Dental pathology, wear and developmental defects in South African hominins

IAN EDWARD TOWLE

A thesis submitted in partial fulfilment of the requirements of Liverpool John Moores University for the degree of Doctor of Philosophy

June 2017

Abstract

Studying different types of dental pathology, wear, and developmental defects can allow inferences into diet and behaviour in a variety of ways. In this project data on these different variables were collected for South African hominins and compared with extant primates. The species studied include *Paranthropus robustus*, *Australopithecus africanus*, *A. sediba*, early *Homo*, *Homo naledi*, baboons, chimpanzees and gorillas. Macroscopic examination of each specimen was performed, with a 10X hand lens used to verify certain pathologies. Variables recorded include antemortem chipping, enamel hypoplasia, caries, occlusal wear, tertiary dentine, abscesses, and periodontal disease. Clear differences in frequencies were found in the different South African hominin species. *Homo naledi* displays high rates of chipping, especially small fractures above molar wear facets, likely reflecting a diet containing high levels of contaminants. Other noteworthy results include the high levels of pitting enamel hypoplasia in *P. robustus* molars compared to other species, likely due to a species-specific enamel formation property or developmental disturbance. The low rates of chipping in *P. robustus* does not fit with this species being a hard food specialist. Instead, the wear best supports a diet of low-quality tough vegetation. *Australopithecus africanus* likely had a broad diet, with angled molar wear, lack of caries, and high chipping frequencies supporting this conclusion. Seven new carious lesions are described, two from *H. naledi* and five *P. robustus*. Other, rarer, pathologies are also highlighted, including abscesses in an early *Homo* individual, root grooves caused by erosive wear in *A. africanus* and a case of amelogenesis imperfecta in a female chimpanzee. The main conclusion from this thesis as a whole is the substantial difference in frequencies of the different variables among hominin species, supporting the proposition that their diets differed substantially.

Acknowledgments

First and foremost I want to thank my director of studies Joel Irish for the opportunity to complete this research and for all his help and encouragement during the course of this project. I would also like to thank my second supervisor Isabelle De Groote for her guidance and advice over the last three years. Without both supervisor's continuous support this thesis would not have been possible.

I am indebted to various museum curators and researchers who were tremendously helpful during data collection. In particular, I would like to thank Lee Berger and Bernhard Zipfel from the University of the Witwatersrand, Stephany Potze from Ditsong Museum of South Africa and Inbal Livne from the Powell-Cotton museum. I would also like to thank John Hawks, Lucas Delezene, Jacopo Moggi-Cecchi, Sandra Mathews, and Martin Hausler for their help and hospitality during these trips. I am hugely thankful to Marina Elliott for taking additional photos and descriptions of specimens once I was back in the UK and to Matthew Skinner for access to micro-CT scans.

I gratefully acknowledge Liverpool John Moores University for providing funding for this project. This was in the form of a stipend and lab equipment but also travel bursaries which allowed me to attend and present my work at conferences. I am also appreciative of the administrative staff, including Gill Beesley and David Jones, who helped with travel arrangements and purchasing equipment. I would like to thank my colleagues at Liverpool John Moores University, in particular the fantastic group of postgraduates who made this PhD so enjoyable. As well as my main project I have been fortunate enough to undertake side projects, for which I am extremely grateful to Eleanor Dove, Alessio Veneziano and Cal Davenport for these collaborations.

I am extremely thankful to my family for all their support and encouragement. Lastly, I am eternally grateful to Clare Davies for her encouragement and support both before and during this project.

Contents

List of Figures

List of Tables

Glossary

ATML: Antemortem Tooth Loss

PEH: Pitting Enamel Hypoplasia

LEH: Linear Enamel Hypoplasia

CEJ: Cementoenamel Junction

EDJ: Dental-Enamel Junction

Chapter 1: Introduction

Dental pathologies, developmental defects, and different types of wear can give insight into the diet, behaviour and health of individuals and groups. In this project, data on these various indicators are presented for South African fossil hominins with the aim of providing further insight into diet and the ecological niche these species may have filled. Profiles for each species are created using these different dental components followed by comparisons with one another, as well as extant primates and modern humans. Lastly, these frequencies are likened to those from other dietary-reconstructing techniques, such as dental morphology, isotopic analysis, and microwear.

Data was collected on dental caries, ante-mortem chipping, tertiary dentine, enamel hypoplasia, periodontal disease, abscesses, malocclusion, attrition, and unusual wear. Other, rarer, pathologies are also noted. The hominin material studied includes specimens assigned to *Paranthropus robustus*, *Australopithecus africanus, Australopithecus sediba, Homo naledi* and 'early *Homo'*. These specimens are curated at Ditsong Museum and the University of the Witwatersrand in South Africa. The comparative primate sample consists of chimpanzees (*Pan troglodytes*), gorillas (*Gorilla gorilla gorilla*), drills (*Mandrillus leucophaeus*), and baboons (*Papio anubis* and *Papio hamadryas*), curated at the Powell-Cotton Museum in the UK, and medieval and Roman era human samples that are curated at Liverpool John Moores University. The common names for the extant primate samples are used in the rest of this thesis as a quick way to differentiate them from the fossil hominins.

It is often cited that dental enamel is the hardest tissue in the human body (Duverger et al., 2016). The high mineral content of this material, as well as other dental tissue, has led to exceptional levels of preservation in the archaeological and fossil records. Indeed, the majority of hominin fossils are isolated teeth, although there are some very well preserved examples in which an entire jaw is present. Two other characteristics of teeth make it very fortunate they preserve so well in the fossil record. Firstly, once enamel forms in childhood, it stays virtually unchanged, barring pathologies and wear, meaning that any defects offer a snapshot of the individual's development history (Guatelli-Steinberg, 2015; Hillson, 2014).

Secondly, the oral cavity is directly exposed to the environment in which these early hominins lived, meaning that a unique insight into their way of life can be discerned.

1.1. Structure of thesis

In the succeeding chapters, different types of pathology, wear, and developmental defects are dealt with separately within their own chapter, followed by an overall discussion. A concise materials and methods section precedes these, with more in-depth description and explanations to why certain methods were used are detailed in the appropriate chapter. First, however, a brief overview of the material is presented as well as an overview of the literature to give a framework on which this work hopes to build. The chapters, therefore, are as followed, introduction (Chapter 1), sites and species (Chapter 2), dental pathology, developmental defects and wear (Chapter 3), materials and methods (Chapter 4), chipping (Chapter 5), caries (Chapter 6), enamel hypoplasia (Chapter 7), macro-wear (Chapter 8), abscesses, periodontal disease and antemortem tooth loss (Chapter 9), tertiary dentine (Chapter 10), case studies (Chapter 11), discussion, conclusions and future research (Chapter 12).

Chapter 2 outlines the hominin material found in the cradle of humankind as well as when and how particular sites and specimens were discovered. Additionally, this chapter outlines the species represented along with information on preservation and taphonomy. Chapter 3 reviews the literature with focus on dental pathology and wear research already carried out on South African hominins. Other types of dietary-reconstructing methods are also outlined and discussed. Chapter 4 gives an overview of the methods used for each variable and gives a summary of the specimens studied, and their condition, for each site and species. Chapters 5 to 10 each focus on one variable (except Chapter 9 which has three) and are structured similar to a journal article, with separate introduction, materials and methods, results, and discussion. Chapter 11 highlights a couple of rare lesions and defects, structured as two separate case studies. Chapter 12 compares the results of the previous chapters and then gives an overview of what these results may mean in terms of diet and behaviour for each of the species studied. The main findings are then highlighted and a brief overview given on how these results can be built upon.

1.2. Justification of research

In this project dental pathologies, wear and developmental defects were recorded in *H. naledi* for the first time, and therefore a new insight into this intriguing species can be given. Additionally, although individual studies have looked at particular indicators in *P. robustus* and *A. africanus*, broad-scale comparisons of multiple pathologies and developmental defects have not yet been conducted. A brief outline on the significance of this research for each species is outlined below.

1.2.1. Homo naledi

The recently discovered material assigned to a new hominin species, *H. naledi*, has not been subjected to the decades of research that *P. robustus* and *A. africanus* specimens have. However, the open access and large-scale nature of the project, led by Professor Lee Berger, means a surprisingly large amount of research has already been conducted (Berger et al., 2015; Cofran et al., 2016; Feuerriegel et al., 2016; Harcourt-Smith et al., 2015; Kivell et al., 2015; M.M. Skinner et al., 2016; Williams et al., 2017). Microwear, photolith and enamel property analysis are all being conducted and will be able to be compared with the data from this project. Additionally, dental morphology and enamel property analysis have already been performed (Berger et al., 2015; Hawks et al., 2017; M.M. Skinner et al., 2016). The large sample size of this material means different frequencies of pathologies can be compared to other hominins and extant primates, making dietary inferences possible. The small and more humanlike dentition of these individuals compared to most of the other South African material means it will be fascinating to gain any information on their diet and behaviour, but also the stresses that they faced (Berger et al., 2015; Hawks et al., 2017). Comparisons between rates of dental pathologies and wear will be made with the other South African hominins, but also, given its classification into the genus *Homo*, with other *Homo* specimens in the literature.

1.2.2. Australopithecus sediba

The sample size of *A. sediba* is much smaller than the other species studied in this project, meaning large-scale species comparisons for different pathologies is difficult. Nonetheless, these remarkably well-preserved specimens still offer a fascinating insight into the behaviour of these unique hominins, with occlusal wear particularly informative. In recent

years unusual pathologies have been described on other skeletal elements of these individuals, and therefore it will be interesting to see if there are any dental indicators of particular pathologies (Randolph-Quinney et al., 2016; Williams et al., 2013).

1.2.3. Paranthropus robustus

The robust masticatory apparatus of *P. robustus* has led many researchers to suggest such morphology must have been an adaptation to a highly specialised diet (Daegling et al., 2011; Scott et al., 2005). The suggestions have, however, covered almost all items consumed by all extant primates. For example, suggestions have included a specialised diet of meat, grasses, leafs, fruit, small hard objects, and termites (Daegling et al., 2013; DuBrul, 1977; Grine et al., 2012; Merceron et al., 2004; Peters, 1987; Scott et al., 2005; Walker, 1981). Two of the most insightful ways of interpreting diet in fossil hominins are dental microwear and isotopic analysis. The former of these methods has led to much discussion regarding dietary interpretation, and the latter shows a mix of C3 and C4 foods were likely consumed (Grine, 1981, 1986; Grine et al., 2012; Merceron et al., 2004; Scott et al., 2005; Sponheimer et al., 2005; Sponheimer et al., 2013; Teaford & Glander, 1996; Teaford & Robinson, 1989; Ungar & Grine, 1991). Therefore, it is still very much open to debate what *P. robustus* ate, and therefore also why such large masticatory equipment evolved. The aim of this study is to try a different approach in reconstructing diet by looking at dental pathology, developmental defects and macrowear, which will hopefully provide further insight into the diet of these individuals. This data will also be able to be combined with the microwear and isotopic analysis and hopefully suggestions on what sort of food could cause these different patterns and signatures can be suggested. Particularly, it will be interesting to see how many ante-mortem dental chips these individuals have compared with extant primates with known diet, as well as with other fossil hominins, to see if hard object feeding was common. Severe pitting enamel hypoplasia has been recorded on certain specimens of *P. robustus* (Moggi-Cecchi, 2000), but as yet the scale of these defects, as well as a differential diagnosis, has not been explored. Therefore, developmental defects and chipping will be particularly interesting to study in this species.

1.2.4. Australopithecus africanus

In this project the largest hominin sample, in terms of number of individual teeth, is *A. africanus*. Some specimens are subjected to much discussion in terms of phylogeny (Calcagno et al., 1999; Fornai et al., 2015; Grine, 2013; Lockwood & Tobias, 1999; Moggi-Cecchi, 2003; Wood & Richmond, 2000). The species as a whole has been suggested to be ancestral to *Homo* and *Paranthropus*, but such debate is very much ongoing. Similar to *P. robustus*, a wide variety of diets have been suggested for this species (e.g., Strait et al., 2009). The debate regarding occlusal wear, chipping, biomechanics of the masticatory system, and developmental stress have all remained discussion points (Grine et al., 2010; Scott et al., 2005; Sponheimer et al., 2013; Van Der Merwe et al., 2003). Given the large scale, cross-species, nature of the comparisons in this study it is hoped insight into these debates can be made. For example, biomechanical research has proposed *A. africanus* individuals may have evolved to process high-stress loads in the premolar region of the dentition; therefore it will be interesting to see if these teeth are particularly affected by ante-mortem fractures compared to other teeth, as well as other species. No carious lesions have yet been found on any tooth belonging to *A. africanus*, which is unusual given the large sample size and the growing number of lesions found on other hominin specimens. This will be explored by comparing caries frequencies with other fossil hominins and extant primates.

1.2.5. Early Homo

This sample consists of a few individuals, but the exact number of teeth is highly debated in the literature, with some specimens often attributed to another known species such as *P. robustus* or *A. africanus* and others suggesting them to belong to a separate new species (Grine et al., 2013). Which *Homo* species, or number of species, is represented is also highly debated (Braga & Thackeray, 2003; Grine et al., 1993; Moggi-Cecchi et al., 1998). These specimens are therefore simply called early *Homo* in this thesis. This material is morphologically distinct from the *H. naledi* material and therefore not grouped together. Comparisons with the other South African material will be intriguing. For example, it will be interesting to see if this early *Homo* material has more in common with much later *H. naledi* specimens or the more contemporary material assigned to *Paranthropus* and *Australopithecus*.

The recently published *A. sediba* material is similar in age to other species discussed above, particularly early *Homo* and *P. robustus* (Chapter 2). Clearly, there were multiple hominin species thriving in South Africa during roughly contemporary times. This raises the possibility that these different species filled different ecological niches, an idea where dental pathology and wear data can perhaps provide some insight. Additionally, the surprisingly young age of the *H. naledi* material offers a fascinating insight into the continued use of the cradle of humankind. Diet is often noted as changing dramatically in both the genus *Homo* and *Paranthropus*; particularly the use of fire and high intake of meat is often cited for the former. Therefore, it will be interesting to see if wear and pathology differences are visible between these different genera or if variation appears relatively unrelated to phylogeny.

1.2.6. Comparative samples

The comparative sample consists of baboons, chimpanzees, gorillas, drills, and agricultural humans. The extant primate comparative material offers insight into what effect certain foods and behaviours can have on the teeth since such dietary and behavioural information is widely available for these species. Therefore, if similar patterns are observed in the hominin fossils, it can be speculated that the same aetiology is responsible. It will also be interesting to look at these species independently given the small amount of data on these variables in the literature. The same data and analysis will be performed on all species, and any rare defect, wear and pathology also recorded and described.

1.3. Project aims

This is an exploratory study given that few macroscopic dental pathology studies have been conducted on the South African hominins. Indeed, as yet no study has been conducted that compares multiple variables. The overall aim of this study is first to elucidate dietary and behavioural components for these hominins but also to create a data set that can be easily compared and contrasted with other hominin and primate groups. Additionally, a further aim of this project is to explore the findings of the limited number of studies that have already looked at specific dental pathologies, wear and development defects in these specimens (see Chapter 3), and to compare these previous findings with the results of this study in each individual chapter.

1.3.1. Research questions to be addressed

Because this study is largely exploratory, several broad research questions will be addressed rather than testing specific hypotheses. These questions include:

- Can prior studies of diet in these various hominins based on microwear, isotopic analysis etc. be supported or refuted by the study of basic dental pathological indicators?
- Can pathology, wear, and enamel defect data suggest differences in diet between the South African hominins?
- Is there a link between the different variables and species? That is, are there certain species which consistently have similar patterns of wear and pathologies?
- Do dental pathology and wear data fit with known ecological information on the extant primate samples, and if so what diet is therefore most applicable to the different hominin species?
- Are differences observed between the different hominin species substantial enough to infer different ecological niches?

These questions will be addressed throughout this thesis.

Chapter 2: Sites and species

The sites in which the South African hominin material was found are not scattered evenly across this vast country. In fact, all the material studied comes from an area of only 47,000 hectares called the “cradle of humankind” (Figure 2.1). Located 50 kilometres Northwest of Johannesburg in the Gauteng province it was made a World Heritage Site by UNESCO in 1999. There are many reasons for the abundance of fossils, both hominin and other animals, in the cradle of humankind, but the main factors are the geology of the area and the climate at the time of deposition. However, it is also possible that these same factors not only allowed better preservation of remains but also influenced, to a certain extent, the diversity and number of hominins at the site.

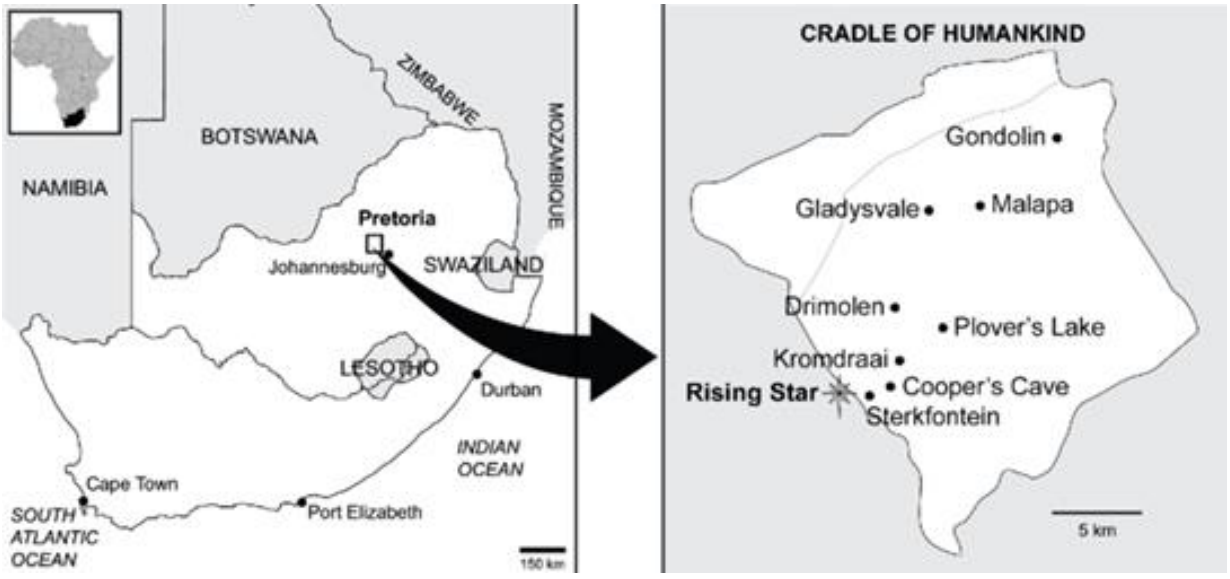

Figure 2.1. Map showing the location of the cradle of humankind, with hominin bearing caves highlighted. Edited from Hawks et al. (2017).

2.1. The cradle through time

In the last few hundred years, extensive quarrying and mining has taken place in the cradle of humankind. Over half the world's gold in current circulation was mined from the surrounding area. Platinum mines have also been widespread. Gold was first discovered in

the area in 1884, with extensive mining following soon after. Lime was needed for both gold processing and the manufacture of cement, so the area in and around the cradle of humankind was extensively mined. This led to the discovery of hominin fossil sites, but unfortunately almost certainly destroyed many specimens. As Robert Broom wrote:
"*It is sad to think that for nearly 40 years no scientist ever paid the slightest attention to the caves; and probably some dozens of skulls of ape-men and all the bones of their skeletons were burnt in lime kilns*" (Broom & Schepers, 1946).

2.2. Sites

2.2.1. Taung

In 1924 the Taung child was discovered by Raymond Dart hundreds of miles away from the cradle of humankind in Buxton in the Northern Cape. The Taung child is the type specimen of the most numerous of South African hominin species, *A. africanus.* Due to the fragility and importance of this specimen, it was not available for study.

2.2.2. Sterkfontein

The first fossil discoveries were made by Marist Brothers College students, from Johannesburg, in 1895 (Clarke, 1988). However, a mining company that owned the land intended to extract large quantities of lime from the cave system, which they did, but fortunately a geologist called David Draper persuaded the company to leave the main cave untouched, using the stalactite and stalagmite formations, as well as an underground lake, as reasoning.

In April 1947 Robert Broom and John Robinson discovered what is now one of the most famous fossils in the world, Mrs Ples (STS 5). Dynamite blasting was used, to the dismay of modern researchers, as well as many at the time, to dislodge the breccia and allow fossils to be found. This separated the skull into two fragments, but luckily, no serious damage was done. This was the first adult *Australopithecus* discovered, and it helped cement the idea that along with the Taung child these fossils did indeed belong to the hominin lineage (Broom, 1949; Robinson, 1954, 1956).

The site has been continually excavated since 1966 making it one of the longest-running archaeological digs in the world (Clarke et al., 2003; Hughes & Tobias, 1977; Moggi-

Cecchi et al., 2006). Hominin specimens from this cave are predominantly *A. africanus*. Between 1966 and 1989 Alun Hughes and Phillip Tobias collected and catalogued 505 hominin specimens (Moggi-Cecchi et al., 2006). It is worth noting the number of other animal fossils that were found at this time, with Charles Brain recovering 350,000 fossil specimens (Brain, 1983).

The first discovery of a fossil that could confidently be assigned to the genus *Homo* was made in 1976, under the supervision of Alun Hughes (Hughes & Tobias, 1977). What species is represented is unknown, although it was originally catalogued as *H. habilis* with some suggesting *H. ergaster* (see below).

There is also a well-preserved specimen known as Little Foot, STW 573 (Clarke, 1998). Ron Clarke discovered some hominin foot bones while going through mislabelled fossil bags, after finding more leg bones that fitted this specimen he was convinced the rest of the skeleton was still in Sterkfontein caves, in particular, the area known as Silberberg Grotto. Other skeletal elements have since been found in this area, but extraction of the material from the breccia proved difficult. These specimens have now been removed from the cave but were not available for study in this project, but virtually all other specimens from Sterkfontein were.

Member 4 from Sterkfontein has yielded large quantities of hominin fossil material (Pickering et al., 2004). This formation was originally called the Lower Breccia (Robinson, 1963), and fossil hominins have been extracted since the 1930s. Some fossil have been found in situ deposits within the cave, whereas others come from breccia blocks in lime miners dumps (Tobias & Hughes, 1969). With some blocks, their origin is less certain. However, some are more certainly originally from the Member 4 formation, including dumps 9, 10, 13, 14, 15, 17, and 18 (Tobias et al., 1967).

2.2.3. Kromdraai

In June 1938 hominin teeth were found by a local school boy, Gert Terreblanche, and after some initial misleading and confusion from a Sterkfontein quarryman, the site and the fossils (including the teeth recovered by the school boy) were found. The fossils came from the cave known as Kromdraai and now have the specimen number TM 1517. These remains

are very robust, with a large jaw and molars and therefore thought worthy of a distinct and new species name, *Paranthropus robustus*, of which TM 1517 is the type specimen.

2.2.4. Coopers

Controversy surrounds an isolated tooth found at Coopers in the 1930s, with some suggesting it was transported there from Sterkfontein (Hilton-Barber & Berger, 2004). This tooth has since gone missing from the University of the Witwatersrand. However, a different tooth was found in what is now Ditsong Museum which was enough to convince Lee Berger to carry out excavations. These excavations were successful with further hominin material uncovered, and there was also the unusual discovery by Christine Steininger of a partial hominin skull within the Ditsong Museum's fossilised fauna collections (Berger et al., 2003; Steininger et al., 2008). This material has been classified as *P. robustus*. A few more teeth have also since been found (de Ruiter et al., 2009).

2.2.5. Drimolen

This is the only site not included in this project, as these specimens were not available for study at the time of data collection. This relatively newly discovered hominin fossil bearing site has produced a significant amount of dental material thought to belong to *P. robustus* (Keyser et al., 2000; Moggi-Cecchi et al., 2010). However, where possible, this material will be incorporated into this thesis by using published information on these specimens from the literature.

2.2.6. Swartkrans

There are five Members defined for Swartkrans, with the first three containing hominin material (Clark, 1993). Member 1 has produced abundant remains of *P. robustus* but no other distinct hominin species. Member 2 also contains a large number of specimens assigned to *P. robustus* but also some attributed to the genus *Homo*. Similarly, Member 3 contains evidence of both. However, only a small number of postcranial bones represent *Homo* in this member (Grine, 2005).

