steyn, 1993; Mason, 1986)	Olifantspoort
A 4222	WITS	3	ND	mature	>20				Oppiesmariesdrift
A 4223	WITS	2	M	mature	>20				
A 4224	WITS	2	[F]	mature	>20				Queen's Hill
A 4226	WITS	1	M	mature	35-49	mid-17th century AD		(Hall, 1985)	Rooikrans
A 4227	WITS	1	F	mature	>35	mid-17th century AD		(Hall, 1985)	Rooikrans
A 4228	WITS	2	M	mature	35-49				Tavhatshena
A 4229	WITS	1	[F]	mature	>20				Tavhatshena
A 4230	WITS	1	[M]	mature	>20	mid-17th century AD		(Hall, 1985)	Rooikrans
A 4231	WITS	2	M	mature	>35				Clarens
A 4248	WITS	3	[F]	mature	35-49				
A 4249	WITS	2	[F]	mature	>20				Beryl
A 4251	WITS	2	A	mature	>20				Strathmore Mine
A 4253 [A]	WITS	3	[M]	mature	>20				
A 4253 [B]	WITS	3	ND	immature	2-9				
A 4253 [C]	WITS	3	[M]	mature	>20				
A 4253 [D]	WITS	3	[F]	mature	>20				
A 4253 [E]	WITS	3	ND	immature	2-19				
A 4253 [F]	WITS	3	ND	immature	2-19				
A 4255 (I)	WITS	3	[F]	mature	>20				Lindley
A 4255 (II)	WITS	3	[F]	mature	>20				Lindley
A 4255 (III)	WITS	3	A	mature	>20				Lindley

Accession numbers	Institution	Total preservation score[a]	Sex[b]	Binary age[c]	Broad age (yrs)	Date: General	Date: Radiocarbon	Publications[d]	Location
A 4255 (IV)	WITS	3	M	mature	>20				Lindley
A 4255 (V)	WITS	3	A	mature	>20				Lindley
A 4256	WITS	2	A	mature	35-49				
A 4257	WITS	2	F	mature	>20				
A 4258	WITS	3	[F]	mature	>20				
A 4260	WITS	1	ND	immature	10-19	LIA		(de Villiers, 1991)	Vhuneyla
A 4277	WITS	2	M	mature	35-49				

[a] Total preservation score: 1 = more than 75% of the skeleton is present, 2 = 25-74% of the skeleton is present, 3 = less than 25% of the skeleton is present.

[b] Sex: F = female, [F] = probably female, A = ambiguous, M = male, [M] = probably male, ND = not determined.

[c] Age: mature = all epiphyses fused, immature = epiphyses not formed, not fused or fusing.

[d] Publication relevant to chronological dates.