The two main concentrations of hominin fossils from all sites come from Swartkrans Hanging Remnant (Member 1) and Sterkfontein Member 4. What is remarkable about the finds is not just the sheer number but also the proportion of all faunal remains that have been

excavated that are hominin. Brain (1983, 1993) found that a minimum of 90 individuals are represented in the Swartkrans Hanging Remnant, which make up 20% of the total assemblage MNI. Sterkfontein Member 4 was found to have a minimum of 45 individuals represented, comprising 13% of the assemblage MNI. Furthermore, more recent work has shown that the number of individuals in Sterkfontein Member 4 is likely much higher than thought, with at least 87 individuals represented (Pickering et al., 2004).

2.2.7. Makapansgat

Apart from potential recent *H. sapiens* remains, the site consists of only material belonging to *A. africanus* (Reed et al., 1993). It will be interesting to see if there are any difference in pathologies and wear with the Sterkfontein *A. africanus*.

2.2.8. Malapa

The geology of the area surrounding the Malapa Cave means that water seeps deep into the bedrock and prevents the thin layer of top soil from supporting larger plants. However, where there are underground streams, sinkholes or cave entrances, there is potential for larger plants and trees to take root. Although many of these trees have since been felled for use in mining operations, the stumps can still be used in modern day prospecting potential fossil sites. This method has been enhanced in recent years with new satellite technology allowing vast areas of landscapes to be viewed remotely, one of the methods used by Lee Berger and his team in his discovery of *A. sediba*, using Google Earth (Berger et al., 2010). Skeletal elements from a minimum of six individuals have since been found, all thought to belong to *A. sediba,* although it is two individuals, MH1 and MH2, that stand out as being remarkably complete (Berger et al., 2010; Irish et al., 2013; Kibii et al., 2011; Zipfel et al., 2011).

2.2.9. Rising Star

The most recently discovered of the samples in this thesis is *H. naledi*. The material comes from the Dinaledi chamber within the Rising Star cave system, discovered in 2013 (Berger et al., 2015). A minimum of 15 individuals are represented, with the very unusual circumstance of virtually no other animals remains found in the chamber (Dirks, 2015). Due to how the remains are preserved and the difficulty in getting to this chamber, it is suggested

deliberate burial of these individuals may have taken place (Berger et al., 2015). Already many publications have studied *H. naledi* with preservation, phylogeny and diet the focus (e.g., Berger et al., 2015; Cofran et al., 2016; Feuerriegel et al., 2016; Harcourt-Smith et al., 2015; Kivell et al., 2015; M.M. Skinner et al., 2016; Williams et al., 2017). All the material from this cave belongs to a single species, *H. naledi*, with uniform dental morphology between individuals. The discovery of further *H. naledi* material in a second chamber within this cave system has recently been published (Berger et al., 2017; Dirks et al., 2017; Hawks et al., 2017). This material promises to provide further insight into this species, with a very complete individual as well as the presence of other animal fauna.

2.3. Site codes

Site codes are used in tables and the text. TM is material from Sterkfontein and Kromdraai and comes from 'Transvaal Museum' which is the original name of what is now the Ditsong National Museum of Natural History, in which the material is still curated. STS is from the same site and stands for 'Sterkfontein Type Site'. STW is also from Sterkfontein. MLD stands for 'Makapansgat Limeworks Dumps' which is a literal description of where the material was found. SE stands for 'Sterkfontein Extension'. UW 101 refers to the *H. naledi* material, which is curated at the University of the Witwatersrand. SK and SKX are both site codes for Swartkrans. KB is the site code for Kromdraai. MH is the site code for Malapa, which is represented by *A. sediba* material.

2.4. Species classification

There is debate regarding almost all hominin fossil remains as to which species the remains should be assigned to. The South African material has been lumped and split into different groups over the years, and, particular specimens, as well as all specimens from particular sites, are still subjected to ongoing debate.

Most comparisons in this thesis are made among five main groups: *A. africanus, P. robustus, H. naledi, A. sediba* and early *Homo*. Evolution, however, does not tend to produce such distinct populations over such time frames, and therefore it is not surprising that debate surrounds many specimens. Instead, variation is ubiquitous, and what one person thinks of as a distinct species others will argue it is simply variation of an already-defined species. However, in studies looking at diet and behaviour, it is nonetheless useful to group specimens

so that large scale comparisons can be made. Furthermore, the specimens studied show significant variation among samples, with distinct morphological differences among most of the species studied, meaning that such splitting is justified (Grine, 2013; Irish et al., 2013). However, results will also be presented by site where appropriate, to see if there are differences within these groups. Table 2.1 shows the material assigned to each species with the sites and time span represented for each displayed.

Table 2.1. Sites, number of teeth and dates for each species studied. YBP: years before present.

Species	Number of teeth	Dates	Sites
H. naledi	181	335,000 to 236,000 YBP	Rising Star
A. sediba	20	1.980 to 1.977 million YBP	Malapa
P. robustus	511	2.0 to 1.2 million YBP	Kromdraai, Swartkrans
A. africanus	504	3.3 to 2.1 million YBP	Sterkfontein, Makapansgat
Early *Homo*	81	2.0 to 1.5 million YBP	Sterkfontein, Swartkrans
Olive baboons	867	19th/20th century	*
Chimpanzees	3256	19th/20th century	*
Drills	246	19th/20th century	*
Gorillas	2597	19th/20th century	*
Hamadryas baboons	90	19th/20th century	*
H. sapiens	921	12th to 17th century	Gloucester, UK

*wild-shot, see text for details

Below is a short summary of some of the main issues surrounding the classification of different species and individuals, and how such issues are taken into account in this project.

2.4.1. Australopithecus africanus

When hominin material was first discovered at Taung, Sterkfontein, and Makapansgat, it was assigned to three species across either two or three genera (Broom, 1936, 1938, 1950; Dart, 1925, 1948). After these early years, however, the consensus was that all such material represented a single species, *A. africanus* (Rak, 1983; Robinson, 1954; Tobias, 1967). Recently the high variation in tooth size has led researchers to suggest more than one species is represented in Member 4 deposit in Sterkfontein (Grine et al., 2013). Even amongst those researchers that would split this material, there is much debate as to which specimens are allocated to which species (see Grine et al., 2013). A particularly contentious debate centred on material from the site of Makapansgat. Although Tobias (1967) considered this material the same species, he did note the robustness of the remains compared to those from

Sterkfontein. Aguirre (1970) went on to suggest that such robusticity in some specimens was due to two species being represented in the assemblage, *A. africanus* and *P. robustus*. This work also suggested the same may be the case for the Sterkfontein collection. This possibility would not receive significant attention for a couple of decades (e.g., Clarke, 1988; Grine, 2013).

The majority of the *A. africanus* remains come from Sterkfontein. A large proportion of these *A. africanus* specimens (including well-preserved specimens such as STS 5, STS 17, STS 19, and STS 71) were discovered between 1947 to 1949 by Broom and Robinson, describing them all as one species (Broom, 1949). A well-represented skeleton from Sterkfontein was discovered in the 1980s and assigned the specimen number STW 151. This individual has been described as potentially more derived in the direction of *Homo* relative to other *A. africanus* from Member 4 (Moggi-Cecchi et al., 1998). However, following the majority of other studies, this specimen is recorded as *A. africanus*. Two other parts of the Sterkfontein cave system that are fossil-bearing is Silberberg Grotto and the Jacovec Cavern. The famous find from Silberberg Grotto is STW 573, also known as 'Little Foot.' At present little research has been published on these specimens. The remains are very well preserved with many elements represented (Clarke, 1998). It seems likely that this individual is dated much earlier, perhaps to 3 Ma (Bruxelles et al., 2014; Clarke, 1998), than the material from Sterkfontein Member 4, although there is still debate to the actual date due to potential movement within the cave.

2.4.2. Paranthropus robustus

Originally, Robert Broom suggested the material now assigned as *P. robustus* was made up of two species, but this theory was later discounted by John Robinson; since then there has been much debate in the literature, although most studies tend to keep all remains within one species. However, some researchers maintain there are multiple species represented. Most notably, morphological differences between specimens from Kromdraai and Swartkrans, particularly in deciduous dentition, have been highlighted (Grine, 2007). Wallace (1975) suggested that a single lineage was observed in the specimens now assigned to *A. africanus* and *P. robustus* with the latter evolving from the former. He saw the Makapansgat specimens as intermediate in form between these two types and thought they

represent a species that partly linked the two groups. In this study, all Swartkrans teeth, except those assigned to *Homo* (see below), as well as Kromdraai specimens, are assigned to *P. robustus*.

A specific specimen that has been debated in the literature is SKX 5013. It is from Member 2 of Swartkrans and is represented by part of the left mandible, with a complete first molar present. It has been assigned to both *P. robustus* and *Homo* (Grine, 2005; Schwartz & Tattersall, 2003). It is clear that this individual is noticeably smaller than other *P. robustus* specimens but as Grine et al. (2015) point out size is not often a good way to distinguish species. They suggest instead that *P. robustus* may have had a level of sexual dimorphism similar to gorillas and therefore SKX 5013 may simply represent a small female of this species (Grine et al., 2012; Lockwood et al., 2004). Following this logic, SKX 5013 is here classified as *P. robustus*.

2.4.3. Early Homo

In the article describing *H. naledi*, Berger et al. (2015) view the comparative specimens assigned to *Homo* from Swartkrans as belonging to *H. erectus*, although this was mainly for convenience of comparisons. Specimens included in this category were: SK 15, SK 18a, SK 27, SK 43, SK 45, SK 68, SK 847, SK 878, SK 2635, SKW 3114, SKX 257/258, SKX 267/2671, SKX 268, SKX 269, SKX 334, SKX 339, SKX 610, SKX 1756, SKX 2354, SKX 2355, SKX 2356, and SKX 21204. These are not all represented by teeth, but here the same format is followed in including SK 15, SK 27, SK 43, SK 45, and SK 2635 as 'early *Homo*'. Unfortunately apart from a few well represented and preserved examples such as SK 15, SK 45, and SK 27, the early *Homo* dental sample from Swartkrans is predominantly isolated, broken, teeth.

From Sterkfontein the samples designated here as early *Homo* are specimens from the Sterkfontein Extension site (SE 255, SE 1508 and SE 1937). Also debated in the literature are STW 19 and STW 42, both of which have been classified as *Australopithecus* and *Homo;* here they are included with *Homo*, though their small number does not affect the results for any pathology except chipping (see Chapter 5). Additional *Homo* specimens from Sterkfontein are STW 53, STW 75, STW 80, and STW 151 (Clarke, 1985; Hughes & Tobias, 1977; Kuman & Clarke, 2000; Moggi-Cecchi et al., 1998, 2006; Robinson, 1963; Tobias, 1979).

2.4.4. Homo naledi

Although only recently published, *H. naledi* has been subjected to debate concerning phylogeny. At present, the majority of objections to its placement and new species status has largely been in the media rather than in peer-reviewed journals. The unique morphology across these individuals, in a variety of different skeletal elements, is supportive of the new species classification in the genus *Homo* (Berger et al., 2015). Furthermore, the dental morphology is remarkably uniform across individuals; as such, there is little doubt that these individuals represent the same species (Berger et al., 2015; Cofran et al., 2016; Hawks et al., 2017; M.M. Skinner et al., 2016).

2.4.5. Australopithicus sediba

The unique morphology of this material has led to debate regarding how the Malapa material should be characterised, including the genus and species (Berger et al., 2010; Carlson et al., 2016; Irish et al., 2013; Kibii et al., 2011; Pickering et al., 2011; Zipfel et al., 2011). Much of this debate revolves around interpretation of juvenile remains, but also the relationship with the genus *Homo* and other *Australopiths* is common. In this thesis, all material from Malapa is recorded as *A. sediba*.

2.4.6. Family Tree

As mentioned, the phylogenetic relationship among species in this thesis has been debated in the literature (e.g., Berger et al., 2017; Moggi-Cecchi et al., 2006). Although not an essential part of this project, with analysis mainly among the five species mentioned, such relationships will be discussed in Chapter 12 concerning ecological niches that the species may have filled. These niche differences are of particular interest given the overlapping occurrence of potentially three of the species, *P. robustus*, A. sediba and early *Homo* (Figure 2.2). The three genera in this study, *Homo*, *Paranthropus*, and *Australopithecus* will also be interesting to compare variable frequencies, particularly because they are made up of a similar number of teeth (Chapter 4). The *H. naledi* material is much more recent than any other samples in this study, between 236 ka and 335 ka (Dirks et al., 2017), raising an interesting question as to whether their diet was substantially different than the earlier hominins (Figure 2.2).

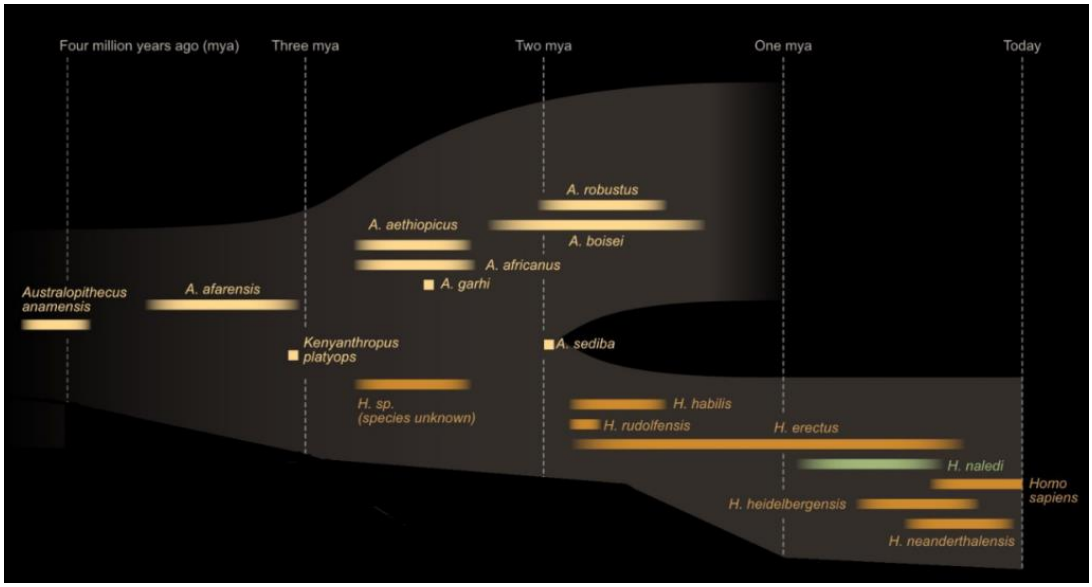

Figure 2.2. Phylogenetic tree showing the five main species used, along with other hominin groups. Adapted from the National Geographic.

2.5. Geology and deposition

The cradle of humankind is dominated by 2.6 billion-year-old dolomitic formations, with cave systems formed in numerous locations (Dirks & Berger, 2013; Gommery et al., 2008; Witthüser & Holland, 2008). Once a cave has formed, due to the process of erosion and dissolution of dolomite, animal remains, along with other debris in the area, are washed or otherwise transported in, causing a build-up of material. If the remains are located in a zone that has lime-bearing water dripping or seeping through it, then there is a chance it may become calcified (Dirks et al., 2015). The fossils that are not then eroded have the potential to be stumbled upon hundreds of thousands of years later by an observant archaeologist.

More broadly, there are many ways for an animal or plant to become fossilised, including an impression of footsteps or original specimen, void filling with minerals seeping through the surrounding rock, and total replacement of the original specimen with mineral (Grimes et al., 2001; Pfretzschner, 2004). The caves in the cradle of humankind have a mix of different preservation types, but concerning hominin material, the most important are fossilisation of material through calcium carbonate precipitated from the bedrock (Hilton-Barber & Berger, 2004). Once such replacement takes place the resulting block of cemented material can be incorporated into breccia. This is true for all the hominin specimens in this study, although it should be noted that the *H. naledi* material offers a unique and fascinating preservation history that is still being studied (Dirks et al., 2015; Dirks et al., 2017). Luckily, in general, this

process has little effect on the appearance of enamel and other dental tissues and therefore allows most of the pathologies and wear recorded in this thesis to be observed in the majority of specimens.

2.6. Archaeological record and preservation

The environment in which fossil hominins lived is a vital component to incorporate in any study looking at diet and behaviour. Another important aspect to consider is that specimens of *P. robustus* and *A. africanus* cover a large time span. The environment and their behaviour potentially changed significantly during this time. However, it is interesting to note that isotopic analysis results for different specimens show there is no trend over time (Sponheimer et al., 2005), although there is high variability in isotopic results in both species, particularly *A. africanus*, at any given time. Environmental components such as fluoride and mercury levels as well as deficiencies such as vitamin D may also be important to consider as they can have an impact on dental formation (Alvarez et al., 2009; Ioannou, 2015; Ogden et al., 2007).

When discussing the ecology and behaviour of early hominins, it is important to remember that they were unlikely to have been at the top of the food chain (Chapter 12). Large cats including lions, leopards and the now extinct sabre-toothed and false sabre-tooth cats roamed this area, as well as numerous species of hyena and dog species. Birds of prey that would have been capable of taken small hominins were also present around the cradle of humankind, including large eagles and owls. This is highlighted by the fact hominin bones have shown evidence of damage caused by such predators (e.g., Berger & Clarke, 1995; Pickering et al., 2000).

2.6.1. Taphonomy, excavation and curation

The process of fossilisation, excavation, and curation can all have dramatic effects on the preservation of teeth, and therefore methods must be implemented to allow for comparisons to be made between sites to try and counter any potential bias. The archaeological and fossil record very rarely approaches completeness, in terms of a realistic cross section of skeletal elements as well as how pristine are the elements that survive. In fossil hominins, different assemblages will be more predisposed to contain certain individuals

within the populations, selected by size, age, sex or health. A good example of this is research carried out on the Swartkrans material by Charles Kimberlin Brain and later by Travis Pickering and colleagues. This work suggests that the accumulation of animal remains in Members 1 and 2 was likely due to the actions of large carnivores such as leopards and hyena. Pickering provides evidence suggesting such action may also have led to the accumulation of such remains in Member 3; Brain favoured hominin activity as the cause of accumulation of fossils in this member (Brain, 1969, 1970, 1983, 1993; Pickering, 2001; Pickering et al., 2004, 2005, 2007, 2008).

Therefore, there may be clear differences between fossil sites depending on how material was accumulated. Clearly, a site that consists mainly of animals that were killed and carried to that location by leopards or other carnivores will have a different set of characteristics than that of a site resulting from a natural disaster. Similarly, a modern collection that is made up of wild-shot individuals, such as the primates from the Powell-Cotton museum, should also have a different pattern and set of biases.

Likewise, due to the varying circumstances in which an animal can be fossilised, there are often significant differences in preservation of certain skeletal elements between sites. This selection of elements can create biases in data sets if such circumstances are not taken into account. For example, if a site is made up of skeletal material that has been washed into a cavern that has later fossilised, then skeletal elements may get mixed up and some potentially washed away and therefore not fossilised. This scenario can cause issues for dental studies because anterior teeth tend to become dislodged from the maxilla and mandible more often than molars, due largely to differences in root morphology.

Even before fossilisation takes place, there are numerous factors that lead to varying degrees of preservation, including: time exposed on the surface, climate, biodegradation and soil type. Such information is principally important for the small amounts of maxilla and mandible available for study rather than teeth. Although there have been some examples of microbiological degradation of dental material in fossil mammals (e.g., Fostowicz-Frelik & Frelik, 2010; Kalthoff et al., 2011), it has been noted that most teeth show little or no evidence of such osteolysis (Grine et al., 2015). This lack of deformation is likely due to enamel being a particularly uninviting host to such bacteria but also because fragmentary remains tend to be less affected by such processes.

Although, as will be highlighted in the following chapters, taphonomic processes are unlikely to be misidentified as ante-mortem; certain features are, nonetheless, important to consider. Many teeth in this study are heavily discoloured, making the recording of certain conditions difficult and creating potential error. For example, many hominin specimens have a black discoloration. This colouration occurs due to the presence of manganese in dolomitic bedrock in which the fossils were entrapped, with the possibility that biodegradation in the sediment may have been needed to facilitate fossilization (Grine et al., 2015). Figure 2.3 illustrates this staining on a specimen from Swartkrans (SKX 5013), with black colouration on the mandible as well as dentine islands on the occlusal surface of the first molar. It is worth noting that this staining does not extend far beyond the surface in specimens (Kuczumow et al., 2010). It has also been suggested that this layer may have protected the material from further intrusion from organic matter (Kuczumow et al., 2010). Such colouration rarely makes a tooth unobservable for all variables; however some, such as tertiary dentine on the first molar in Figure 2.3 may be affected.

Other taphonomic processes can leave a tooth in perfectly recordable condition for all variables and simply change the colour of material through contact with particular minerals during fossilisation; some strikingly colourful examples can result (Figure 2.4).

Perhaps surprisingly, the main problem encountered during data collection was not caused by morphological differences among species, but rather preservation of the material. The most striking difference is between fossil specimens and modern day extant ape samples. However, variation in the preservation even within the South African hominins is significant. For example, *H. naledi* material is made up of very pristine teeth with little post-mortem damage and are associated with many other teeth (Chapter 4). Commonly, this is not the case for *P. robustus* and *A. africanus*. How these different processes affect the different species are highlighted in the individual chapters, along with methodological techniques used to counteract potential bias.

Figure 2.3. Black discolouration on the mandible and left lower first molar of SKX 5013, caused by taphonomic processes.

Figure 2.4. Blue colouration to the dentine in SK 873.

Some teeth have also been broken post-mortem, before, during and after fossilisation. Such teeth can be removed from analysis for any variable that cannot be recorded (see Chapter 4). Rarely is a tooth damaged in such a way that it is mistaken for an ante-mortem pathology or defect (see Chapter 5). Sometimes the overall shape of a tooth or jaw can be changed. For example, the lower left first molar of SK 63 has moved inferiorly compared to the deciduous second molar, although in this case, it is clearly post-mortem in origin, it is important to keep such factors in mind during data collection.

Another post-mortem mechanism that is worth mentioning is how fossils have been excavated and curated. It was common up until the 1950's for dynamite to be used to retrieve fossils, with, undoubtedly, much damage caused. This issue should not have much effect in this study since such post-mortem damage does not seem to have extensively affected isolated teeth. However, two other components may have had an effect. The handling of specimens may, over time, erode pathologies or developmental defects so that they are slightly less visible, or potentially for such pathologies as calculus, be completely removed. It could be argued that such handling likely has little effect, barring calculus, and should theoretically be similar across sites and therefore not of great concern. However, that is not the case for methods used to remove debris. In many of the Ditsong Museum specimens acid was used to remove different matrix, whereas much of the material curated at the University of the Witwatersrand was removed mechanically using instruments. Acid removal almost certainly removed more calculus deposits than the mechanical method, and therefore it is difficult to compare these groups. Other pathologies, wear and developmental defects are likely relatively un-affected by such factors. For these reasons, calculus is not included in the analysis or results of this thesis. Results are too unreliable, in that deposits are likely lost through a variety of taphonomic processes as well as handling and processing.

Chapter 3: Dental pathology, developmental defects and wear

3.1. Introduction

Research involving dental pathologies can provide valuable insight into diet, social systems, cultural practices, systemic stress, idiosyncratic behaviour, health, and disease of individuals and groups, as well as information on when certain traits and behaviours developed in the hominin lineage. Research involving the dentition is common in the paleoanthropological literature, partly due to the preservation bias in favour of teeth, but also because of the wealth of information that can be gained from these remains. Indeed, prior research has looked in depth at dental pathologies and anomalies in hominins; however much of this work has been primarily descriptive in nature and often limited to one species, or focuses on a single variable. A brief overview of different pathologies, developmental defects, and wear is given below, with a more in-depth discussion in each specific chapter. In this section, the focus is on research that has been conducted on hominin material, particularly the South African specimens, as well as giving a concise summary into what inferences each defect can give into diet and behaviour. Additionally, other evidence for diet and behaviour in these hominins is explored to allow a base literature on which this works builds.

3.2. Caries

Caries is present in a wide range of hominin populations, but varies in prevalence, severity and location depending on the group, population and species studied (Figure 3.1; Byun et al., 2004; Gussy et al., 2006; Humphrey et al., 2014; Lanfranco & Eggers, 2012; Larsen et al., 1991; Lukacs, 1996). Carious lesions can provide information on the diet and oral health of individuals, with the position and severity key to attributing behaviours and diets to populations as a whole (Hillson, 2005). Additionally, the teeth most affected can give insights into food processing habits. For example, the high attrition of anterior teeth in early 20th century Greenland Inuit, due to a diet consisting predominantly of meat, means the little worn third molars have a high level of caries (Pederson, 1947), although still relatively rare in contrast to most agricultural groups. In contrast, the teeth of contemporary Australian

aboriginal groups, with a diet high in grass seeds, have a much higher rate of caries, particularly in the first molars (Hillson, 2005). Other factors that can be gleaned from the presence, location and severity of caries include dental hygiene techniques, cooking technology, frequency of food consumption and sex differences (Crittenden et al., 2017; Hillson, 2001; Tayles et al., 2000). Caries has been extensively researched in recent human populations, allowing many samples in which comparisons can be made (e.g. Humphrey et al., 2014; Larsen et al., 1991; Novak, 2015; Rohnbogner & Lewis, 2016; Walker, 1986). Carious lesions have already been found in South African hominins (Grine et al., 1990; Robinson, 1952) as well as numerous fossil *Homo* specimens (e.g., Lebel & Trinkaus, 2001; Tillier et al., 1995; Trinkaus et al., 2000; Walker et al., 2011). That said, the South African fossil hominins have not yet been studied for caries using micro-CT scans and specimens belonging to *H. naledi* have not been studied at all for the presence of caries.

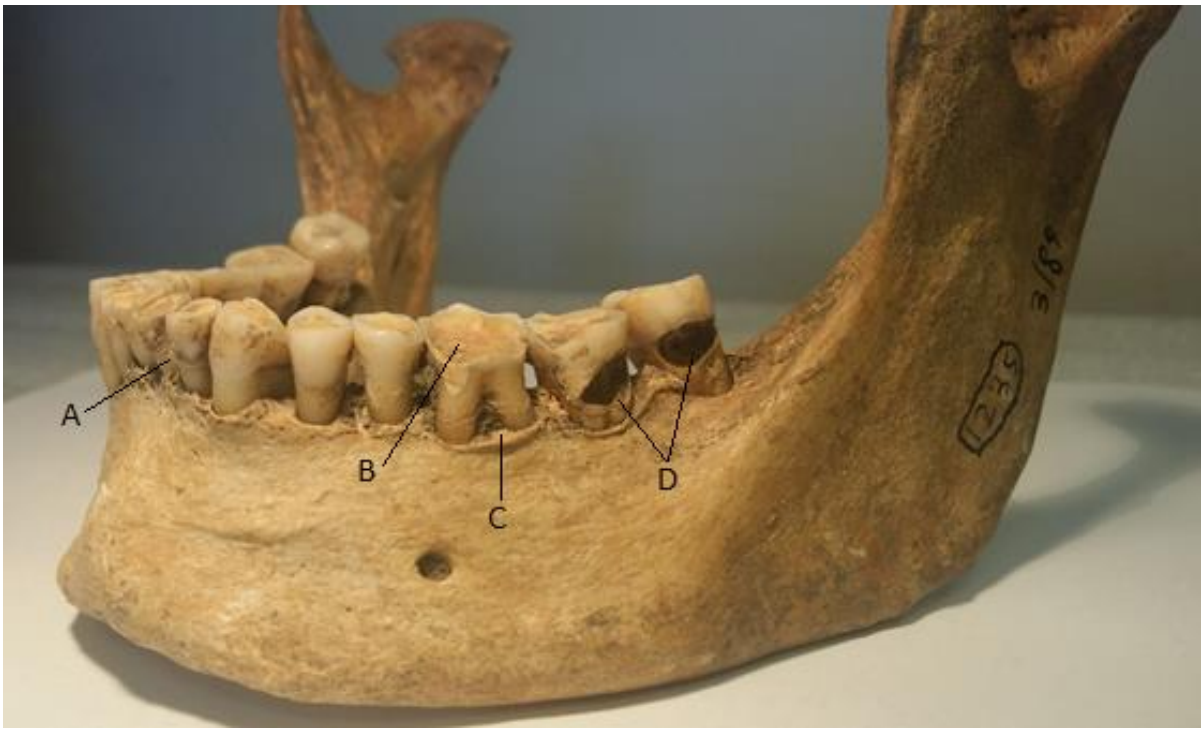

Figure 3.1. Dental pathology and wear on a mandible of skeleton 1235 from the medieval Gloucester collection. A: calculus; B) heavy/angled occlusal wear; C) periodontal disease; D) caries.

3.3. Dental wear

Dental wear comes in the form of attrition, abrasion and erosion, although in reality, dentitions can show a combination of these (Burnett et al., 2013; Kaidonis, 2008). Attrition is caused by the occlusal surfaces of teeth coming into contact with each other, whereas abrasion occurs when other materials introduced to the mouth wear teeth during mastication or via localised wear from food processing (Burnett et al., 2013). Attrition and abrasion can provide in-depth detail on an individual's diet, cultural practices and idiosyncratic behaviours. Particular wear patterns are associated with different practices, and by comparing various groups, it is possible to then infer these ecological and social behaviours in archaeological remains. For example, particular wear patterns are associated with certain types of agriculture, high meat intake, and a more varied hunter-gatherer lifestyle (Deter, 2009; Fiorenza, 2011; Smith, 1984). Wear research has been carried out on a variety of hominin species and has been used to deduce diet, behaviour and even potential cultural practices in *Australopithecus* (Clement & Hillson, 2013; Estebaranz, 2009; Grine, 1986; Tobias, 1967; Ungar & Grine, 1991) and *Homo* (Fiorenza, 2011; Ungar et al., 2001). Another aspect of wear research is occlusal and interstitial wear, which can be used to estimate the age at death, diet, environmental factors and potential cultural practices (Burnett et al., 2013; Fiorenza et al., 2011). Wear from food processing, tool use or using an instrument to relieve a toothache have all also been observed in different archaeological samples (e.g., Estalrrich et al., 2016; Ungar, 2001). Conflicting results have been presented for occlusal wear in South African hominins (Cachel, 1975; Grine, 1981, 1986; Robinson, 1956; Wallace, 1975). However, now that a larger dental sample is available further inferences into this debate can be made. Additionally, unusual dental wear, such as through erosion or abrasion, has not been extensively explored, except in search of 'toothpick' grooves (Ungar et al., 2001; Wallace, 1974). In this thesis, all signs of unusual wear were recorded.

3.4. Chipping

Ante-mortem dental fractures, or chips as they are more commonly referred to, can give insight into diet and environmental contaminants as well cultural and food processing practices (Figure 3.2; Belcastro et al., 2007; Constantino et al., 2010; Daegling et al., 2013; Lucas et al., 2008; Scott & Winn, 2011). The teeth and crown positions most affected, as well

as the frequency and severity of chips, can all, therefore, give insight regarding diet and behaviour (Belcastro et al., 2007; Larsen, 2015; Lous, 1970; Molnar at al., 1972). Some authors have researched dental chipping in South African hominins, most commonly to compare chipping frequencies between *P. robustus* and *A. africanus* but also to look at chip size and masticatory processes (Constantino et al., 2010; Grine et al., 2010; Robinson, 1954; Tobias, 1967). However, there is much debate in the literature regarding frequencies as well as issues concerning methods used (see Chapter 5). Recent human groups and other extant primates have also been extensively studied, making a large database on which comparisons from the results of this thesis can be made (Belcastro et al., 2007; Constantino et al., 2012; Gould, 1968; Lous, 1970; Molnar et al., 1972; Scott & Winn, 2011; Silva et al., 2016; Turner & Cadien, 1969).

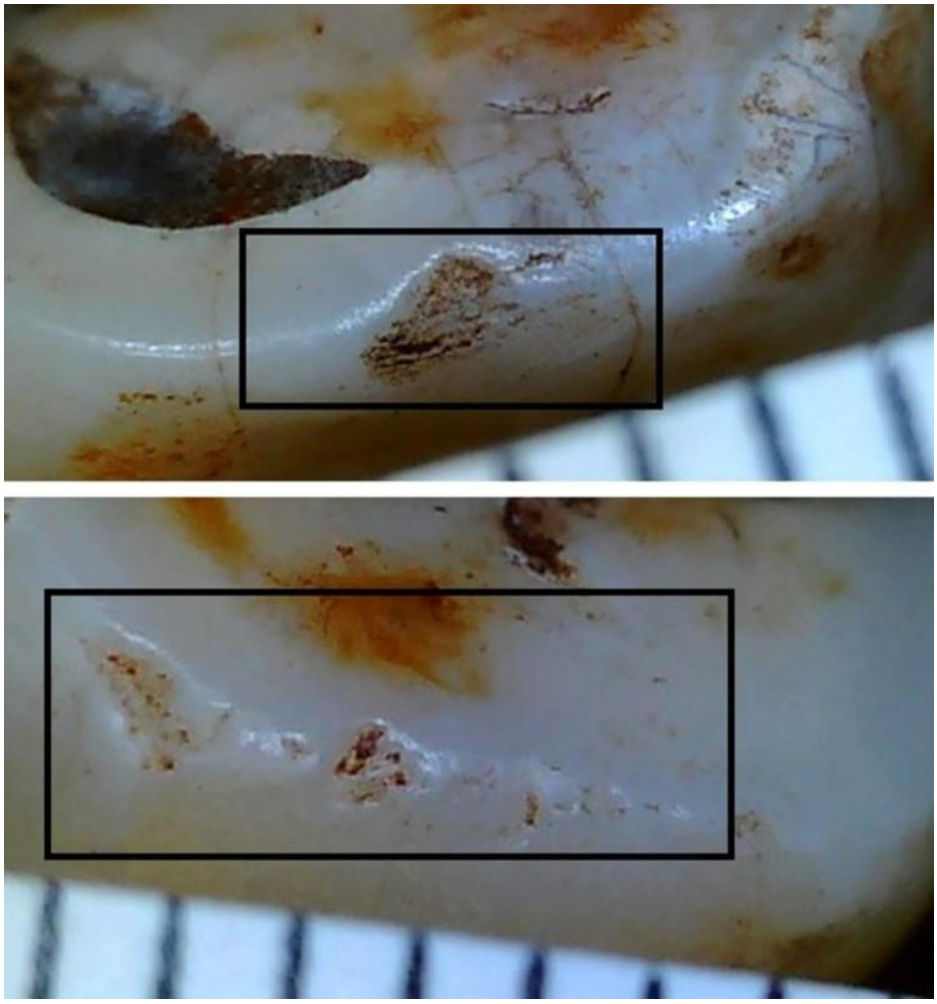

Figure 3.2. Chipping examples. Both teeth belong to *H. naledi*. Bottom image: UW 101–1401 upper right second premolar, multiple small chips on distal surface; top image: UW 101–1402 upper right first premolar, mesial chip. Scale is in millimetres.

3.5. Periodontal disease

Periodontal disease is a common condition in present-day humans and is predominately characterised by loss of alveolar bone (Figure 3.1). It results from the accumulation of specific bacteria close to the gum margins and is associated with certain diets, pathologies and oral hygiene behaviours (Clarke & Hirsch, 1991; Lieverse, 1999; Morris et al., 2001; Ogden, 2008). As well as recent humans, this condition has been recorded in a variety of hominin specimens (e.g., Gracia-Téllez., 2013; Ripamonti, 1988; Smith, 1977).

3.6. Dental abscesses

Abscessing and, more generally, periapical voids in the maxilla and mandible are found commonly in archaeological remains (Cawson et al., 2002; Ogden, 2008). The characteristics of the bone surrounding the void can give insight into its aetiology (Dias & Tayles, 1997). Heavy wear, trauma, and caries are often associated with abscesses, and therefore their presence can provide information on diet and behaviour (Hillson, 2005; Linn et al., 1987; Nair, 2004; Ricucci et al., 2006).

3.7. Calculus

Calculus is mineralized plaque which adheres to enamel surfaces (Figure 3.1; Greene et al., 2005). Its presence and location can provide information on diet as well as facilitate comparison with other pathologies. For example, there is a trend toward higher rates of periodontal disease with more calculus deposits (Greene et al., 2005), due to calculus displacing the epithelium around the gum line, allowing bacteria from living plaque to reach the alveolar bone. Both calculus and caries tend to increase when more carbohydrates are ingested, so an inference on diet can be gained from quantity and severity (Delgado-Darias et al., 2006; Larsen et al., 1991; Lieverse, 1999; Novak, 2015; Turner, 1979). Calculus is frequently recorded in modern day case studies (e.g., Keenleyside, 2008; Lieverse, 1999; Lillie, 1996; Lillie & Richards, 2000; Littleton & Frohlich, 1989; Pechenkina et al., 2002; Šlaus et al., 2011) and has been recorded in fossil hominins (Pap et al., 1995; Vandermeersch et al., 1994).

3.8. Enamel hypoplasia

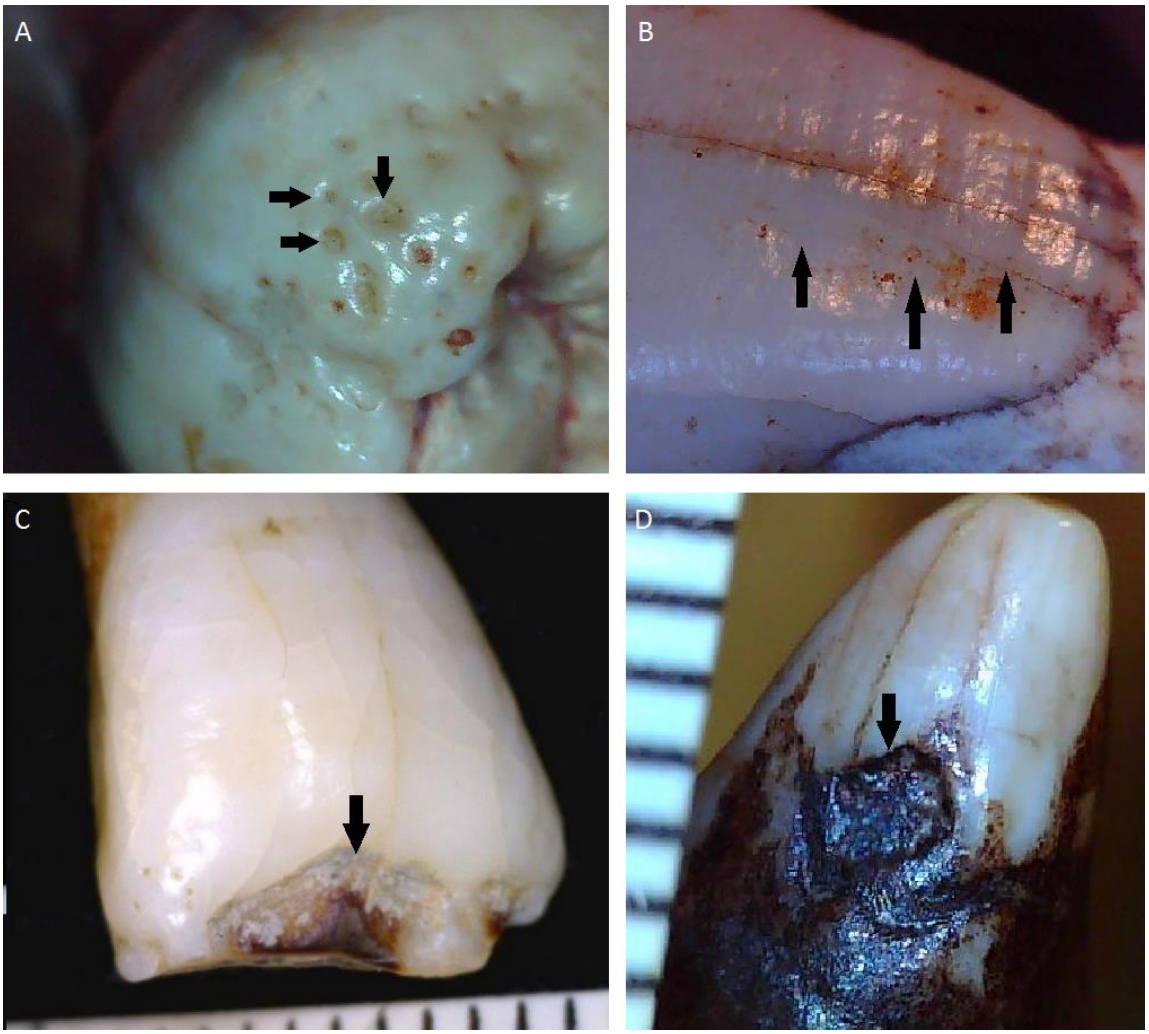

Figure 3.3. Enamel hypoplasia examples. A) pitting enamel hypoplasia (*A. africanus*, SK 9); B) linear enamel hypoplasia (*H. naledi*, UW 101-38). C) plane-form enamel hypoplasia (*H. sapiens*, Gloucester skeleton 1672); D) localised hypoplasia (gorilla, M 667).

Enamel hypoplasia are developmental defects appearing as grooves and pits, as well as more irregularly shapes, of missing enamel on the surface of a tooth crown (Figure 3.3; Goodman & Rose, 1990; Guatelli-Steinberg, 2015; Pindborg, 1970). Most types of hypoplasia are caused by periods of stress, such as disease or malnutrition, which disrupt the usual productivity of ameloblasts (Guatelli-Steinberg, 2003; Hillson, 2014; Weerheijm, 2003). Although the exact cause of such periods of systemic stress can often not be deduced from archaeological remains, they nonetheless record a very accurate account of duration and

severity of stress episodes (Goodman et al., 1980; Goodman et al., 1984; Goodman & Armelagos, 1985). Although few in number there have been some studies looking at hypoplasia differences in hominin species, including those in South Africa (e.g., Bombin, 1990; Guatelli-Steinberg, 2004; Moggi-Cechi, 2000; White, 1978), as well as individual case studies (Robinson, 1956; Tobias, 1991). However, a large scale study looking at all types of enamel hypoplasia has not yet been conducted on this material. Enamel hypoplasia has also been extensively researched in recent humans, and other mammals, due to the insight defects can give into disturbed development (e.g., Guatelli-Steinberg, 2015; Hillson & Bond, 1997; Ogden et al., 2007). Some specific types of illness, disease, and trauma have been associated with certain types and forms of hypoplasia (Dirks et al., 2002; Hillson, 1996, 2014; Radu & Soficaru, 2016; Skinner et al., 2015). Other abnormalities that are often recorded include tooth rotation and malocclusion, most notably as widely reported in a *Homo floresiensis* specimen (Brown, 2004), as well as genetic conditions (e.g., Crawford, 2007; Mehta et al., 2013). Rare abnormalities are infrequently searched for in fossil hominins and therefore are almot certainly overlooked in certain collection.

3.9. South African Literature

Some of the most exciting research in biological anthropology in recent years has centred on new finds from South Africa. The first *A. sediba* fossils were discovered in 2008, and since then over 220 skeletal fragments have been extracted. Their cranial and post-cranial skeletal morphology shares much in common with *A. africanus* but also with contemporary and later members of *Homo*. It has even been suggested that they may represent a species that gave rise to the *Homo* genus (Berger, 2012). Their dental crown and root morphology also suggest a very close association with *A. africanus* as well as links with members of the genus *Homo* (i.e., relative to other australopithecine species) sharing numerous apomorphies (Irish et al., 2013). Already studied in these remains are various indicators of bipedalism, the upper limb and manipulative abilities, mandibular remains, lower limb, geology setting, diet, dental morphology, pelvis, phylogeny and the vertebral column (Carlson et al., 2011, 2016; Churchill et al., 2013; Daegling et al., 2016; de Ruiter, 2013; DeSilva et al., 2013; Dirks et al., 2010; Henry, 2012; Irish et al., 2013; Kibii et al., 2011; Kivell et al., 2011; Ledogar et al., 2016; Pickering et al., 2011; Schmid et al., 2013; Williams et al., 2013; Zipfel et al., 2011).

Homo naledi is an even more recent discovery, with the description of this species less than two years ago (Berger et al., 2015; Dirks et al., 2015). However, the open access nature of this material has meant numerous studies have already been conducted (e.g., Feuerriegel et al., 2017; Harcourt-Smith et al., 2015; Kivell et al., 2015; Williams et al., 2017), with several relating to the dentition (Berger et al., 2015; Cofran et al., 2016; M.M. Skinner et al., 2016).

Most of the early *Homo*, *P. robustus* and *A. africanus* specimens have been studied intensively over the past 50 years. Research has focussed on teeth, predominately studies looking at phylogeny and diet (e.g., see Clement & Hillson, 2013; Grine, 2013). Studies on dental chipping, linear enamel hypoplasia, and occlusal wear have been studied several times each in these specimens (Bombin, 1990; Clement & Hillson, 2013; Constantino et al., 2010; Grine, 1986; Guatelli-Steinberg, 2004; Moggi-Cechi, 2000; Robinson, 1954; Tobias, 1967; Ungar & Grine, 1991; White, 1978).

3.9.1. Other South African hominin sites

In this study, only hominin material from the cradle of humankind is studied. There are, however, samples from other parts of South Africa dating to the middle and upper Palaeolithic. These sites are briefly outlined here as their dates are not too far from the *H. naledi* material. A human skull from Hofmeyr has been dated to 36.2 thousand years ago (± 3.3), a morphological study found its closest affinities, perhaps surprisingly, with Upper Paleolithic Eurasians (Grine et al., 2007). Another South African site with hominin remains is Klasies River, which dates to the middle Palaeolithic. The remains include some teeth, a mandible and several other skeletal elements (Rightmire et al., 2006; Rightmire & Deacon, 2001). Dental pathology and wear have not been studied in detail in these specimens; however in the descriptive paper Rightmire & Deacon (2001) do mention an interproximal groove on an upper second molar.

3.10. Archaeological evidence

Although a controversial subject, it is worth mentioning the use of fire in early hominins due to the clear impact on diet, social systems, and the ecological niche filled. Evidence for the period 1-2.5 million years ago is limited, with the possible exception of Swartkrans. Brain (1993) found that 270 specimens of animal bone at Swartkrans appear to

have been deliberately burnt, along with evidence for 20 hearths in one section of the cave. The fact that this evidence was found in different areas (and ages) of the site led to the suggestion of long-term fire use by hominins from before 1 Ma. Such potential evidence is only found in Member 3 deposits (Brain, 1993; Brain & Sillen, 1988). It is, therefore, worth keeping in mind that fire may have been exploited by some hominins studied here. The date of the *H. naledi* material, as well as its intriguing deposition, suggest these individuals may have been directly introduced to fire use by other hominins or were perhaps even efficient in its use and transport.

Stone and bone artefacts have also been found at these sites. These include stone artefacts found by Brain in loose breccia at the 'Extension Site' at Sterkfontein, followed by in situ finds of Robinson and Mason in 1957 and 1958 -- adding 286 lithics and a bone tool (Grine, 2013). Many cultural artefacts have been found at Swartkrans, with Members 1, 2, and 3 all containing stone tools (Clark, 1993). Additionally, over 80 limb bones and horns from bovids were shown to have been potentially used as tools (Backwell & d'Errico, 2001; Brain, 1989; Brain & Shipman, 1993; d'Errico & Backwell, 2003; Grine et al., 2015). The stone tool technology these materials should be defined as has been debated as well as which species likely used them, and what the different tools were used for (Backwell & d'Errico, 2001; Clark, 1993; Grine et al., 2015). However, evidence does suggest differences in frequency, types and processing of tools between site Members (Brain & Shipman, 1993; Clark, 1993; Pickering et al., 2008). As well as cut marks on animal bone and stone/bone tools found in these caves, another interesting observation is the presence of grooves on the skull of a hominin specimen (STW 53); the latter were suggested to be cut marks created by other hominins (Pickering et al., 2000). If true, this would likely signify the disarticulation of this individual. Usually the behaviours outlined above are suggested to be caused by species in the genus *Homo*. However, tool use has been suggested for *P. robustus*, with a suggestion of termite fishing (Backwell & d'Errico, 2001; Macho, 2015).

3.10.1. Other work on diet

For the most part, the current understanding of diet and behaviour in these hominin fossils is not influenced by dental pathology and developmental defects. Occlusal wear has received slightly more coverage. Daegling et al. (2013) provide an in-depth summary of the

most common methods for reconstructing diet in hominins. Below the general understanding of diet in South African hominins is explored with reference to these methods. These studies are used in the discussion section of this thesis (Chapter 12) to allow comparisons with the results.

3.10.2. Morphology

Dental morphology, biomechanics, and allometry have been studied extensively in South African hominins, either directly relating to diet, or with a focus on phylogeny but with a dietary or behavioural component to conclusions (Constantino et al., 2010; Daegling & Grine, 1991; Hylander, 1988; Jungers & Grine, 1986; Kay, 1985; Lucas et al., 2008; Rak, 1983; Robinson, 1952, 1954; Strait et al., 2009; Ungar, 2004; Wood & Abbott, 1983). The conclusions from such research has varied dramatically, and debate is very much ongoing (e.g., Grine et al., 2010; Strait et al., 2012).

It is well known that anterior and posterior teeth often play a significantly different role in mastication. For example, Cachel (1975) points out that anterior teeth are often used for processing foods, such as tearing and cropping, whereas posterior teeth tend to process the food through grinding and crushing. Therefore the overall size and morphology of teeth within the dental arcade may give insight into diet and food processing behaviour. Allometric factors will also play a role. A larger hominin will need larger posterior teeth, all else being equal, to allow a larger volume of food to be consumed (Cachel, 1975). Such factors are discussed and incorporated into the discussion of this thesis (Chapter 12).

As well as the morphology of the teeth, the overall shape of the dental arcade is also important. For example, the presence of diastema, how teeth occlude, and the palate shape will all have an effect on how the masticatory process affects teeth (Daegling et al., 2013; Robinson, 1954; Strait et al., 2012). Therefore, such factors are important to consider when comparing species, as they may influence the frequencies and position of different pathologies and wear. Due to the close phylogenetic relationship between the hominins studied in this thesis, it may be less of an issue than when comparing more distantly related species. Tobias (1967) highlighted such similarities between these species, stating there is little difference in overall shape of the dental arcade between *Paranthropus* specimens and other hominins. He also points out that these hominins are distinctly different from extant

non-human great apes. In particular, in extant non-human great apes, the canine and posterior teeth form an approximate straight line which is parallel and in some cases even divergent anteriorly. Additionally, they tend to have a diastema between the canine and anterior teeth. These two characteristics, in particular, are not found in these hominins.

3.10.3. Microwear

Microwear research has been carried out multiple times on South African hominin specimens, with conflicting conclusions. Microwear analysis involves comparing the number of microscopic scratches and pits on the crown of a tooth. Microwear analysis can, therefore, provide direct evidence to the foods that were processed and eaten by individuals in the days and weeks leading up to death. This technique has been used to suggest broad dietary categories for particular species as well as for distinguishing between subtle differences among closely related species (Grine, 1981, 1986; Grine et al., 2012; Merceron et al., 2004; Scott et al., 2005; Teaford & Glander, 1996; Teaford & Robinson, 1989; Ungar & Grine, 1991). However, in isolation, the precise cause of a wear pattern can often be difficult to interpret (Daegling et al., 2013). Early studies on microwear of *P. robustus* suggested hard objects may have made up a significant part of their diet. However, some of the more recent research has suggested that the microwear pattern may not be as conclusive (see Grine et al., 2012).

An issue with microwear analysis is that it can only give insight into foods consumed in the days leading up to death, so a bias may be introduced. One such bias may be created due to the individual being ill or weak and therefore not eating a ‘normal' diet. That said, this is one of the most direct and reliable methods of reconstructing diet in archaeological specimens, and reference to microwear results will regularly be made, particularly in Chapter 12.

3.10.4. Stable isotope analysis

Another direct line of dietary evidence comes from stable isotope analysis (Copeland et al., 2011; Sponheimer et al., 2005, 2006). Stable isotope analysis can give insight into the diet of past populations due to different foods leaving different isotopic signatures in the teeth of individuals. This method has been used on many hominin specimens over the last couple of decades (for an overview see Sponheimer et al., 2005; Sponheimer et al., 2013). C3 foods include fleshy fruits and hard objects such as seeds and nuts along with most other tree

biomass. C4 foods include items such as grasses and sedges but also animals that eat these foods. Therefore, one drawback of using this technique is that it can be difficult to identify the specific foods eaten. However, this can be partially overcome by also analysing certain potential foods in the area studied. For example, Sponheimer et al. (2005) took into consideration the C3/C4 values of termites and sedges in the South Africa when looking at stable isotope analysis in *P. robustus* and *A. africanus,* meaning the actual foods consumed could be narrowed down. It was expected that South African hominins would show C3 isotopic signatures, mainly due to our closest living relatives, chimpanzees and bonobos, having a predominantly frugivorous diet (Carter, 2001; Schoeninger et al., 1999).

All the carbon isotope studies conducted so far on *P. robustus* and *A. africanus* have shown a relatively strong C4 signature suggesting non-fruit foods must have been an important component of their diets (Sponheimer et al., 2005). Stable carbon isotope analysis has also been used in other mammals found at hominin fossil sites in South Africa (Lee-Thorp et al., 2007). From this data, the authors could then see if there were trends towards assemblages being dominated by certain types of diet, this could then provide environmental information. Their results highlight a general trend in the direction of open environments after 3 Ma, which could potentially help explain the higher than expected quantities of C4 foods in the diet of *P. robustus* and *A. africanus*.

As mentioned, bone tools found at Swartkrans have been suggested to be instruments for digging termite mounds. This has led researchers to suggest that the 13C-enriched isotopic signature in *P. robustus* may be caused by the consumption of termites which feed on C4 grasses (Backwell & d'Errico, 2001). However, although eating certain termites could produce the C4 levels needed to explain these results, they are very unlikely to be the only C4 food eaten as many termites are either C3 or mixed C3/4 (Sponheimer et al., 2005).

Earlier hominins seem to have had a diet closer to that of savannah chimpanzees, with *Ardipithecus ramidus* and *A. anamensis* diets dominated by C3 foods (Schoeninger et al., 1999). *Paranthropus boisei* on the other hand, shows even higher levels of C4 intake than the South African species suggesting they may have specialised in the consumption of a type of C4 vegetation or, less likely, species that fed on them. In contrast, *A. sediba* shows a remarkably different isotopic signature than the other South African hominins with a dietary

signal high in C3 material (Henry et al., 2012). These isotopic results are discussed in relation to the results of this study in Chapter 12.

3.10.5. General diet

Different dietary proxies discriminate different aspects of diet, meaning a full appreciation of the food consumed by a fossil hominin cannot be fully deciphered. For example, dental microwear predominantly provides information on the abrasiveness of foods consumed and isotopic analysis can infer the proportion of C3 and C4 foods eaten, but the actual foods consumed can only be approximated (Pineda-Munoz et al., 2016). Another important factor is the time scale that is actually being tested for diet. Different methods will be inferring diet at different times of the individual's life. For example, dental microwear is often referred, usually negatively, as the 'last supper effect' as these scratches and pits that are used to estimate dietary objects may be worn away in as little as weeks. The opposite is true of methods such as isotopic analysis in which the carbon and oxygen isotopes present while teeth are forming are incorporated into the enamel (Martínez et al., 2016). Such factors are also important to consider for the variables studied in this thesis. For example, dental chipping records periods of days to several years, depending on the size of fracture. Macrowear, caries, and calculus essentially measure diet over many years. Developmental defects on the other hand only form in the first few years of life. Therefore it may not be surprising that different methods show different results across species. Comparing different methods may, therefore, allow the most reliable conclusions on diet as a whole.

3.10.6. Tooth properties

When studying pathologies and wear, and particularly when comparing rates between different groups, it is important to take tooth properties into account. For example, the structure and morphology of the dentine, cementum, and enamel can all influence frequencies of the different pathologies and wear studied in this project (Lucas et al., 2008). These are discussed and taken into account in each chapter.

Over time there has been much debate about the fossil record regarding the evolution of human ontogeny (See Smith et al., 2015 and references therein). It has been suggested that Plio-Pleistocene hominins, including those studied here, show a developmental pattern more similar to extant non-human great apes than to modern humans (Bromage & Dean, 1985;

Dean et al. 1993). Such differences in timing makes a big difference to estimating the age of death of fossil hominins. For example, the age of death of the Taung child has been estimated to be anywhere between three and six depending on which great ape species is used as the proxy (Conroy & Vannier, 1991; Mann, 1988; McNulty et al., 2006). This goes back to the circular problem of using an extant species as a reference for studying developmental timings in extinct species. Attempts have been made however to counter this problem by using methods that do not depend on extant species (e.g., Smith et al., 2015). Difference in formation and eruption times between the samples in this project, particularly with the inclusion of the comparative extant primate samples are therefore vast. For example, chimpanzee deciduous teeth erupt at around half the age of modern humans, with, by the age of one and a half, all their deciduous teeth erupted (Smith & Tafforeau, 2008).

Chapter 4: Materials and methods

A total of 9,274 teeth were studied for this project; of these, 1,297 comprise South African hominin specimens and the rest are divided among the comparative samples. This material is summarised below, along with an overview of the methods used.

At the time of data collection, over 1,500 *H. naledi* specimens were available for study, from which over 15 individuals are represented (Berger et al., 2015; Dirks et al., 2015). The teeth, and the small number of mandibles and maxillae are in excellent condition. To the extent that they offer almost as good of preservation as the modern day comparative samples (Table 4.1). The *A. sediba* material is also very well preserved, however with much less dental material represented. *Paranthropus robustus* and *A. africanus* are the largest samples but also have the highest rate of post-mortem broken teeth, both in total and proportionally. That said, given their large sample sizes, the amount of teeth observable for all variables is high (Table 4.1). Broken teeth are not recorded when post-mortem damage renders multiple pathologies, wear, and developmental defects impossible to record. However, in certain circumstances, such teeth can be recorded for particular variables. For example, if a tooth is broken half way down the crown, yet the occlusal surface is still fully visible, then occlusal wear can be recorded. The criterion for each variable is explained in the subsequent chapters.

Table 4.1. Number of permanent teeth for each sample.

Species	Observable Teeth	Broken Teeth	Total Teeth	% Broken
H. naledi	147	12	159	7.55
A. sediba	16	4	20	20.00
P. robustus	318	136	454	29.96
A. africanus	328	164	492	33.33
Early *Homo*	44	23	67	34.33
Olive baboons	790	2	792	0.25
Chimpanzees	2498	5	2503	0.20
Drills	212	0	212	0.00
Gorillas	2090	10	2100	0.48
Hamadryas baboons	90	0	90	0.00
H. sapiens	918	3	921	0.33
Total	7451	359	7810	4.60

The deciduous tooth sample is significantly smaller than the permanent sample in both the hominins and comparative primates. There are also different proportions for the different species, with *P. robustus* having by far the largest sample size of deciduous teeth (Figure 4.2). The other hominin samples are relatively small, making comparisons for certain pathologies difficult.

Table 4.2. Number of deciduous teeth for each sample.

Species	Observable Teeth	Broken Teeth	Total Teeth	% Broken
Hominins				
H. naledi	20	2	22	9.09
P. robustus	46	11	57	19.30
A. africanus	7	5	12	41.67
Early *Homo*	13	1	14	7.14
Comparative samples				
Olive baboons	75	0	75	0.00
Chimpanzees	749	4	753	0.53
Drills	34	0	34	0.00
Gorillas	495	2	497	0.40
Totals	1439	25	1464	1.71

The *A. africanus* material originates from two sites, Sterkfontein and Makapansgat. *P. robustus* specimens are from Kromdraai and Swartkrans, with additional observations added from the literature on specimens from Drimolen and Coopers. The early *Homo* material originates from Sterkfontein and Swartkrans. Both *H. naledi* and *A. sediba* are only found at one site, Rising Star and Malapa, respectively. Table 4.3 displays the number of teeth represented for each site.

As discussed in the preceding chapters, much debate in the literature has centred upon phylogeny for certain specimens. However, most are consistently identified into the five main species used in this thesis due to sharing key morphological traits. The few examples where this is not the case is due to the specimen being small/broken or because a variety of characteristics from two or more species has been argued. For distinguishing which samples should be included in these different categories, the following three articles are predominantly used (Berger et al., 2015; Grine et al., 2013; Moggi-Cecchi et al., 2006).

Table 4.3. Number of teeth for each hominin site, and the percentage of all teeth that are deciduous.

Species/Site	Permanent Teeth	Deciduous Teeth	% Deciduous
H. naledi			
Rising Star	159	22	12.15
A. sediba			
Malapa	20	0	0.00
P. robustus			
Kromdraai	30	9	23.08
Swartkrans	424	48	10.17
A. africanus			
Makapansgat	45	2	4.26
Sterkfontein	447	10	2.19
Early *Homo*			
Sterkfontein	47	12	20.34
Swartkrans	20	2	9.09

The vast majority of specimens from Sterkfontein are assigned to *A. africanus*, with those assigned to *Homo* including: SE 255, SE 1508, SE 1937, STW 53, STW 75, STW 80 and STW 151, and one is assigned to *P. robustus*: STW 566 (Clarke, 1985; Hughes & Tobias, 1977; Kuman & Clarke, 2000; Moggi-Cecchi et al., 1998, 2006; Robinson, 1963; Tobias, 1965; 1978). STW 80 and STW 151, in particular, are both classified regularly as *A. africanus* and early *Homo*. Here they are counted as early *Homo*, although the debate is very much ongoing to their classification (Braga & Thackeray, 2003; Grine et al., 1993; Moggi-Cecchi et al., 1998). Specimens from Swartkrans are predominantly assigned to *P. robustus*, with the following specimens regarded as early *Homo*: SK 15, SK 27, SK 43, SK 45, SK 2635 (Berger et al., 2015; Grine et al., 2013; Moggi-Cecchi et al., 2006). The Kromdraai specimens are all classified as *P. robustus*, and the Makapansgat specimens all *A. africanus*. A full list of specimen studied from each site can be found in Appendix A for permanent teeth and Appendix B for deciduous teeth.

A couple of teeth have been sacrificed for particular studies over the years, such as isotopic analysis, and therefore only the casts are left to study (see specimen list in Moggi-Cecchi et al., 2006). Although the casts can be of extremely high quality, it is not at present known how much bias would be introduced if recording pathologies and wear on these casts was compared to the real tooth. Therefore data on casts are not presented.

As mentioned in the introduction, the fossil record is not made up of equal amounts of different skeletal elements. Even teeth, which are by far the most frequently preserved elements, have variable frequencies of preservation depending on what part of the jaw they are from. The main factor is that anterior teeth tend to have less complex roots and therefore are more likely to be dislodged during taphonomic processes and subsequently washed away. This effect can be strikingly seen in this sample, with posterior teeth preserved in much greater numbers than anterior, in both dentitions (Table 4.4). These differences justify splitting the results into tooth types, especially with variables that show significant differences between different teeth.

Table 4.4. Total number of teeth for all hominins. # per tooth position refers to the total number of teeth divided by the number of tooth positions for that tooth type.

Tooth type	Permanent teeth	# per tooth position	Deciduous teeth	# per tooth position
Incisors	185	23.13	12	1.50
Canines	124	31.00	20	5.00
Premolars	317	39.63	n/a	n/a
Molars	567	47.25	73	9.13

4.1. Comparative samples

The primate comparative sample was chosen because of the close phylogenetic relationship of gorillas and chimpanzees with hominins as well as similar behavioural and environmental factors suggested in the literature for baboons (e.g., Codron et al., 2008; Jolly, 1970; Macho, 2014). Data were collected in material at the Powell-Cotton Museum, which was amassed by Percy Powell-Cotton and includes a variety of faunal remains as well as botanical and ethnographic specimens. He collected the primate material during expeditions to Africa between 1887 and 1939 (Guatelli-Steinberg & Skinner, 2000). The majority of the primate specimens included here were collected between 1927 and 1936, with the great apes killed in their natural habitat in Cameroon and the Congo (Dean & Jones, 1992; Lukacs, 2001). Most of the samples were collected near the villages Batouri and Lomie in south-east Cameroon, with rainfall for the region averaging 1500 mm/year and an altitude of up to 750 meters above sea level (Lukacs, 2001). The great ape specimens include western lowland gorillas and common chimpanzees. The baboon sample consists of olive and hamadryas

baboons that, along with the drill sample, are all wild shot and come from a variety of locations (Guatelli-Steinberg & Skinner, 2000). The majority of monkey specimens are olive baboons (Table 4.1), and for most variables, only olive baboons are displayed due to small sample sizes for drills and hamadryas baboons. Therefore, the main three comparative extant primate species are western lowland gorilla (*Gorilla gorilla gorilla*), common chimpanzees (*Pan troglodytes*) and olive baboons (*Papio Anubis*), and unless stated when chimpanzees, gorillas and baboons are mentioned it refers to these three species/subspecies.

Due to the quality of this collection for research on wild specimens, many interesting studies have been conducted. Manning and Chamberlain (1993) used the collection to look at asymmetry in the canine teeth of Old World primates, with the hope of providing insight into sexual selection. Measurements of the size and shape of the teeth have also been used in research looking at phylogenetic relationships (Wood & Willoughby, 1991). Lukacs (2001) studied enamel hypoplasia in deciduous teeth, finding that gorillas and orangutans have significantly more enamel defects on their canines than chimpanzees. Skinner (1986) compared LEH rates in chimpanzees and gorillas from this collection, finding that the latter had more LEH grooves than the former. Later research by Guatelli-Steinberg and Skinner (2000) again looked at LEH frequencies in these same primates but with a focus on differences between a larger sample of primates and the influence of crown height on LEH rates. Dean and Jones (1992) looked at the relationship between tooth wear, continuous eruption and periodontal disease concluding that when studies look at periodontal disease in archaeological material continuous eruption is an important component to consider. The teeth in this collection have also been used in research on ageing individuals (Dean & Wood, 1981), sexual dimorphism (Wood, 1976), and abscesses (Legge, 2012).

For comparisons with the fossil hominins it is important to incorporate dietary data on the extant primate samples. Western lowland gorillas eat higher quantities of fruit than other gorillas (Tutin, 1996), but leaf matter still makes up a large proportion of their diet (Table 4.5). This diet varies over the course of the year, with fruits eaten more regularly in the wet season (Conklin-Brittain et al., 2001). Chimpanzees are best described as omnivorous frugivores, eating more fruit and less leaf matter than gorillas (Table 4.5). In addition to leaves, fruit and other vegetation, chimpanzees have also been observed eating birds, small mammals, eggs, insects, and honey, although these make up a small portion of their diet

(Table 4.5; Isabirye-Basuta, 1989; Boesch et al., 2002; Phillips & Lancelotti, 2014). Baboons, in general, are also omnivorous, although their diet is predominated by herbaceous material (Table 4.5). Olive baboons in particular are noted for their varied diet which differs from place to place because they are highly adaptable (Whiten et al., 2011). Baboon diet can include fruits, tubers and leaf matter but also insects, roots, eggs and animal material (Macho, 2014; Lodge et al., 2013). Therefore, these three groups of primates are justifiably good specimens to use as comparisons with the fossil hominin since their diets differ substantially from each other and cover a wide range of food items.

Table 4.5. Percentage of different food items in the diet of wild gorillas, chimpanzees and baboons (Conklin-Brittain et al., 2001; DeCasien et al., 2017; Doran et al., 2002; Leonardet al., 2003; Mitani et al., 2012).

Species	Leaf matter	Fruits and seeds	Other*
Gorilla	37	63	0
Chimpanzee	16.6	77	6.4
Baboons	34.79	53.5	11.71

*Predominantly animal matter

A modern human comparative sample was also studied, with the same methods used. This medieval collection from Gloucester, UK, consists of 41 individuals (Table 4.1). A variety of work has been completed on the diet and health during this period, so inferences into the effect on dental pathology and developmental defects are possible (Albarella, 2006; Connell et al., 2012; Novak, 2015; Thomas et al., 1997; Wadsworth, 1992; Woolgar et al., 2006).

4.2. Background to methods

The methods used for each species and site, including the comparative samples, are the same. Methods were selected based on their appropriateness for recording fossil teeth. The most important selection criteria, therefore, were methods that did not rely on whole jaws for comparisons. Methods that use colouration differences were also not used, as post-mortem processes can often affect colour. Finally, methods used had to be appropriate for all species and populations studied, and therefore not reliant on certain morphological features. These criteria were easy to meet, with most commonly used techniques usually appropriate.

There have been numerous recording systems created to allow successful collection and analysis of dental data. For this research only methods that take into account hard tissue

are applicable, but other issues are important to consider when looking at archaeological remains. For example, using DMF scores (Decayed Missing Filled; Broadbent & Thomson, 2005) is not appropriate, because if teeth are missing (e.g. through periodontal disease or post-mortem tooth loss) an accurate total cannot be calculated. Therefore, appropriate recording methods are selected by using relevant systems already published, or altering them slightly, allowing direct comparison with published data.

The position of pathologies and defects were recorded, both the tooth type affected but also the position on the tooth, with analysis often split by such groupings. Figure 4.1 highlights the terminology used in this thesis to refer to these different positions since there is often confusion created by the variety of terminology in current use. Perhaps the most note-worthy aspects are the use of the word buccal for all teeth, the premolars being called 1 and 2 rather than 3 and 4, and posterior deciduous teeth being called deciduous molars, not premolars. Although the use of the word buccal for use on incisors and canines, rather than labial, is technically wrong, it is gaining popularity due to being much easier to use regarding descriptions and comparisons. Therefore buccal in this thesis refers to the outside (i.e. the side not facing into the oral cavity) of all teeth.

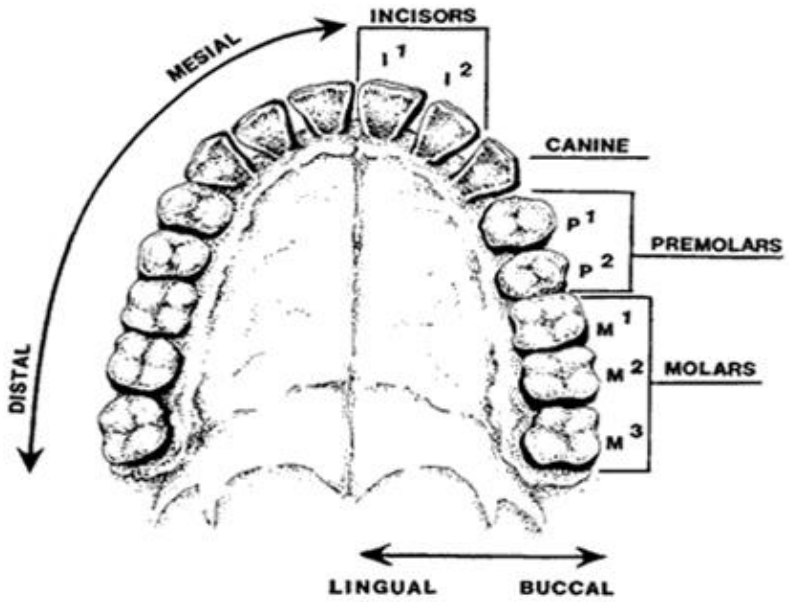

Figure 4.1. Common terminology used in this thesis. Image credit: Lorraine Heidecker (Redwoods.edu). Teeth shown are all the upper permanent teeth.

Dental pathologies, wear, and developmental defects can lead to increased levels of ante-mortem tooth loss in a population. Similarly, many of these variables can be worn away during life. For example, any trace of a chip, a carious lesion, or developmental defect can be lost through attrition long before death. Therefore, in studies such as this, the actual number of such indicators that existed on an individual's dentition in their lifetime is not what is displayed. Instead, what is presented is a snap shot of the pathologies and wear that were present when the individual died. This has led to a variety of authors suggesting methods to attempt to counteract for such factors (e.g., Duyar & Erdal, 2003; Kelley et al., 1991; Lukacs, 1995). However, given the complex interactions between these different factors and the fact that the true overall rate (whatever that may mean) for most variables is not possible to reconstruct, the best way to display results is the actual frequencies found for the different pathologies. For example, in modern human studies in which the vast majority of ante-mortem tooth loss is through caries, perhaps such correction factors are justified, but in these fossil hominins, ante-mortem tooth loss is likely caused a variety of factors, but most commonly heavy attrition. Therefore, when comparing populations in which the interaction between different pathologies, defects and wear is complicated and not fully understood such corrective methods may lead to misleading results. Instead, each variable is displayed as found and the interaction between the different pathologies and wear explored. This method is now commonly used (e.g., Meinl et al., 2010; Novak, 2015).

Developmental defects are perhaps the exception to this rule. With increasing wear, all else being equal, fewer enamel defects should be visible on a tooth crown. This is due to the defects being worn away as a tooth wears towards the cementum enamel junction (CEJ). Therefore methods to attempt to counteract the defects lost through wear are justified (see Chapter 6). The best way to do this is to simply split teeth into wear categories and only compare similarly worn teeth between species.

A number of steps were taken to prevent biases being introduced due to recording data at the different times and locations. The same lamp (Lumen MDL-NR-942, 230V) was used throughout to prevent any effects of lighting affecting pathology frequencies. This is particularly important in the recording of enamel hypoplasia. All teeth were placed on the same black cloth, and the same hand lens was used (10x).

How the fossils have been curated and arranged have the potential to create biases in the data set. For example, the loose teeth in the Sterkfontein collection at the University of the Witwatersrand have boxes full of loose teeth of a certain type, e.g. lower second molars, clearly from a variety of different individuals, but they are curated this way to help with studies, such as on dental morphology. Whereas the Rising Star material was (at the time of collecting the data) arranged in individuals. Material from Distong museum has a mixture of these different techniques. These various curator techniques highlight the importance of using identical methods and the same amount of time for each specimen studied.

4.3. Recording and quantitative methods

It is useful to be able to split deciduous and permanent teeth up for analysis due to differences in formation times, morphology, and tissue properties. Therefore individuals that have both permanent and deciduous teeth are split up, and most analysis in this thesis is done separately for the two dentitions. Unless stated, all analysis is done by tooth and not by individual. This is due to the scarcity of complete, or close to complete, hominin dentitions, but has the added benefit of the majority of literature also using per tooth frequencies. The scale in all figures is in mm unless otherwise stated.

Figure 4.2 shows the recording form used for recording all samples. Deciduous teeth are simply noted with a “d” on the appropriate tooth position. This technique allows the same sequence of recording, minimising bias but also making it difficult to repeat or neglect particular variables. A mark of 0 is used to signify that a pathology is not present but that the area of the tooth that it would appear on is present. Teeth where it would not be possible to tell if the pathology is present are marked with an 8 (except for wear data). If just left blank then the figure would be inflated because teeth that are broken may have had a particular pathology during life and now due to post-mortem damage that pathology is not visible. Therefore, although shrinking the sample size for the different pathologies, the only fair way to get an accurate reading of the rates is only to include teeth where it can be recorded with near certainty that a certain pathology could have been present ante-mortem. Such processes are becoming more common to attempt to reduce error. However, older studies often display these frequencies as a percent of all teeth. Such study differences will be explored by comparing rates with and without the inclusion of broken teeth (Table 4.1).

A difficulty occurs when trying to record a tooth that has post-mortem damage, yet a defect or pathology is partly visible. These pathologies are recorded in notes, as they are still useful information to have, but because the tooth is marked as broken in most analysis these teeth are excluded. In each chapter, the exact protocol for inclusion is defined. If a tooth is recorded as broken in Table 4.1, it means most pathologies and defects cannot be recorded, but a certain variable may still be possible to record. For example, if a tooth is broken, but the occlusal surface is still complete, it can still be registered for wear. Due to this, the number of teeth used in the different chapters varies slightly. The sample of South African hominins is now large enough to allow detailed analysis of only complete, undamaged, teeth, and therefore this is the most reliable method to employ. Teeth are therefore marked as present if the vast majority of the crown is present and at least the upper most part of the root(s). Only the most superficial damage is acceptable, in the area in which the variables would be affected. Teeth missing parts of the roots and crown are therefore marked as broken, likewise if only part of the tooth is visible in breccia.

Loose teeth were recorded individually for wear first, and then the other pathologies and defects were checked for in the same order as they are on the form (Figure 4.2). Collecting data in this order helped reduce the risk of overlooking a certain pathology. Teeth were viewed using the naked eye at first and then a 10x hand lens to double check for and confirm pathologies. A lamp was used to highlight the tooth surface. Teeth were placed on black cloth which was also used for photographs unless the tooth was partially black in which case white cardboard was used. After the recording form was completed any pathologies that were identified were photographed using a Dino-Lite® camera followed by numerous photos of the whole tooth using a Ricoh® digital camera (Ricoh GR compact digital camera, macro setting; Dino-Lite AM2111 handheld microscope). The photos were then added to a file so that all were in the same place, filed under the date and the specimen number. A brief summary of the tooth was then written in Microsoft Word unless there was nothing significant to note.

Dental Pathology: **Maxilla**

Institution:_____ Site:_____ Context/Skeleton number:_____ Age:_____ Date:_____

	Right								**Left**							
Maxilla	**M3**	**M2**	**M1**	**PM2**	**PM1**	**C**	**I2**	**I1**	**I1**	**I2**	**C**	**PM1**	**PM2**	**M1**	**M2**	**M3**
Inventory																
Attrition Score																
I, C and PM (1-8)																
Mesio-Buccal (1-10)																
Mesio-Lingual (1-10)																
Disto- Lingual (1-10)																
Disto- Buccal (1-10)																
Calculus (0-3)																
Calculus Location (B, L)																
SDF: Present (1);NotPresent(2) No dentine=0 (can't view=8)																
Chipping (0-3)																
Chipping Location																
Abscess(0-observable: not present,severity: 1-3) 8- unobservable																
Periodontitis General (0-3)																
Pockets (0-3)																
Enamel Hypoplasia																
Systemic/Localised (1,2)																
LEH (1)/PEH (2) Both (3)																
Amelogenesis Imperf Dentinogenesis Imperf Fluorosis (0-5)																

Supernumerary teeth/other anomalies/unusual wear patterns:

Figure 4.2. Recording form used for all samples. The mandible is the same except the right and left are reversed. The number '8' is marked if the variable is not recordable (except wear). See text both details.

4.4. Specific methods

Specimens are individually examined regardless of any adjacent teeth. Macroscopic inspection of each tooth is carried out within each museum under good lighting. A 10x hand lens is used for certain pathologies as well as to confirm the presence of others (e.g., LEH and caries). Statistical tests used are described in each section, with multi-variable analysis performed in Chapter 11.

For caries, both location and severity was recorded, the former following Schollmeyer and Turner (2004) and the later Koritzer (1977). Due to difficulties with recording early stage caries in archaeological remains, this 1-4 severity scale is preferred over other methods such as that proposed by Hillson (2001). Calculus, periodontitis and abscesses are measured on a scale of 1 to 3 (Brothwell, 1981; Ogden, 2008). The location, severity and type of enamel hypoplasia is recorded. Additionally, the distance in mm from the cementoenamel junction (CEJ) to the defect, for LEH, is recorded, and whether it is acute or chronic in nature. Tooth wear is recorded in different ways for the anterior and posterior teeth. Molars are scored on a scale of 1-10 following Scott (1979) with each tooth being divided into quadrants so that direction of wear can be inferred. Anterior teeth are scored on a scale of 1-8 following Smith (1984). The position, severity, and number of dental chips are also recorded. Severity is based on the three-point scale of Bonfiglioli et al. (2004) and the number of chips following Belcastro et al. (2007). Tertiary dentine is marked as present or absent for each tooth that has dentine exposed ante-mortem (Geissler et al., 2015; Pampush et al., 2016). An in-depth description of these different methods are given in the appropriate chapters.

The benefit of using micro-CT scans to study teeth has long been recognised (e.g. Rossi et al., 2004; Swain & Xue, 2009). These scans have been used to identify and interpret pathologies including caries, calculus, enamel hypoplasia, and hereditary reduction of enamel thickness or density (Crawford et al., 2007; Marchewka et al., 2014; McErlain et al., 2004; Neves et al., 2010; Schuurs, 2012; Xing et al., 2015). Micro-CT scans are also regularly used in studies to compare the morphology and thickness of different dental tissues (Kato et al., 2011; Kono, 2004; Xing et al., 2015), and have even been used to disprove the notion that a *H. floresiensis* tooth contained a filling (Jungers & Kaifu, 2011). In Chapters 6 and 7 micro-CT scans are used to show the presence and extent of caries and enamel hypoplasia.

To assess intraobserver error, 218 baboon teeth were recorded on two separate occasions (see Chapter 5). Interobserver error was not possible to conduct directly on the fossil hominins during this project; however previous research using the same methods made evaluations possible. Therefore, for chipping (Chapter 5) and enemal hypolasia (Chapter 6) interobserver error is assessed through comparsons with studies already carried out on these specimens that have used the same methods. For caries, abcesses, periodontal disease, and ATML all cases are presented individually due to small sample sizes, negating the need for interobserver error since all cases are described and discussed separately.

Chapter 5: Chipping

5.1. Introduction

Dental fractures, or ante-mortem chips as they are more commonly described, have been recorded in numerous populations and species. These chips are produced when a tooth comes into contact with a hard object with sufficient force to fracture dental tissue (Chai & Lawn, 2007; Constantino et al., 2010). Most commonly this process affects the enamel as it tends to be the material most exposed in the oral cavity. Because enamel is strong, but brittle, a chip is created with little prior plastic deformation (Thomas, 2000). Consequently, although enamel can sustain considerable stress before breaking, a fracture may occur when this threshold is reached (Constantino et al., 2010; Scott & Winn, 2011). Chipping is technically a type of dental wear, due to crown height being reduced. However, it differs from other forms of wear in not being a gradual process and tending to not leave a smooth occlusal surface. Instead, irregular fractures commonly form on the occlusal edge of the enamel, with, in severe cases, the dentine involved. Different dietary items cause enamel fractures at different rates and sizes -- from soft fruits that rarely cause chipping to hard seeds and nuts that may lead to large chips. However, the propensity of some foods for dental chipping is harder to discern. Bark and low-quality terrestrial herbaceous vegetation tend to envelope the crown surface, thereby spreading out stresses to make chipping unlikely (Chai et al., 2009; Lucas et al., 2008). Environmental contaminants may also be important, such as grit incorporated into the diet (Belcastro et al., 2007; Nystrom et al., 2004). The size and shape that an object must be to cause chipping are subjects of debate (e.g., Constantino et al., 2012; Daegling et al., 2013; Lucas et al., 2008); yet, the teeth affected, position on the tooth, and severity can all give insight into the etiology producing such chips (Belcastro et al., 2007; Constantino et al., 2010, 2012; Sauther et al., 2002; Scott & Winn, 2011; Stojanowski et al., 2015; Van Valkenburgh, 2009).

Comparatively low chipping rates are found in gorillas and chimpanzees relative to orangutans (Constantino et al., 2012). The rate in gorillas is a result of their infrequent ingestion of hard seeds and fruits while feeding predominantly on foods like low-quality herbaceous vegetation (Conklin-Brittain et al., 2001; Doran et al., 2002). Similarly,

chimpanzees commonly consume soft fruits (Conklin-Brittain et al., 2001). Orangutans, however, have far higher chipping rates than other great apes, with Constantino et al. (2012) reporting three to six times more chips on their posterior teeth. This high rate is attributed to the large hard foods that make up a significant part of their diet (Galdikas, 1982).

A variety of recent human populations have also been studied (e.g., Belcastro et al., 2007; Bonfiglioli et al., 2004; Gould, 1968; Lous, 1970; Molnar et al., 1972; Scott & Winn, 2011; Silva et al., 2016; Turner & Cadien, 1969), and the findings are useful for inferring chipping etiologies in fossil hominins. In general, hunter-gatherers tend to have higher rates in their posterior teeth, whereas agriculturalists have more chipping of the anterior teeth. Also, the former groups are most affected by diet or environmental contaminants, while the latter are more often affected by diet and tool use (Scott & Winn, 2011; Stojanowski et al., 2015). Non-masticatory behaviour is usually the focus of chipping studies in *H. sapiens*, with different activities leading to a variety of patterns (e.g., Bonfiglioli et al., 2004; Gould, 1968; Larsen, 2015; Lous, 1970; Molnar at al., 1972). Chipping frequencies may also allow insight into sex and social status differences. In modern day populations, this mainly reflects non-masticatory activities, but dietary differences may also be inferred. A dietary example of differences within a population is that of Australian and African foragers that actively hunt or fish more meat/fish than other members of their group (Bonfiglioli et al., 2004; Larsen, 2015). Non-masticatory behaviour has also been shown to cause different frequencies of dental chipping between the sexes (Gould, 1968; Lous, 1970; Molnar, 1972).

Chipping frequencies have also been recorded in hominin fossils, with South African specimens particularly well studied (Constantino et al., 2010; Grine et al., 2010; Robinson, 1954; Tobias, 1967). For example, there has been much debate in the literature concerning what the frequencies of dental chipping in *P. robustus* and *A. africanus* indicate in terms of diet. Alternate explanations include grit introduced into the masticatory process from eating roots (Robinson, 1954), crunching of bones (Tobias, 1967), and consumption of seeds and nuts (Constantino et al., 2010). Chipping has also been noted in the teeth of *A. afarensis* (Johanson & Taieb, 1976), *A. anamensis* (Ward et al., 2001), *P. boisei* (Tobias, 1967), and *H. neanderthalensis* (Fox & Frayer, 1997). Neanderthal teeth exhibit high rates that are likely caused, at least in part, by non-masticatory processes (Fiorenza & Kullmer, 2013; Fox & Frayer, 1997).

Although dental chipping can be useful in reconstructing hominin diets, a few issues have not yet been thoroughly addressed in the literature, including the effects that enamel microstructure, thickness, and morphology have on susceptibility to fracture, and the time spent in occlusion and wear of the tooth. It has been suggested that fractures may follow lines of weakness such as lamellae and tufts, which means cracks can form differentially or more easily at certain locations (Lucas et al., 2008). Similarly, the orientation of enamel microstructure, as well as the dietary object is important (Xu et al., 1998). Most research on fractures assumes that enamel has similar properties across the occlusal surface, as well as between tooth types and populations. However, more recent work suggests that enamel mechanical properties differ across the surface of a single tooth, as well as between teeth (Cuy et al., 2002; Macho & Shimizu, 2009; Ziscovici et al., 2014). Enamel property differences among species could also mean that two closely related species with nearly identical diets have markedly different patterns of chipping. In this regard, it was proposed that thick enamel may have evolved in certain lineages to resist tooth loss through fracture (Kay, 1981; Lucas et al., 2008). This possibility could lead to bias in the data if these same species evolved other adaptations to cope with consuming large amounts of hard foods. Efforts to quantify bite forces in extinct species may be especially influenced by such factors (Chai & Lawn, 2007; Chai et al., 2011; Constantino et al., 2010, 2012). However, species differences are just beginning to be researched (e.g., Ziscovici et al., 2014). There are also issues concerning how samples are chosen, such as the inclusion of incomplete crowns and differences in the presentation of results that may yield substantial differences among studies of the same species (Daegling et al., 2013).

That said, if care is taken in choosing the methods and enamel property differences are considered, dental chipping should be able to provide some insight into the diet and behaviour of extinct species. By recording position, severity, and frequency, a unique sample-specific pattern may be obtained for comparison with other samples.

Sections of this chapter have incorporated edited text, figures, and tables from Towle et al. (2017). Additional analysis and interpretations are added in this thesis, with comparisons made between species, but chipping patterns for each hominin species are also explored separately.

5.2. Materials and methods

A variety of methods have been used to record chipping, although simply recording presence/absence on a tooth is often used. Other methods for separating chips depending on their severity include Pattersons' (1984) technique of splitting fractures into two categories (minor enamel chipping vs. major segments of enamel loss). This is only slightly different from the method used in this study with effectively severity one and two being equivalent to "minor enamel chipping" and severity three to "major segments of enamel loss". The Bonfiglioli et al. (2004) method is favoured in this research due to its finer scale of distinguishing between chips. Another example is that of Turner et al. (1991) in only recording individual chips when less than 10 teeth in a dentition show chipping, if more; it is referred to as generalised. This method made sense as the focus of their study was on dental morphology. This highlights the issue of making sure methods are compared before frequencies between studies are contrasted. The inclusion of post-mortem damaged teeth and differences in presenting results may also cause significant differences between studies looking at the sample (Daegling et al., 2013).

Similarly, some studies have only looked at frequencies of chipping per individual and not per tooth (e.g., Turner & Cadien, 1969). The drawback of using this method has been pointed out by Scott & Winn (2011). The main issue is that bias will be introduced if only certain teeth represent an individual, as different tooth types may be more likely to have enamel fractures in different populations. The number of teeth represented by each individual will have a similar effect and may introduce error. These issues are heightened when looking at older archaeological material in which the only remains from an individual may be one or two teeth.

In other animal groups, because the chipping is caused by different dietary factors, chips are often called and described differently. For example, there has been research on dental chipping in carnivores, but this is usually called fractures or breakages rather than chips. This is because the most striking cases are on canines which have broken due to high impacts such as hunting activities or interspecies conflicts (Valkenburgh, 1988). Apart from terminology, another issue in species comparisons is methods used to define anti-mortem chipping. It is common only to include teeth that show further wear on the chip to allow only

ante-mortem chips to be included (Scott & Winn, 2011). However, some researchers have limits on the size of the chip they include. Additionally, some researchers go further and include other types of wear within their sample, such as Patterson et al. (2003) who included any tooth that had worn down to the pulp chamber (by any means of wear) and included this along with chips as 'damaged' teeth. Such studies are therefore not appropriate for comparison with this research.

Post-mortem damaged teeth were not recorded for chipping. Therefore even if a broken tooth had a chip, it was not recorded for analysis. The threshold for a tooth to be recorded as post-mortem broken was deliberately low, with only very minimal damage, such as a small crack or patch of discoloration, to the occlusal surface allowed. Damage to other parts of the tooth did not warrant the exclusion of these teeth as chipping data could still be gathered. This is to prevent an underestimate of the total number of chips, as if severely broken teeth are included in analysis then teeth that did have chips, but that part of the tooth was lost post-mortem, would be marked as not present. Therefore, this method, although limiting sample size, is the only way to get a true representation of actual chipping rates. This is particularly important in this study since the different samples clearly have much variation in their preservation (Chapter 4). As pointed out by Scott & Winn (2011) intraobserver error is likely minimal considering the difference between ante-mortem and post-mortem fractures. However, to make sure no such effects may occur 218 baboon teeth were recorded on two separate occasions (see below).

Fracture rates are analysed and presented per tooth rather than per individual. This is for two reasons, firstly the fossil record rarely provides complete dentitions for examination so comparisons with the extant primate samples would have been difficult, but secondly, because in some samples, particularly baboons and *H. naledi*, virtually all adult individuals have at least one chip, so comparisons based on per tooth provides much more detail. Overall tooth frequencies are presented, but teeth are also divided into tooth type.

It is common in the literature to only study permanent teeth. Splitting the dentitions is justified due to the different properties between permanent and deciduous teeth (Mahoney et al., 2000). Data is therefore also displayed, separately, for deciduous teeth for species with large enough sample sizes. Results are displayed for all specimens; however, for individual figures and tables, a subset is used. For example, for comparing chip frequencies

by tooth type and jaw, the samples of *A. sediba* and early *Homo* are too small to offer helpful insight, with many tooth positions not represented. There is a specific section for *H. naledi* in the results and discussion due to the unusual results this species displays.

5.2.1. Specific materials and methods

Following Daegling et al. (2013), damaged teeth as well as those not subjected to chipping, due to non-eruption or limited occlusion (based on negligible or no crown wear), are not recorded. The same criteria for tooth exclusion were followed when recording chipping in all samples. Table 5.1 displays the number of permanent teeth for each sample. The same criteria are used for deciduous teeth. However, unless stated, the results refer to permanent teeth only. To assess the level of intraobserver error, 218 baboon teeth were recorded on two separate occasions; no significant difference was detected ($\chi 2=0.008$, 1 df, $p=0.927$). Analogous data in seven additional samples of recent humans were derived from the literature (see Table 5.3).

Teeth were observed macroscopically with a 10x hand lens to determine whether a chip occurred ante- or post-mortem. Distinguishing between post- and ante-mortem fractures was based on criteria of Scott & Winn (2011), where only chips evidencing subsequent attrition were included in the latter category. The severity, position and number of chips were also recorded. Severity is based on the three-point scale of Bonfiglioli et al. (2004): 1) slight crack or fracture up to 0.5 mm in width or larger, but with only superficial enamel loss, 2) larger irregular fracture up to 1 mm with the enamel more deeply involved, and 3) chip larger than 1 mm involving both enamel and dentine. The number of chips per tooth was recorded following Belcastro et al. (2007). Position was recorded as buccal, lingual, mesial, and distal. If multiple chips are present then the tooth surface with the most fractures was recorded, whereas if the number is equal between two or more sides then the surface with the largest chip was recorded. Statistical significance was tested between tooth groups using a chi-square test of homogeneity, with significance set at the 0.05 alpha level.

To further explore the chipping frequencies in *H. naledi* teeth are also subdivided according to the severity of occlusal wear. Extensively worn teeth are often excluded from study over concerns that previous chips have worn away, or the enamel has become more susceptible to chipping (Bonfiglioli et al., 2004; Scott & Winn, 2011). However, this strategy

can omit important dietary trends, particularly when comparing species. Occlusal wear is a normal part of the masticatory process, so eliminating from consideration data on teeth worn past a certain point may mask dietary differences. Wear data for molars were scored following the method of Scott (1979) and for all other teeth the method of Smith (1984). This approach was employed to determine whether dental attrition is related to chipping frequencies. Scott's (1979) method divides teeth into quadrants, where each quadrant is given a score from 1 to 10. The former value refers to a tooth that is unworn or has very small wear facets, while the latter describes a complete loss of the enamel. Smith's (1984) method is similar but uses a scale of 1 to 8. In this study, molars are separated from the other teeth based on the total of their four quadrants into categories of high (i.e., 20+), medium (13-19), and low wear (5-12). Anterior teeth, here including premolars, are divided into high (5+), and medium-to-low wear (2-4) categories. Individuals referred to in the original publication (Berger et. al., 2015) and those defined as likely individuals at the time of data collection were included in a separate analysis.

For all samples, if a tooth is listed as grade 1 for either wear method outlined above, it was not included in the analysis due to the likelihood it was not in occlusion (Scott, 1979; Smith, 1984). For the comparative sample, in which the sex is known for the vast majority of specimens, an analysis is also performed to compare sexes. This is again tested using a chi-square test of homogeneity, with significance set at the 0.05 alpha level.

Table 5.1. Sample size for each species.

Species	Total teeth	Wear score 1	Damaged/incomplete teeth
Chimpanzees	2501	248	262
Gorillas	2090	263	309
Baboons	883	30	93
Drills	194	12	19
P. robustus	402	35	132
A. africanus	477	65	147
H. naledi	156	24	6
A. sediba	20	1	6
Early *Homo*	66	13	20

5.3. Results

Chipping frequencies are extremely variable between species (Table 5.2), with *H. naledi* and baboons having up to 10 times the amount of chipping than other samples. The hominin specimens do not cluster together and show as high variation as the extant primates (Figure 5.1).

Table 5.2. Chipping frequencies (%) for the different species.

Species	% chipped teeth
H. naledi	44.44
Hamadryas baboons	40.26
Olive baboons	22.58
A. africanus	21.13
Early *Homo*	20.59
Drills	17.01
P. robustus	12.77
Gorillas	11.13
A. sediba	7.69
Chimpanzees	4.92

The pattern of chipping also varies within dentitions, with certain teeth more susceptible in certain species. For example, *H. naledi* has extremely high rates of chipping on all tooth types, but the molars are particularly affected. Whereas *A. africanus* has many more fractures on premolars, particular maxillary, compared to other tooth groups (Figure 5.1). Similarly, in *P. robustus*, canines show the highest frequency of chipping. It can also be seen in Figure 5.1 that gorillas and chimpanzees have rather uniform chipping patterns with no tooth groups standing out.

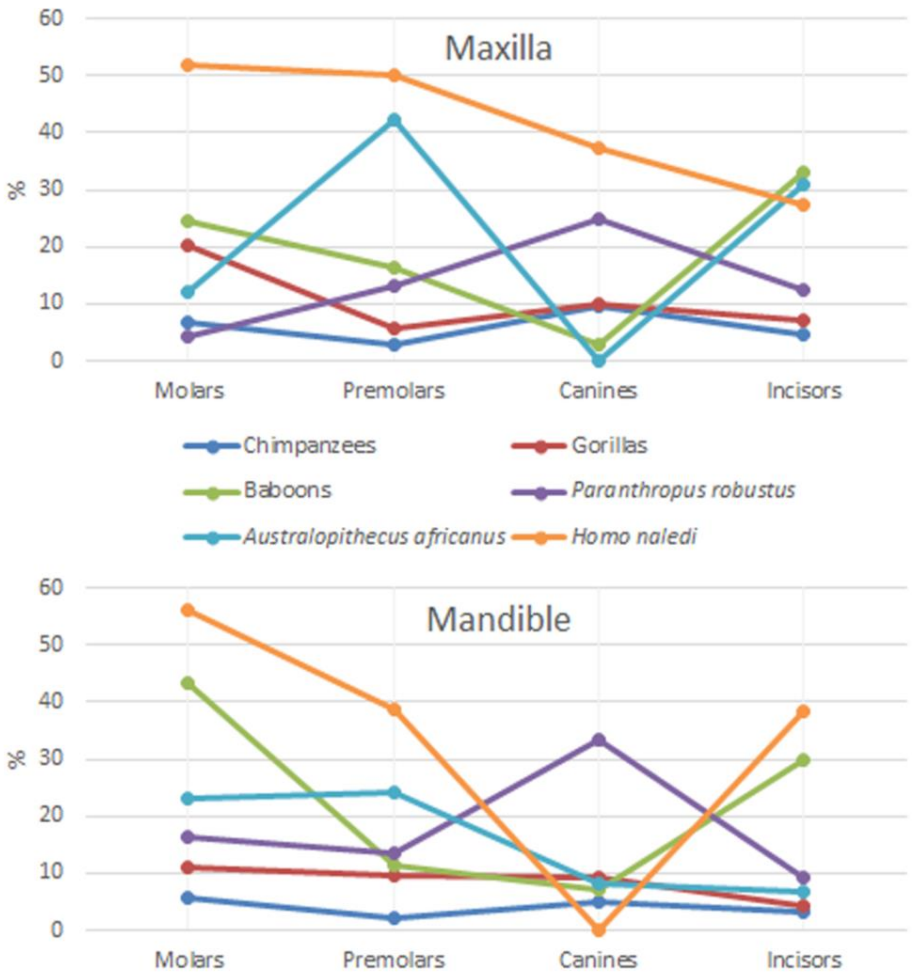

Figure 5.1. Chipping rates (%) for extant primates and fossil hominins, divided by jaw and tooth type. Edited from Towle et al. (2017).

Results are also extremely variable by tooth type for the size and position of chips. Table 5.3 displays the results for South African hominins and comparative extant primates, per jaw and tooth type. This table also shows the number of chips for each severity grade. Particularly noticeable is the low number of large chips in *H. naledi*, and the proportionally greater number of large chips in *P. robustus*. Although, overall, the chipping rate for *P. robustus* is low compared to the other hominins and baboons. Overall chipping rates for the different samples, as well as ratios comparing chip frequency, are provided for maxillary vs. mandibular teeth, posterior vs. anterior teeth, and small vs. large chips in Table 5.4.

Table 5.3. Chipping presence, absence, and severity for upper molars (UM), upper premolars (UP), upper canines (UC), upper incisors (UI), lower molars (LM), lower premolars (LP), lower canines (LC), lower incisors (LI), and all teeth (All). Edited from Towle et al. (2017).

Sample	UM	%	UP	%	UC	%	UI	%	LM	%	LP	%	LC	%	LI	%	All	%
Chimpanzees																		
Total number of teeth[1]	501		292		136		315		497		300		140		320		2501	
Complete teeth with chip(s)	29		6		9		11		25		5		5		8		98	
Complete teeth with no chips	415		248		107		261		432		279		121		278		2141	
Damaged/incomplete teeth	57		38		20		43		40		16		14		34		262	
Teeth with a wear score of 1	24		51		23		26		17		51		24		32		248	
Small chips[2]	19	66	5	83	6	67	9	82	16	64	3	60	2	40	8	100	68	69
Medium chips[2]	10	34	1	17	2	22	2	18	9	36	2	40	3	60	0	0	29	30
Large chips[2]	0	0	0	0	1	11	0	0	0	0	0	0	0	0	0	0	1	1
Chipping frequency %	7		3		10		4		6		2		5		3		5	
Gorillas																		
Total number of teeth[1]	409		247		110		271		411		241		113		288		2090	
Complete teeth with chip(s)	72		9		6		12		37		19		6		8		169	
Complete teeth with no chips	301		201		78		200		332		200		83		217		1612	
Damaged/incomplete teeth	36		37		26		59		42		22		24		63		309	
Teeth with a wear score of 1	17		54		23		43		32		24		25		45		263	
Small chips[2]	68	94	7	78	6	100	8	67	33	89	18	95	6	100	8	100	154	91
Medium chips[2]	4	6	1	11	0	0	4	33	4	11	1	5	0	0	0	0	14	8
Large chips[2]	0	0	1	11	0	0	0	0	0	0	0	0	0	0	0	0	1	1
Chipping frequency %	20		6		10		7		11		10		9		4		11	

Baboons																		
Total number of teeth[1]	174		116		49		107		166		110		49		112		883	
Complete teeth with chip(s)	38		16		1		29		62		10		3		33		192	
Complete teeth with no chips	123		90		41		59		85		80		43		77		598	
Damaged/incomplete teeth	13		10		7		19		19		20		3		2		93	
Teeth with a wear score of 1	5		8		8		0		4		1		4		0		30	
Small chips[2]	31	82	13	81	1	100	27	93	49	79	9	90	3	100	29	88	162	84
Medium chips[2]	7	18	3	19	0	0	2	7	13	21	1	10	0	0	4	12	30	16
Large chips[2]	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Chipping frequency %	24		16		3		33		43		11		7		30		25	
Paranthropus robustus																		
Total number of teeth[1]	78		63		15		38		117		58		10		23		402	
Complete teeth with chip(s)	2		4		2		3		12		5		1		1		30	
Complete teeth with no chips	50		30		7		26		74		34		4		15		240	
Damaged/incomplete teeth	26		29		6		9		31		19		5		7		132	
Teeth with a wear score of 1	4		4		1		5		12		2		2		5		35	
Small chips[2]	2	100	1	25	0	0	1	33	10	83	4	80	0	0	1	100	19	63
Medium chips[2]	0	0	3	75	1	50	2	67	2	17	1	20	1	100	0	0	10	33
Large chips[2]	0	0	0	0	1	50	0	0	0	0	0	0	0	0	0	0	1	3
Chipping frequency %	4		13		25		13		16		14		33		9		13	
Australopithecus africanus																		
Total number of teeth[1]	81		55		20		31		153		65		36		36		477	
Complete teeth with chip(s)	6		11		0		4		24		9		1		1		56	
Complete teeth with no chips	48		24		16		19		90		36		19		22		274	
Damaged/incomplete teeth	27		20		4		8		39		20		16		13		147	
Teeth with a wear score of 1	4		9		8		10		10		8		8		8		65	
Small chips[2]	6	100	10	91	0	0	4	100	22	92	8	89	0	0	1	100	51	91
Medium chips[2]	0	0	1	9	0	0	0	0	1	4	1	11	1	100	0	0	4	7
Large chips[2]	0	0	0	0	0	0	0	0	1	4	0	0	0	0	0	0	1	2

Chipping frequency %	12		42		0		31		23		24		8		7		21	
Homo naledi																		
Total number of teeth[1]	31		21		13		14		28		19		11		19		156	
Complete teeth with chip(s)	15		9		3		3		14		7		0		5		56	
Complete teeth with no chips	16		11		9		11		14		11		10		12		94	
Damaged/incomplete teeth	0		1		1		0		0		1		1		2		6	
Teeth with a wear score of 1	2		2		4		3		3		0		6		4		24	
Small chips[2]	13	87	9	100	3	100	3	100	11	79	6	86	0	0	5	100	50	89
Medium chips[2]	2	13	0	0	0	0	0	0	3	21	0	0	0	0	0	0	5	9
Large chips[2]	0	0	0	0	0	0	0	0	0	0	1	14	0	0	0	0	1	2
Chipping frequency %	52		50		38		27		56		39		0		38		44	

[1]Damaged/incomplete teeth and teeth with a wear score of 1 dropped from the total number of teeth before chipping frequency is calculated.

[2]Small, medium, and large chips are scored according to Bonfiglioli et al. (2004) with three-point severity scale

Table 5.4. Per-tooth chipping frequencies and ratios of dentitions affected. Edited from Towle et al. (2017).

Sample/Location	Chipping rate %	Multiple chipped teeth %	Small:Large[1] chip ratio	Maxilla:Mandible ratio	Anterior:Posterior ratio	Time period	Reference
Fossil hominins							
H. naledi	44.44	50.00	8.33:1**	1.05:1	0.61:1**	Not yet known	This study
A. africanus	21.13	16.07	10.20:1**	1.04:1	0.54:1	Plio-Pleistocene	This study
P. robustus	12.77	6.67	1.73:1	0.66:1	1.25:1	Plio-Pleistocene	This study
Extant primates							
Baboons	25.26	18.75	5.40:1**	0.79:1	0.93:1	19th/20th cent CE	This study
Gorillas	11.13	4.14	10.27:1**	1.48:1**	0.51:1**	19th/20th cent CE	This study
Chimpanzees	4.92	2.04	2.27:1**	1.73:1	0.95:1	19th/20th cent CE	This study
Recent humans							
St. Lawrence Island Inuit	66.40	*	*	1.04:1	0.77:1**	2nd–17th cent CE	Scott & Winn (2011)
Quadrella (Italy)	48.40	*	0.70:1	1.14:1	1.50:1**	2nd–3rd cent BCE	Belcastro et al. (2007)
Vicenne-Campochiaro (Italy)	38.90	*	1.12:1	1.17:1	1.68:1**	4th–10th cent CE	Belcastro et al. (2007)
Taforalt (Morocco)	29.20	*	*	*	0.64:1**	11,000–12,000 BP	Bonfiglioli et al. (2004)
Norway	21.90	*	*	1.24:1**	3.40:1**	11th- 14th cent CE	Scott & Winn (2011)
Spain	5.90	*	*	1.73:1	3.10:1**	11th– 18th cent CE	Scott & Winn (2011)
Cape Cod Woodland (USA)	43.40	*	*	0.79:1	*	5th–10th cent CE	McManamon et al. (1986)

[1]Small chips are those recorded as severity grade 1 and large as grades 2-3, according to Bonfiglioli et al. (2004)

*Data not reported in publication

**Chi-square significant at 0.05 level

5.3.1. Deciduous teeth

The small sample size for deciduous teeth in the hominins does not permit splitting results up by tooth type or jaw. Therefore, the overall frequencies for each species are displayed in Table 5.5. The rate of chipping is lower than in permanent teeth, with much less variability between species. Figure 5.2 displays one such chip on a deciduous second molar of an *A. africanus* specimen (STW 151).

Table 5.5. Chipping frequencies (%) for deciduous teeth.

Sample	Number of chipped teeth	Total number of teeth	% Chipped
H. naledi	3	22	13.64
P. robustus	1	29	3.45
A. africanus	4	30	13.33
Early *Homo*	2	12	16.67
Chimpanzees	16	741	2.16
Gorillas	19	482	3.94
Olive baboons	9	65	13.85

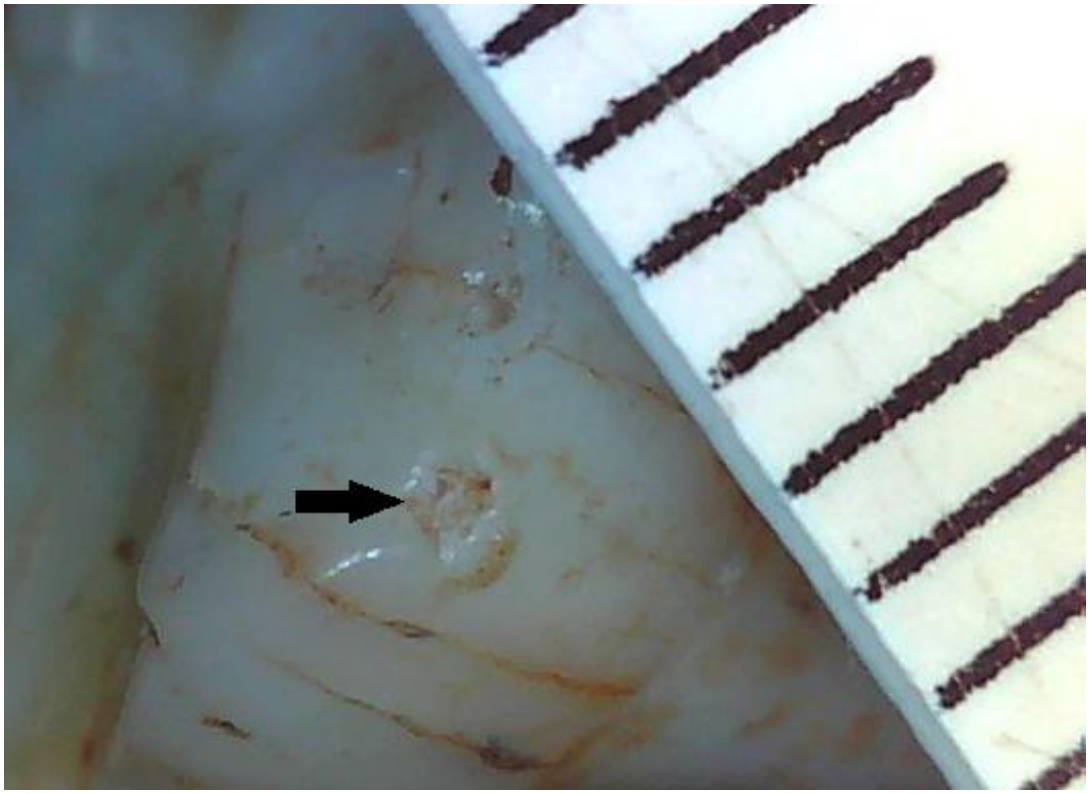

Figure 5.2. STW 151: Mandibular left deciduous second molar, displaying a distal chip (arrow).

5.3.2. Chip position

There are substantial differences between the extant primate and fossil hominin samples in terms of chip position (Figure 5.3). In the former, except for gorillas, the vast majority of chips are on the buccal surface of upper molars and the lingual surface of lower molars. Whereas in hominins there is much more interproximal chipping, especially in *H. naledi* (Figure 5.3). However, *P. robustus* and gorillas do seem to have very similar patterns, with each surface affected by roughly the same amount of chipping.

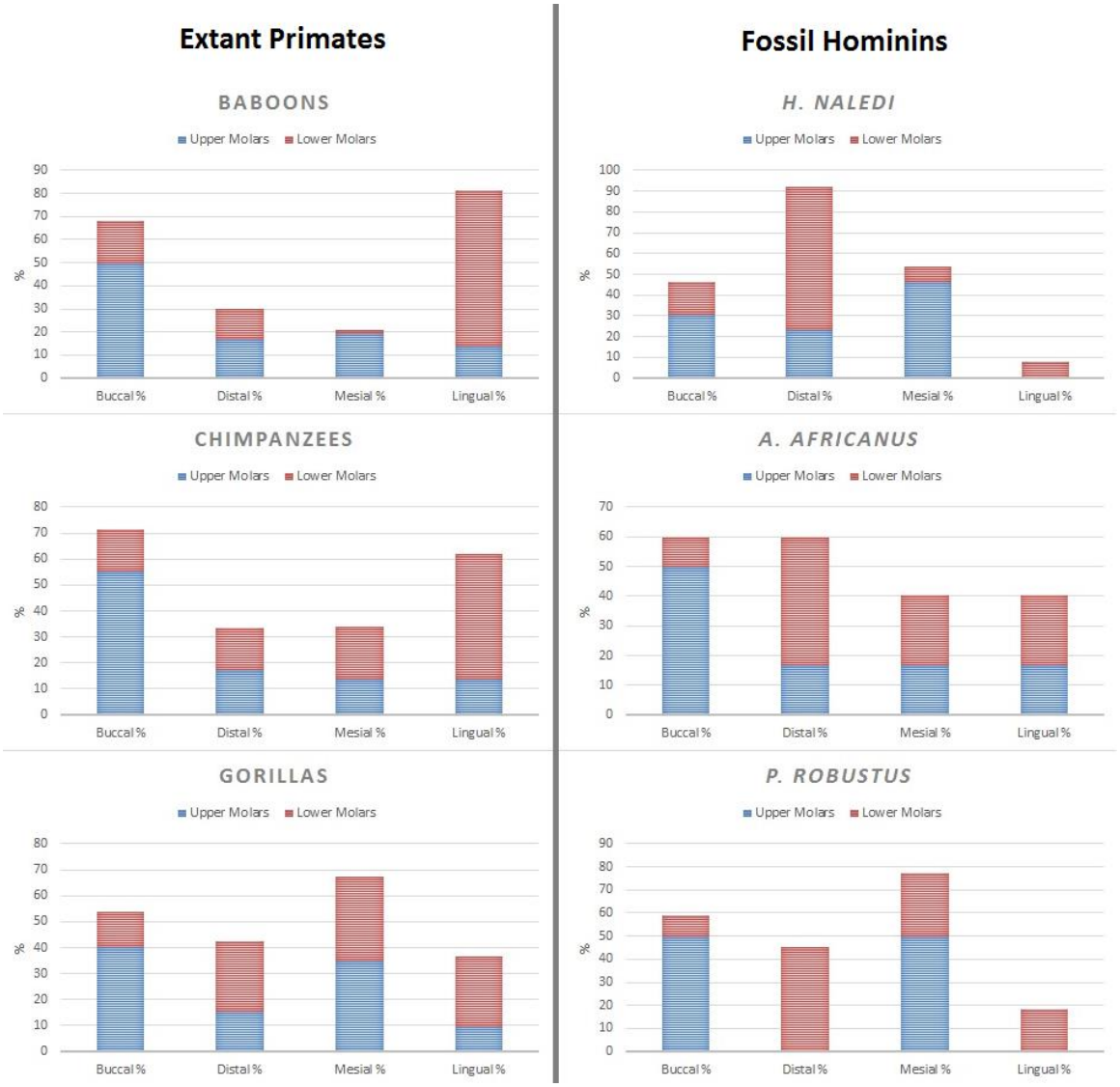

Figure 5.3. Chipping position for the molars of extant primates and fossil hominins. As a percentage of all chipped teeth from each sample.

5.3.3. Sex differences

There is a statistically significant difference between the sexes in chimpanzees (X^2= 13.769, 1 df, p= 0.0002), with males having double the amount of chipped teeth than females (6.87% vs. 3.34%; Figure 5.6). Males also have more chipping in both gorillas and baboons, although these differences are not statistically significant (gorillas: X^2= 2.263, 1 df, p= 0.1325; baboons: X^2= 3.836, 1 df, p= 0.0501). This relationship does not appear to hold for deciduous teeth with no significant differences, and females actually have more chips in chimpanzees (gorillas: X^2= 0.973, 1 df, p= 0.3240; chimpanzees: X^2= 1.959, 1 df, p= 0.1617). The baboon sample is not large enough for deciduous teeth to include.

Table 5.6. Overall chipping frequencies (%) divided by sex and dentition.

Variable	Chimpanzees	Gorillas	Baboons
Permanent teeth			
Males	**6.87***	12.69	25.82
Females	**3.34***	9.99	19.03
Deciduous teeth			
Males	1.25	5.10	-
Females	2.70	3.28	-

*Chi-square significant at 0.05 level

5.3.4. Homo naledi

Due to the extremely high levels of chipping in *H. naledi* compared to the other hominin samples, it is worth investigating further potential etiologies. With 44.4% of permanent teeth affected, *H. naledi* exhibits a far higher chipping rate than the other South African samples. Specifically, 53.7% of molars, 44.4% of premolars, 25% of canines, and 33.3% of incisors have at least one chip; of these, 50% display two or more chips. Only 13.6% of primary teeth are affected. Most chips are small, i.e., severity 1 (n=51), with only six recorded as 2 or 3. Over 73% of those on the molars are located interproximally. Particularly common are several small chips above the wear facets of posterior teeth (Figure 5.4).

Chipping frequencies are presented by wear score and side in Table 5.8. Among other variation evident in these categories, it can be seen that right teeth are affected slightly more often than left, with rates of 50% and 37.7%, respectively, having at least one chip. The average affected right molar has 2.37 chips and the left 2.06, with medians of 2 and 1 in these non-normally distributed data (Shapiro-Wilk, p=0.000). However, differences by side are not statistically significant (X^2= 1.945, 1 df, p= 0.16).

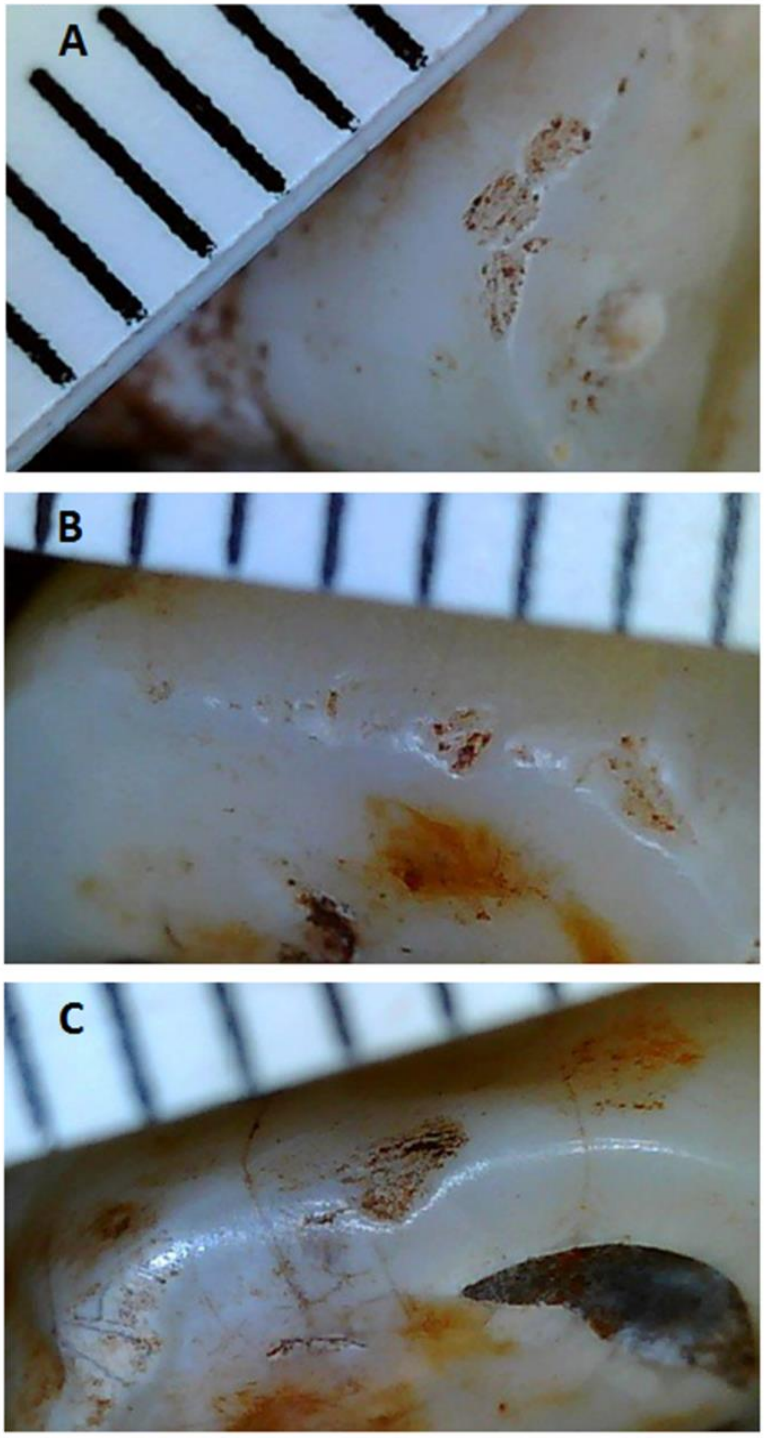

Figure 5.4. Chipping examples: a) UW 101-525 upper right first molar, three chips on mesial surface; b) U.W. 101-1401 upper right second premolar, multiple small chips on distal surface; c) UW 101-1402 upper right first premolar, mesial chip. Edited from Towle et al. (2017).

Table 5.7. Chipping frequencies for different tooth types in *H. naledi*. Edited from Towle et al. (2017).

Sample	Total Teeth	With Chipping	%
All teeth	126	56	44.44
Left teeth	61	23	37.70
Right teeth	66	33	50.00
Primary teeth	22	3	13.64
Molar wear stage[1]			
All molars	54	29	53.70
Light wear (5 to 12)	19	4	21.05
Medium wear (13 to 19)	21	12	57.14
High wear (20+)	14	13	92.86
PM's, C's and I's wear stage[1]			
All anterior and premolar teeth	72	27	37.50
Light wear (2 to 4)	46	15	32.61
Heavy wear (5+)	26	12	46.15

[1]Molar wear was calculated following Scott (1979) with all other teeth using Smith (1984)

The posterior teeth of *H. naledi* have more chips than the anterior teeth, and the average difference in overall frequency is statistically significant (χ^2=3.938, 1 df, p=0.047). Posterior teeth are also more likely to exhibit multiple chips than anterior teeth; to test this, a chi-square test was again used, though with Yates' continuity correction because expected cell size for anterior teeth with multiple chips is $\leq$5. The difference is significant (χ^2_c = 7.240, 1 df, p= 0.007). Of the 12 individuals represented by dental remains, nine have at least one chipped tooth; two of the remaining three are represented by only one tooth, and the third has minimally worn teeth (i.e., scores of <2).

Homo naledi has a higher rate of chipping than other South African hominins and extant non-human primates (Table 5.4). The rate is more comparable to several of the recent human samples. However, many of the latter differ in chipping ratios compared with *H. naledi*; particularly noticeable is the preponderance of small chips vs. large (i.e., ratio of 8.33:1) and

fewer affected anterior vs. posterior teeth (0.61:1). Although the overall rate of chipping in *H. naledi* is more similar to these recent human groups, the nature of the chipping with regard to size and location within the dental arcade is more like that observed in *A. africanus* and in baboons.

5.4. Discussion

Perhaps the most striking aspect of the results is the variability observed in the fossil hominin samples. The extant primate results fit well with the dietary information, with chimpanzees having the lowest chipping rate, baboons the highest and gorillas in between. Chimpanzees do not regularly eat hard seeds and nuts, whereas baboons eat a wide range of foods, many of which are hard or tough (Boesch et al., 2002; Conklin-Brittain et al., 2001; Doran et al., 2002; Isabirye-Basuta, 1989; Lodge et al., 2013; Macho, 2014; Phillips & Lancelotti, 2014; Whiten et al., 2011). Thus, it is surprising to see such a range in frequencies for the hominin sample; as such, a range fills almost the entire spectrum of the extant primate sample. This finding is at least suggestive of dietary or behavioral differences among these hominin species.

5.4.1. Paranthropus robustus

Chipping frequencies in South African hominins have been used to support a variety of different hypothesis regarding diet. For example, as noted by Wallace (1975), frequencies have been used to both support and refute Robinsons dietary hypothesis, with Robinson (1954; 1956) claiming high rates of chipping he found in *P. robustus* is caused by grit-laden roots, whereas Tobias (1967) rejected this as he found similar amounts of chipping in *P. robustus* and *A. africanus*. Of the chipping studies previously carried out on South African hominins, the most similar study in terms of methods to those used in this thesis is Wallace (1975), yet on the face of it the differences in results look rather substantial. Additionally, as has been introduced above, other studies (Robinson, 1954, 1956; Tobias, 1967) that have used different methods have shown contradictory results.

If teeth recorded as wear score one and teeth that have been marked as unobservable are included then the percent of chipped teeth for *P. robustus*, *A. africanus* and *H. naledi* drop to 7.8%, 11.4% and 36.1% respectively. These are very similar to those recorded by Wallace (1975), especially for *P. robustus*, meaning the different rates observed are likely simply a

consequence of Wallace including more teeth that in this thesis were marked as unobservable. It is not stated by Wallace if such broken or obscured teeth were included but judging by the sample size (n= 589), the threshold for including a tooth must have been lower. Additionally, the 34 extra chips that are found in this study can be easily explained by the addition of many new fossils. However, unfortunately, a list of the chipped teeth was not given, so a direct comparison is not possible. When these earlier chipping studies were conducted, there was little in the way of comparative material, both hominin and other mammals, and therefore it was presumed the chipping rate found in *P. robustus* was high. Recent research, however, including the results of this study, have shown that enamel chipping is common in many species, and indeed, even species such as gorillas and chimpanzees that are not known for hard object feeding still show regular fractures.

When the *P. robustus* and *A. africanus* material is split up by site (*P. robustus*: Swartkrans and Kromdraai; *A. africanus*: Sterkfontein and Makapansgat), there is no statistically significance difference in chipping frequency between sites for each species (*P. robustus*: X^2= 0.180, 1 df, p= 0.6718; *A. africanus*: X^2= 0.153, 1 df, p= 0.6959). Given the variability between hominins as a whole, this is a fascinating result. If the diet was substantially different, or if different species are represented as has been suggested, then it would be expected that there would be a difference given how variable chipping rates can clearly be. It also further suggests these two species may have distinct diets. When the Drimolen *P. robustus* material is studied for chipping it will be interesting to see if this material too has a chipping frequency of around 12%.

The low rate of chipping in *P. robustus*, with comparable rates to gorillas, suggests they did not specialise in hard object feeding. The fact that *P. robustus* has similar chipping frequencies to gorillas is in itself interesting. Particularly when it is considered that the position of chips is also the most similar out of all the samples. This may, therefore, suggest that similar proportions of certain food items are being consumed. This does not necessarily mean the same food, but perhaps does suggest *P. robustus* was eating substantial amounts of tough fibrous vegetable matter and fruits, rather than hard food items such as seeds and nuts (Daegling et al., 2013). This is explored further in the other chapters, but particularly in Chapter 12 when all variables are considered together.

It has been suggested that thick enamel may have evolved in certain mammals to improve resistance to fractures caused by the ingestion of hard foods or environmental contaminants (Kay, 1981; Lucas et al., 2008). It has also been proposed that thick enamel may evolve to prolong a tooth's life by allowing more occlusal attrition to be accommodated (Macho & Spears, 1999). Debate has surrounded which of these may explain the extremely thickly enamelled teeth of *P. robustus*. Clearly, the results of this chipping analysis, when compared with the other hominins and extant primates, is supportive of the latter suggestion, due to the relatively low rates of dental fractures.

5.4.2. Australopithecus africanus

Although the chipping rate for *A. africanus* is substantially lower than *H. naledi*, it is higher than that for extant great apes and *P. robustus*. Interestingly, the premolars of *A. africanus* are the most affected teeth, supporting recent biomechanical analyses (Strait et al., 2009; 2012), with this pattern not observed in the other hominins studied. Another, although likely related, possibility is due to the placement of certain foods into this part of the mouth. For example, Morse et al. (2013) show that *Cercocebus atys* has a unique wear pattern in which there is an unusual amount of wear in the p4-m1 region, thought to be caused by the regular consumption of a particularly large and hard nut (*Sacoglottis gabonensis*). The authors suggest this is likely due to the size and shape of the nut making initial mastication of the nut in other parts of the mouth impractical or potentially damaging. This is supported by observations of these primates placing these nuts into this area of the mouth (Daegling et al., 2011). Therefore, a similar effect could explain the high rates of chipping in the premolar regions of *A. africanus*.

5.4.3. Early homo

As has been discussed, *H. naledi* specimens have extremely high levels of chipping, but what about other specimens assigned to the genus *Homo*? Unfortunately, the sample is very small for the early *Homo* sample and, as mentioned, there is much debate to which specimens should be included in this category. One such specimen which has had its fair share of controversy regarding phylogeny is STW 19. This specimen shows chipping (Figure 5.5), and given the small sample size, the inclusion of these specimens has a big impact on the frequency for early *Homo* as a whole. Therefore, any assessment of chipping in these

specimens is speculative. However, worth noting here is the surprisingly similar chipping rates of early *Homo* and *A. africanus*, perhaps hinting at dietary similarities.

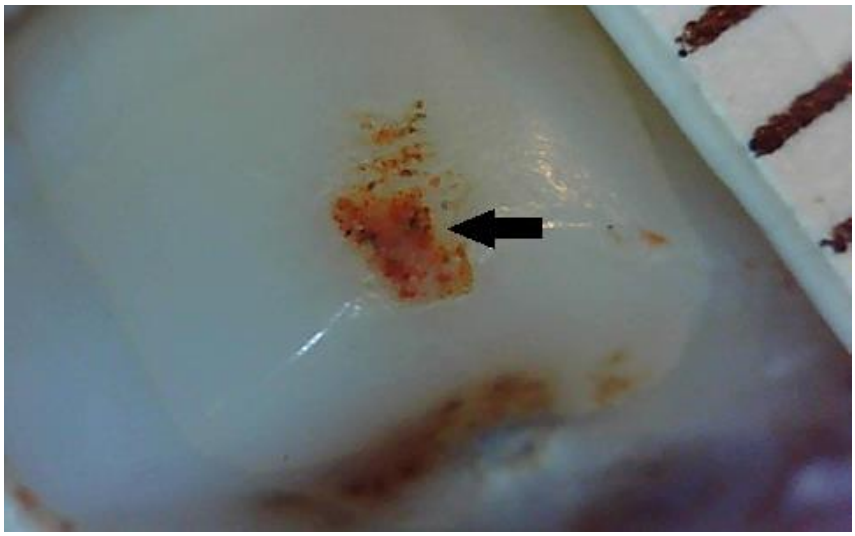

Figure 5.5. STW 19: Chipping on the lingual distal corner of the upper right second molar (arrow).

5.4.4. Homo naledi

The *H. naledi* sample appears quite homogeneous with regard to the location, number, and severity of chipping across individuals, not unlike that of the species' developmental attributes, such as uniformly simple crown morphology on relatively small, thick-enameled teeth (Berger et al., 2015; Cofran et al., 2016; Skinner et al., 2016). The amount of ante-mortem dental chipping across the sample, including multiple instances in individuals with greater attrition, indicates that the teeth were exposed to acute trauma on a regular basis. Interproximal surfaces are more affected than buccal surfaces and posterior teeth more than anterior teeth, which is suggestive of a dietary rather than a non-masticatory cause (Belcastro et al., 2007). This patterning can result from contaminants in the diet, like grit when consuming foods such as roots and tubers (Belcastro et al., 2007; Robinson, 1963; Stojanowski et al., 2015).

Clearly, there will be a point when an object is too small to create a visible chip and instead results in enamel microwear. The point at which this occurs likely varies, depending on the properties and shape of both the enamel and the object (Daegling et al., 2013). The contaminants consumed by *H. naledi* would have had to at least occasionally been above this size threshold. Certain environments make contaminants more likely to be consumed, such

as dry and arid conditions or areas affected by such phenomena as ash clouds following a volcanic eruption (Belcastro et al., 2007; Riede & Wheeler, 2009; Spradley et al., 2016). It will, therefore, be useful to incorporate data about the environment in which *H. naledi* lived.

As mentioned in the introduction, the effects of enamel thickness, occlusal wear, and enamel microstructure on chipping frequencies are not well understood at present. It has been suggested that thickness is not important in terms of chip number (Constantino et al., 2012). Nevertheless, thicker enamel can accommodate larger chips and hence may skew inferences drawn from assessments of chip size. Severe wear will have a similar effect on frequencies, with chip size being limited. These factors do not seem to be responsible for small chips in this sample, because chip size is consistently small regardless of wear and despite the presence of thick enamel (Skinner et al., 2016). It is also unlikely that *H. naledi* has significantly different enamel microstructural properties than other hominin species, given their presumed phylogenetic relationship. Additional research on masticatory and enamel properties should offer further insight into the susceptibility of these individuals to dental fractures. Chipping rates clearly increase with wear, due to enamel properties or, more likely, time in occlusion. However, it is clear that the high rate in this sample is not simply a consequence of heavy wear, for even lightly worn teeth have far higher rates of chipping than those observed in the other hominin samples. It seems probable that the objects responsible for this chipping were consumed regularly, especially given that small chips should wear away more quickly than large chips.

Dental chipping in *H. naledi* differs notably from the other fossil hominin samples examined for this study. The rate is roughly twice that of *A. africanus* (44.44% vs. 21.13%) and more than three times that of *P. robustus*, among whom only 12.77% of teeth are affected. The patterning of chipping differs as well, particularly relative to *P. robustus* (see ratios in Table 5.4). The extant primate samples may offer more useful comparisons for *H. naledi*. For example, in a microwear study by Nystrom et al. (2004), baboons in dry environments were reported to consume large amounts of grit. In the combined sample of hamadryas and olive baboons, we found similarities to *H. naledi*, with frequent small chips and a higher rate of chipping among posterior teeth relative to anterior teeth.

Recent human samples with comparably high rates, such as the Inuit and medieval Italian Quadrella (Table 5.4), have different patterns of chipping than observed in *H. naledi*;

either their anterior teeth are more affected from extra-masticatory activity, or all teeth evidence severe chipping as a result of dietary and cultural behaviors (Belcastro et al., 2007; Scott & Winn, 2011; Turner & Cadien, 1969). However, there are some human parallels. A Late Woodland sample from Cape Cod in the U.S.A. has a pattern like *H. naledi* in terms of frequency and position (McManamon et al., 1986). The overall frequency is 43%, and molars are reported as the tooth type most prone to chipping, with interproximal surfaces most affected. Unfortunately, frequencies for tooth types and positions in that study are not reported. McManamon et al. (1986) suggest that the cause of this patterning was the incorporation of sand, gravel, and shell fragment contaminants into the food. Another sample with somewhat similar frequencies to *H. naledi* is from the site of Taforalt. Bonfiglioli et al. (2004) concluded that the frequent interproximal chipping in these epipalaeolithic Moroccans was due to chewing hard, abrasive snail shells and fruit stones. Many seeds and nuts were also consumed (Humphrey et al., 2014). Dietary contaminants may have been a factor as well, given the environmental conditions and presence of grindstones (Humphrey et al., 2014). However, direct comparison is confounded, because these peoples practiced maxillary incisor avulsion (De Groote & Humphrey, 2016). Lastly, the medieval Vicenne-Campochiaro sample exhibits a chipping pattern similar to *H. naledi*; it has high rates of interproximal chipping on posterior teeth, especially in females. Belcastro and coworkers (2007) suggest the cause was grit incorporated into the diet.

Numerous ways of incorporating grit into the diet of *H. sapiens* have been documented; these include grit from food processing and ash from roasting pits (Hartnady & Rose, 1991). It has even been noted that some historic populations in South Texas added dirt to sweeten food (Newcomb, 2010). This behaviour demonstrates that incorporating grit into the diet may not necessarily be accidental, although clearly, this is far more likely. Instead, the most likely explanation is probably that *H. naledi* was consuming large amounts of foods that already had grit or sand sized particles attached, such as tubers.

If a specific food item is responsible for chipping in *H. naledi*, then these individuals must have specialised in the consumption of a particular type of very small hard object. Additional evidence, such as the steeply-angled wear and slight cupped, i.e., scooped-out, wear of dentine on several posterior teeth in *H. naledi* – both of which can result from consumption of grit, generally in conjunction with softer foods (Brace, 1962; Hinton, 1981;

Smith, 1984; Figure 5.6). It also cannot be ruled out that these individuals were processing foods, at least to the extent seen in chimpanzees who dismantle seeds and nuts before ingestion (Boesch & Boesch, 1982; Daegling et al., 2013; Wrangham & Conklin-Brittain, 2003). So, potentially, only small hard objects were masticated in the mouth, with larger hard items processed to some extent prior to mastication. That said, other evidence for this hypothesis is lacking. Moreover, although conjectural, perhaps the higher rate of chipping in the right teeth of *H. naledi* resulted from preferential placement of the food or objects (and contaminants) in this side of the mouth. Greater wear on right relative to left teeth has been reported in several fossil *Homo* specimens and has been attributed to right-hand dominance in the manipulation of objects during oral processing (Estalrrich & Rosas, 2013; Fiore et al., 2015; Frayer et al., 2016). Yet, as noted, side differences in chipping in *H. naledi* were found to be non-significant; recovery of additional specimens may provide clarification, while micro- and macrowear analyses by side should provide interesting comparisons.

Figure 5.6. Steeply-angled wear on an upper left second molar (UW 101-528).

5.4.5. Enamel properties

The extant primate sample has an extremely high rate of lingual chipping, especially on mandibular teeth, whereas in the hominin sample lingual surfaces are little affected. This is likely, at least partly, due to dental morphology, enamel properties and the masticatory cycle (Chai & Lawn, 2007; Constantino et al., 2010; Daegling et al., 2013; Lawn et al., 2009; Ziscovici et al., 2014). However, there may be dietary input, but such research is yet to be

carried out. Perhaps such factors may also be important in explaining why *H. naledi*, and hominins in general, have such a high rate of chipping on interproximal surfaces. This will be explored in the wear chapter by comparing chipping rate with angle and severity of wear for the different species.

Cuy et al. (2002) suggest studies that do not consider enamel properties might be overly simplifying the process. They found that enamel mechanical properties varied between the lingual and buccal side of upper second molars studied, with the lingual side of the occlusal surface harder than the buccal. The opposite relationship was true for the interior enamel. These aspects taken as a whole could lead to the issue that two species with near identical diets could have different chipping patterns based simply on enamel properties. These results the authors put down to being possibly related to differences in function. This is an intriguing possibility; unfortunately, little has been done on this topic since this publication. It would be interesting to know if the same (or opposite) pattern holds for mandibular molars and if other hominin species have the same pattern. Likewise if it is related to function, then a good hypothesis would be that hominin species with certain molar pattern should share similar enamel properties across the crown.

Other enamel property differences still need to be explored further, for example it has been suggested that heavily worn teeth may be weaker and more susceptible to chipping (Scott & Winn, 2011), although this does seem logical on the face of it, little work has been done to clarify if this is true in practice. Similarly, it is not known if enamel stays consistently susceptible to fracture through life or if the enamel properties change slightly with age.

Tooth property differences may also explain why deciduous teeth are less affected than permanent teeth, although dietary differences and time in occlusion are also likely significant factors. It is interesting that there seem to be no sex differences in deciduous teeth yet in adult chimpanzee teeth there is. This is perhaps not surprising given they are likely still being cared for during most of the period of having deciduous teeth. However, it does raise the issue of potential biases being introduced if you have an uneven mix of juveniles and sexes when comparing species and populations.

Enamel cracks will most often run along interprismatic pathways (Bajaj et al., 2008; He et al., 2006; He & Swain 2008). Therefore, fractures will cross many prism boundaries on

their way to the lateral surface of the crown. Due to this, it has been suggested that an increase in crisscrossing of enamel prisms will, therefore, mean a larger chip in such a species would need a higher bite force to have formed (Constantino et al., 2012). If such differences exist in South African hominins, this could, therefore, be an important factor in the size and amount of chips. Although some work has been conducted on species differences, at the moment such sample differentiating research has not been specifically conducted (e.g., Lacruz, 2007). It may seem unlikely that the species included in this thesis would have significantly different enamel micro-structure given their close phylogenetic relationship. However, the fact that *P. robustus* and to a lesser extent *H. naledi* show signs of, potentially, evolving traits to facilitate hard object feeding, such as large posterior teeth (in *P. robustus*), thick enamel relative to tooth size and large masticatory muscles, suggests that caution should be given to presuming the enamel micro-structure may be the same. This is because if these traits have evolved as a defence against fractures then potentially other possible areas of evolving defences against such cracks would be through altering the microstructure of the enamel. However, this has not been researched and, as mentioned, there is still a lot of debate to whether the suite of masticatory characteristics displayed by *P. robustus* are related to hard object feeding (see Chapter 12).

In noting differences between maxillary and mandible chipping Scott & Winn (2011) highlight that in their study all samples showed more fractures on maxillary teeth than mandibular, although only one sample showed a statistically significant difference. They suggest such difference is likely due to fracture mechanics of the different teeth involved (Schatz et al., 2001). There is high variation in the species studied here, with, for example, baboons and *P. robustus* having much more chipping on mandibular teeth. That said, only gorillas show a statistically significant difference (Table 5.4). Therefore, it seems improbable there is a strong trend for a certain jaw to be affected in primates as a whole, although further research is needed to clarify this.

5.5. Future research

It is noteworthy that chimpanzees and baboons have a similar rate of chips on anterior and posterior teeth. However, on closer inspection of the data, this is due to different factors. Chimpanzees have very low chipping in all teeth compared with the other species studied,

with the incisors being no exception to this. However, the upper canines have a surprising number of chips, with almost 10% affected; double the overall percentage. This is likely due to their large size and perhaps even social behaviour. The opposite is true for baboons, in which canines are barely affected whereas incisors, both upper and lower, have chipping rates over 30%. This likely reflects their omnivorous diet and the processing of hard foods but could be due to contaminants affecting teeth more evenly, as is hypothesised for *H. naledi*. Clearly, there are large differences between the teeth affected in different species, an area of research that has received virtually no attention. For example, the little research that has been carried out on extant primates has predominantly only used posterior teeth in the sample. Therefore, such patterns can be missed or misconstrued. Further research on other extant primates would, therefore, offer more insight into what the chipping patterns observed in these hominins may mean in terms of aetiology.

Comparisons with other hominins will also be interesting. Tobias (1967) notes chipping in OH 5, the type specimen of *P. boisei*, although for some of these fractures he is not sure if they are anti- or post-mortem in nature. The left first molar and right second molar have potential ante-mortem chips, although the associated post-mortem fractures make such a classification less reliable. Chipping is often said to be high in *P. boisei*, yet apart from the example above, no research on chipping has been conducted. Even species with very little hard food in their diet, such as chimpanzees, have chips; so, until further studies examine this material, such conclusions cannot be drawn. The isotopic signature for *P. boisei* may suggest a different diet than the South African hominins, but it still seems more likely there will be relatively few chips compared to species such as *H. naledi* or baboons. Such research, as well as on other hominins, will allow further inferences into diet in the specimens studied but also the South African species as further comparative data will be available.

5.6. Conclusions

Homo naledi exhibits high rates of ante-mortem enamel chipping, particularly on the posterior teeth and interproximal areas. These chips are predominately small, and all individuals are affected. These characteristics are suggestive of a dietary aetiology rather than a non-masticatory cause. Once microwear analysis of the teeth is completed, further support may be provided for the possibility that grit underlies the patterns of macroscopic chipping

reported here. In addition, alternative forms of analyses of *H. naledi* specimens (e.g., photolith analyses, etc.), along with chipping research on additional primates, particularly hominins, can help further elucidate whether *H. naledi* regularly ate foods that contained contaminants. Environmental data will be of interest to integrate. However, at present, results from this chipping analysis highlight the fact that *H. naledi* differed noticeably from species comprising the comparative samples studied here – in terms of diet, behaviour, and/or the environment in which they lived.

Given the significant difference in chipping between male and female chimps, as well as sex differences commonly found in recent human samples, it would not be surprising to find similar sex differences in fossil hominins. This cannot be explored in this sample due to the small number of specimens that have been confidently assigned a sex, but it is worth noting for future studies on samples in which sex determination is possible.

Paranthropus robustus has a similar chipping frequency and pattern to gorillas making it unlikely that this species specialised in hard object feeding. *A. africanus* has substantially more chipping than *P. robustus*, with the premolars particularly affected, perhaps supporting recent biomechanical analysis and/or the suggestion that certain hard foods were commonly consumed by placing them in this part of the mouth. There is little difference between the sites from which *P. robustus* and *A. africanus* came, potentially suggesting a very similar diet over time for these species, and further highlighting potential dietary differences between these two species.

Chipping has commonly only been used to compare two species by using overall chipping rates, and often only using certain tooth types, and therefore misses out on other potential patterns that this chapter has hopefully highlighted. The sample size for these South African hominins is high, i.e., for hominins. However, there are also later samples in which these methods can be applied; in doing so a much better understanding of the aetiology of these different patterns will be possible. Further work on enamel properties, both in hominins and other mammalian groups, will also be interesting to incorporate and explore.

Chapter 6: Enamel hypoplasia

6.1. Introduction

Enamel hypoplasia is created during the secretory stage of formation, as opposed to other enamel defects that form during the maturation stage, such as hypocalcification and dental fluorosis; it is caused by the cessation of ameloblast function (Guatelli-Steinberg, 2015; Ten Cate, 1994; Xing et al., 2015). Enamel hypoplasia comes in a variety of forms, most of which are found in fossil hominins (Goodman et al., 1987; Guatelli-Steinberg et al., 2004; Hillson, 2014; Lukacs et al., 2001). A range of factors can create similar defects, making a diagnosis of particular etiologies difficult. Nonetheless, enamel defects can often give insight into diet, genetic disorders, environment and health of individuals and populations (Cunha, 2004; Guatelli-Steinberg et al., 2014; Hillson et al., 1998; Schuurs, 2012).

Enamel hypoplasia is often split into three broad categories, linear-form (LEH), pit-form (PEH) and plane-form (Guatelli-Steinberg, 2015; Pindborg, 1970; Seow, 1990). These defects can look remarkably different from each other but ultimately are all associated with a reduction of enamel caused by a disruption in ameloblast production while enamel matrix secretion is taking place (Eversole, 1984; Hillson, 2014; Hillson & Bond, 1997). It is not always simple to split defects into these three categories (e.g., Ogden, 2007). However, such division is usually justified as certain defects can have specific aetiologies. Genetic conditions, injuries to the tooth during formation, and certain diseases can sometimes cause characteristic hypoplasia defects (Cook, 1980; Crawford et al., 2007; Goodman & Rose, 1991; Ogden et al., 2008; Skinner & Newell, 2003; Weerheijm, 2003).

Enamel hypoplasia was studied in depth during the early to mid-20th century. Research that used rats and mice in experiments was particularly common (e.g., Kreshover, 1960; Schour & Massler, 1945). Even though the use of rodents as proxies for human dental development has since received criticism, these studies nonetheless proved that nutritional deficiencies could lead to enamel defects (Goodman & Rose, 1991). Therefore, even during this period, it was known that hypoplastic defects, particularly LEH, could be caused by a variety of different situations and thus should be best explained as non-specific indicators of stress. Around the same time, it was hypothesised that the majority of enamel hypoplasia in

all human populations occurred in the first year of life (Sarnat & Shour, 1941); however, this idea has since been discredited. Indeed, it is now known that hypoplasia timing varies significantly among groups, with many showing higher frequencies in later years (Goodman et al., 1987; Yamamoto, 1988).

Enamel hypoplasia has been studied in a variety of human populations, with rates varying dramatically in both deciduous and permanent teeth (Goodman & Rose, 1990; Guatelli-Steinberg, 2015; Hillson, 2014; Hillson & Bond, 1997; Moggi-Cecchi et al., 1994; Odgen et al., 2007; Pisanty et al., 1977; Purvis et al., 1973; Seow, 1990; Skinner & Newell, 2003). Enamel defects have also been recorded in numerous fossil hominin specimens, with South African hominins particularly well studied (e.g., Guatelli-Steinberg, 2003, 2004; Robinson, 1952; Tobias, 1967; White, 1978). In the sections that follow, each type of hypoplasia is described and a review of the literature given.

6.1.1. Pitting enamel hypoplasia

Pitting enamel hypoplasia (PEH) is displayed in a variety of forms, from small circular pin like pits up to vast irregular depressions (Hillson & Bond, 1997; Skinner, 1996). These pits also vary in how they are spread around a tooth crown, with some forming rows around the circumference, usually associated with shallow pits, and others more randomly scattered (Goodman & Rose, 1990; Hillson & Bond, 1997; Figure 6.1). Pitting can also be associated with plane-form defects (e.g., Lauc et al., 2015; Ogden et al., 2007), however in the majority of cases PEH is the only defect observed on a tooth.

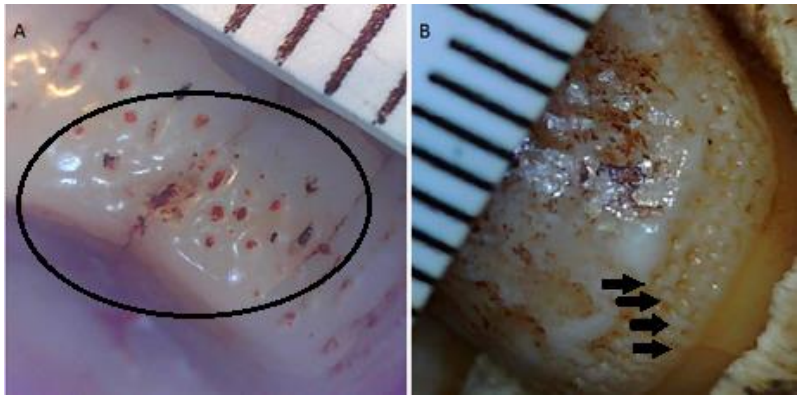

Figure 6.1. A) STW 140: *A. africanus*, maxillary left third molar, relatively random scatter of pitting hypoplasia (black circle); B) M 857: Gorilla, mandibular right canine, more uniform pitting near the CEJ (black arrows).

As pointed out by Hillson & Bond (1997) the position of PEH on a tooth crown does not necessarily give insight into the age the individual was when the defect formed. This is because the depth of a pit is related to its position on the plane of the brown striae of Retzius on which enamel matrix formation ceased. Deep pits may, therefore, represent a disturbance much earlier than their crown position suggests. It is not yet clear why in some cases PEH forms as opposed to other hypoplasia types, particularly LEH. However, the tooth involved, the position on the crown and the cause of the disruption are all likely important factors.

Hillson (2014) notes that PEH is mainly found on the occlusal half of the crown. It is only the occlusal type of perikymata that is affected, and the molars have more of their crowns covered with this type. Thus perhaps explaining why there is usually more PEH on molars than other tooth types (Hillson & Bond, 1997). However, it has also been suggested that because it is relatively uncommon for an individual to have both LEH and PEH, that these different types of hypoplasia may have different aetiologies (Lovell & Whyte, 1999).

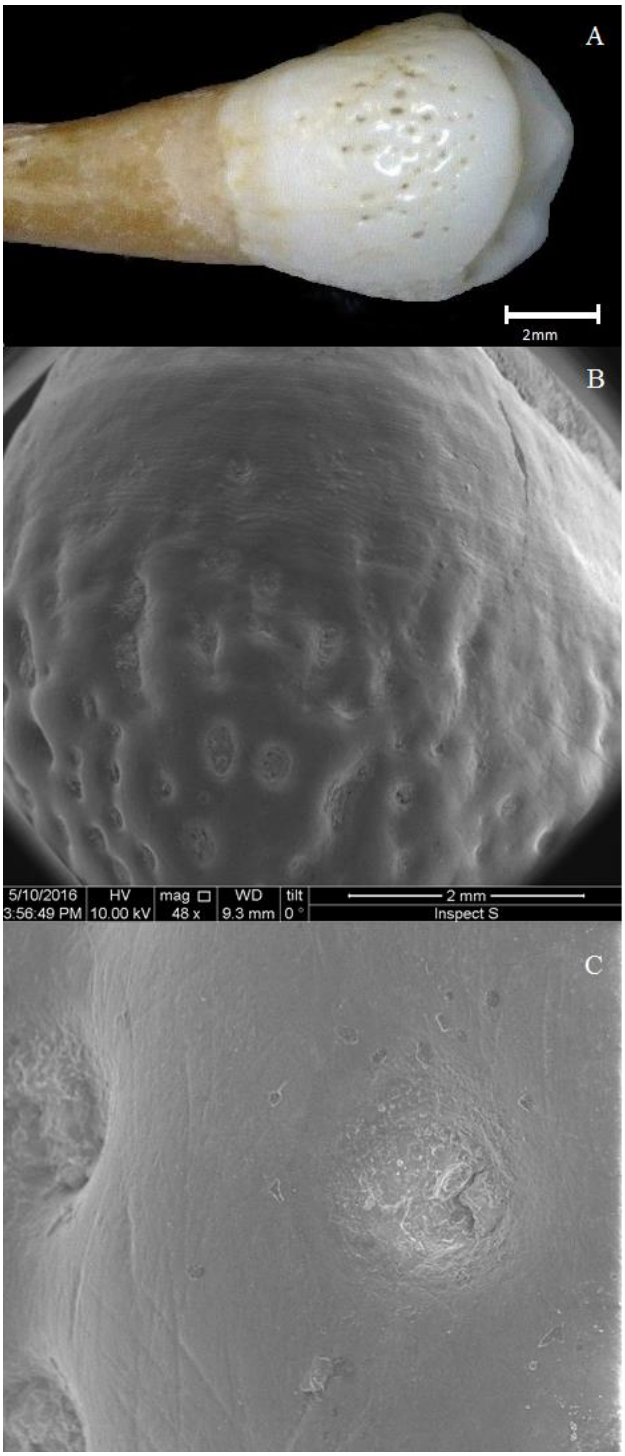

Figure 6.2. Roman era human premolar (Skeleton 1672 from Gloucester): Pitting enamel hypoplasia on the upper left second premolar (A) whole tooth (B) SEM showing pitting and the perikymata below (C) close-up SEM of one of the pits.

Each pit corresponds to the cessation of ameloblast activity at a particular point in enamel formation. It is not clear why only certain ameloblasts are affected along the plane of a brown stria of Retzius during formation (Ogden et al., 2007). In some cases, only a few ameloblasts stop forming enamel matrix, leading to small pits, up to large pits in which hundreds may cease production (Guatelli-Steinberg, 2015). In other forms of systemic enamel hypoplasia, such as LEH and plane form hypoplasia, all ameloblast activity is affected. In most instances of PEH the enamel between pits appears normal (Figure 6.2). Exposed Tomes' process pits can often be observed within pits, showing a sharp end to the ameloblasts (Hillson, 2014). However, some examples show continued deposition of irregular enamel (Hillson & Bond, 1997).

In most cases of PEH in the literature, particularly in archaeological examples, the authors have not been able to specify a cause but rather a non-specific stress was concluded (e.g., Ogden et al., 2007). However, PEH has been associated with a number of specific disturbances in modern clinical studies, including hypocalcaemia, premature birth, low birth weight, hypoparathyroidism, neonatal tetany, maternal diabetes mellitus, kernicterus, vitamin D deficiency, congenital syphilis, amelogenesis imperfecta, as well as general nutritional deficiency (Aine et al., 2000; Croft et al., 1965; Eliot et al., 1934; Gaul & Grossschmidt, 2015; Grahnen & Selander, 1954; Nikiforuk & Fraser, 1979, 1981; Pisanty et al., 1977; Pinhasi et al., 2006; Purvis et al., 1973; Radu & Soficaru, 2016; Seow et al., 1984; Stimmler et al., 1973; Wright et al., 1993). It has been noted that although enamel hypoplasia can relate to genetic conditions, on population bases the vast majority of hypoplasia is a direct response to environmental disturbances (Goodman & Rose, 1990; Moggi-Cecchi et al., 1994; Seow, 1990).

Relatively few studies have reported on different types of hypoplasia and compared their frequencies. Lovell and Whyte (1999) studied a sample of humans from Ancient Mendes, Egypt, finding that linear defects were over three times as common as pitting defects. However, their permanent teeth samples only consisted of anterior teeth. PEH has also been compared between tooth types very rarely. Goodman et al. (1987) compared frequencies of pitting between deciduous and permanent teeth but, again, only for anterior teeth, finding more cases in the permanent dentition. In a modern human sample, Pedersen (1944) found 14% of two to four-year-old children had enamel hypoplasia in their primary teeth. In modern

developed countries, the frequency of hypoplasia in deciduous teeth is typically around 5% or less (Lovell & Whyte, 1999; Robles et al., 2013). However, studies focussing on human samples that have been severely affected by disease, famine or malnutrition have shown much higher rates, ranging from 18% to 62% of teeth affected (Enwonwu, 1973; Infante & Gillespie, 1974; Seow, 1990).

Similarly, differences in PEH frequencies among fossil hominins and extant primates has rarely been explored. PEH has, however, been found on the teeth of various hominin specimens (e.g., Ogilvie et al., 1989; Tobias, 1967; Xing et al., 2015; Zanolli et al., 2016). Some previous studies have noted the presence of PEH in *P. robustus* teeth (Moggi-Cecchi, 2000; Robinson, 1956; White, 1978). These South African hominin studies have been in the context of enamel hypoplasia rates as a whole. Here we build on this research by comparing PEH with other hominins and extant primates to try to highlight species differences of this rarely studied type of defect.

6.1.2. Linear enamel hypoplasia

Linear enamel hypoplasia (LEH) is the most common type of hypoplasia reported in the literature and is commonly found in humans as well as other mammals (Dobney & Ervynck, 2000; Goodman & Armelagos, 1985; Guatelli-Steinberg, 2004; Guatelli-Steinberg & Lukacs, 1999; Skinner et al., 2015). These defects have been studied in a variety of hominin species, including South African fossil specimens (e.g., Guatelli-Steinberg, 2003, 2004). Linear enamel hypoplasia is characterised by grooves, lines or furrows on the enamel surface, which are caused by a variety of systemic stresses (Guatelli-Steinberg, 2015; Figure 6.3).

Linear enamel hypoplasia is found more commonly on anterior than posterior teeth. This is due to anterior teeth having more lateral enamel, on which LEH is found, compared to cuspal enamel. Another potential reason is due to the curved surfaces of posterior teeth which may affect the angle the perikymata are displayed on the crown surface, meaning LEH bands tend to be shallower and less pronounced compared to anterior teeth (Goodman & Rose, 1990; Hillson & Bond, 1997). LEH defects are created when perikymata spacing increases beyond normal width for a particular position on a tooth crown (Hillson, 2014). As well as being an indicator of developmental disturbance, LEH defects are also commonly aged,

through several different techniques that have been proposed (Goodman & Armelagos, 1985; Reid & Dean, 2000).

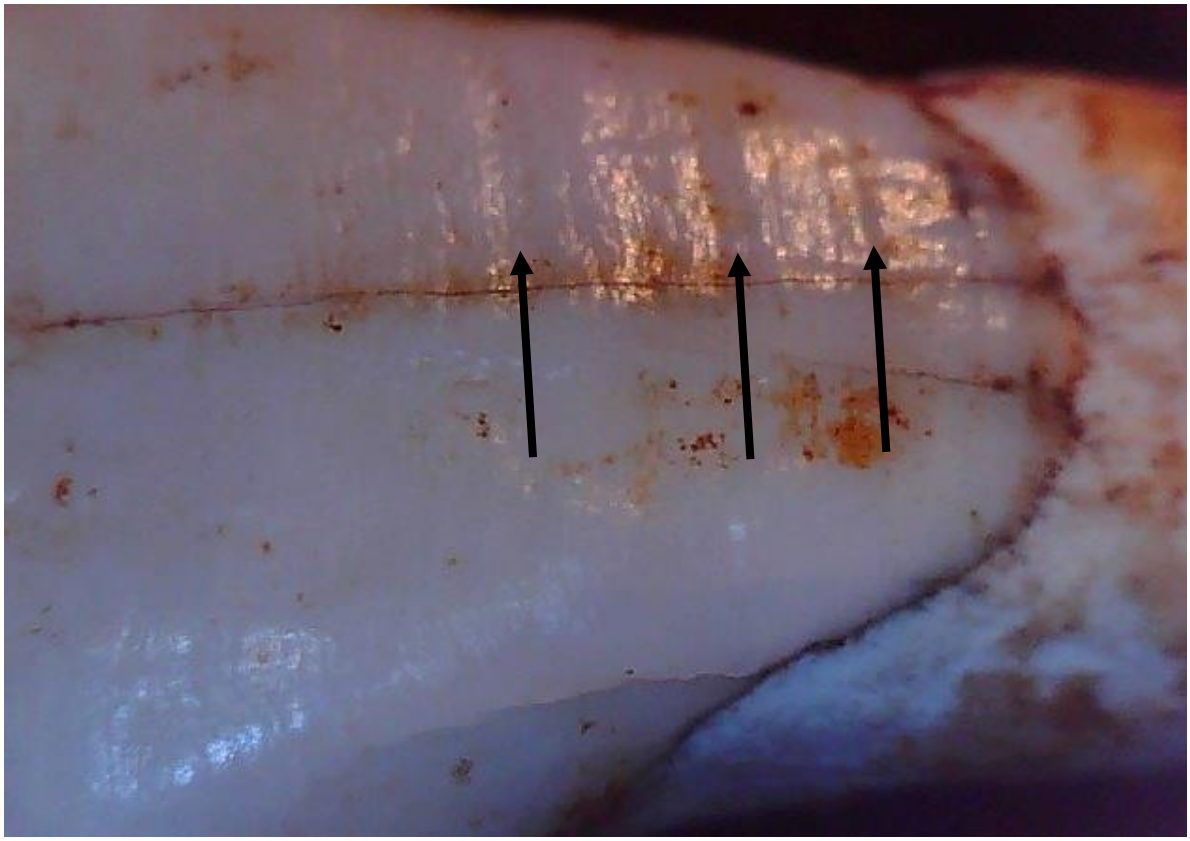

Figure 6.3. *H. naledi* (UW 101-38), upper right central incisor. Numerous bands of LEH on the buccal surface. Arrows highlighting LEH bands.

6.1.3. Localised hypoplasia

Localised hypoplasia is characterised by isolated areas of hypoplasia that typically do not extend around the crown, and are commonly only made up of one or two continuous defects (M.F. Skinner et al., 2016; Figure 6.4). The cause of these localised defects is usually suggested to be trauma during development (see below). Although not commonly recorded, this type of hypoplasia has been studied in depth in certain species. Localised hypoplasia of the deciduous canine is particularly well studied, with rates varying significantly between different primate species (M.F. Skinner et al., 2016; M.F. Skinner & Newell, 2003).

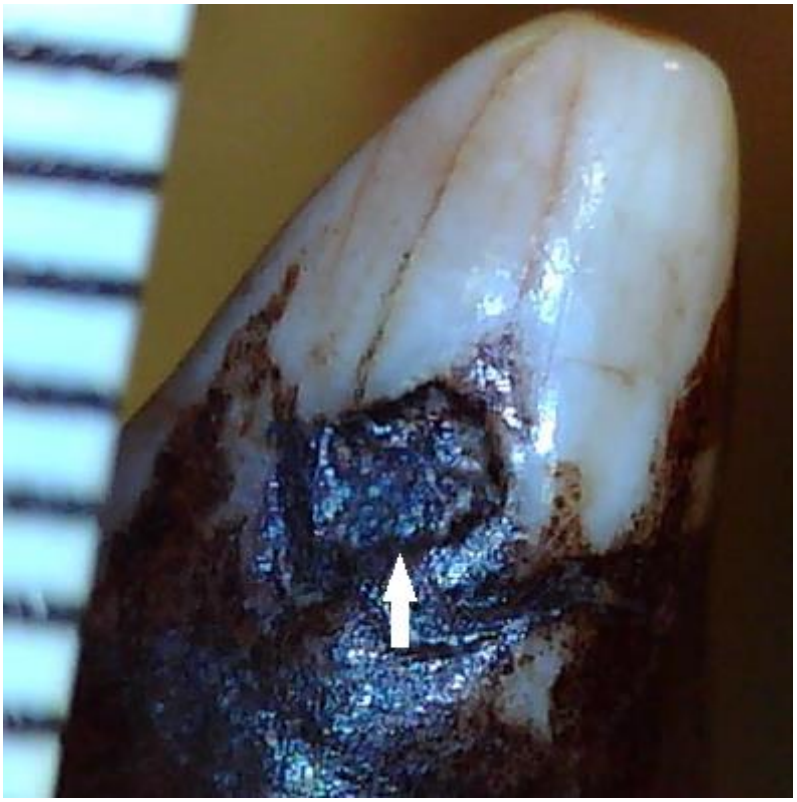

Figure 6.4. Gorilla (M 667): left lower deciduous canine, localised hypoplasia (arrow).

Before teeth erupt into the oral cavity, they are usually present in a crypt of their own. However, occasionally the alveolar bone between adjacent teeth that normally separate crypts may be missing leading to fenestration (M.F. Skinner et al., 2016). It has been suggested that it is this process that commonly leads to the formation of localised hypoplasia, or at least certain kinds (Skinner, 1986; Skinner & Newell, 2003). As pointed out by M.F. Skinner (2016) if this hypothesis is correct, then understanding what may cause these crypt fenestration defects, or at least why there are species and population differences, is the next important question to consider. Skinner and colleagues have started this research by suggesting a link between Vitamin A deficiency and reduced bioavailability and higher instances of localised hypoplasia in primates (Skinner et al., 1994; Skinner & Newell, 2003). Therefore, if correct, the overarching reason for the cause of these defects is that they reflect deficient growth in infancy of the mandible and maxilla (Lukacs, 1999; M.F. Skinner et al., 2016). The hypothesis being that if malnutrition, or another stress, occurs during a critical stage of development, then the jaws may be smaller than they would have been otherwise, and therefore dental crowding could result. Fenestration follows, and ultimately then localised hypoplasia. The only real opposition to this theory is that these defects could be caused by similar effects to

other types of hypoplasia (Hillson, 2014). There does appear to be a link between general ill health and an increase in localised enamel hypoplasia, however neither theories dispute this link (Koch, 1999; Scheutzel & Ritter, 1989; Silberman et al., 1991; Skinner, 1986; Skinner & Hung, 1989).

By studying extant primates it is hoped insight into this debate can be given in this study. Specifically to try and address why certain species have such high levels of localised hypoplasia compared to others. This will be done by exploring if individuals with localised defects are more likely to have other types of hypoplasia.

6.1.4. Plane-form hypoplasia

The terminology used to describe plane-form enamel hypoplasia is often confusing and hard to interpret. Plane form enamel hypoplasia occurs when enamel matrix formation ceases, either entirely, or in part. This results in areas of an affected crown with little or, in severe cases, no enamel deposition (Hillson & Bond, 1997; Krenz-Niedbała & Kozłowski, 2013; Ogden et al., 2007). Such defects have been described as extreme furrow-form defects, with one perikymata significantly widened (Hillson, 2014). In rare circumstances, the whole plane of the brown stria can be exposed down to the dentine surface (Figure 6.5).

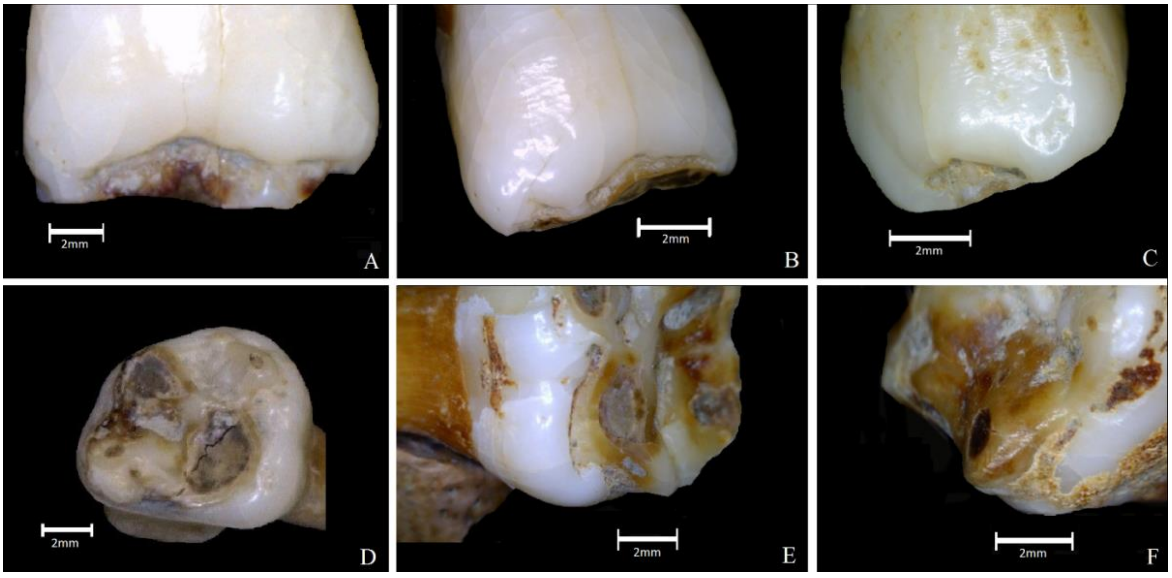

Figure 6.5. Roman era human (Skeleton 1672): Plane form enamel hypoplasia defects. (A) Upper left central incisor; (B) upper right central incisor; (C) upper right canine; (D) upper left first molar; (E) upper right first molar; (F) lower right first molar.

Plane-form hypoplasia has only been reported in the literature on fossil hominins as either mild in form or as part of a localised defect (Guatelli-Steinberg, 2003; Littleton & Townsend, 2005; M.F. Skinner et al., 2016). In this study localised hypoplasia is not included as a plane form defect, as the latter is reserved for systemic defects.

6.2. Materials and Methods

Methodology used in recording enamel hypoplasia varies substantially between studies. Most commonly, researchers only record and compare LEH frequencies (e.g., Guatelli-Steinberg, 2003, 2004; Miszkiewicz, 2015; Smith et al., 2016). Others include all hypoplasia defects (e.g., Goodman et al., 1980, 1984; Goodman & Armelagos, 1985; Ogilvie et al., 1989). Some studies only record defects on certain teeth, with posterior and deciduous teeth often excluded (e.g., Infante & Gillespie, 1974; Lovell & Whyte, 1999). Additionally, it is not clear in some cases if the pitting hypoplasia mentioned only refers to defects found as part of LEH bands (e.g., Goodman et al., 1980, 1984; Goodman & Armelagos, 1985). Other methods have been used to classify defects. This includes M-hypoplasia which is named after May Mellanby due to work she carried out on these defects (Mellanby, 1929). This method splits hypoplastic defects into two categories, gross defects and M-hypoplasia defects (Hillson, 1992; Sognnaes, 1956). However, it is not clear at which point a pitting defect falls into these two categories, with Hillson (1992) remarking that all pitting defects are recorded as gross defects, but earlier work by Sognnaes (1956) seems to include at least some pitting defects in the M-hypoplasia category.

Due to how defects are displayed on the crown of a tooth, Hassett (2012) concluded enamel hypoplasia prevalence based solely on macroscopic observation could be misleading and create biases when comparing populations. To add to this debate, however, it has now also been suggested that microscopic techniques likely miss defects too, with micro-CT imaging showing clearly enamel defects that did not show up on SEM or light microscopy (Marchewka et al., 2014; Xing et al., 2015). In this study defects are only viewed macroscopically. However, micro-CT scans are used when describing the pitting hypoplasia found on *P. robustus* teeth.

All species have a substantial amount of teeth for which hypoplasia was not recorded due to the crown being damaged or discoloured post-mortem (Table 6.1).

Table 6.1. Number of observable teeth for each species.

Species	Teeth observable	Not observable	% Unobservable
Early *Homo*	47	19	28.79
A. sediba	10	1	9.09
P. robustus	304	127	29.47
H. naledi	142	14	8.97
A. africanus	360	122	25.31
Gorillas	1693	392	18.80
Chimpanzees	1837	677	26.93
Baboons	774	92	10.62

6.2.1. Specific methods

Specimens are examined macroscopically, with a 10x hand lens used to clarify defect types. The presence and position of linear, localised, pitting and plane-form hypoplasia is recorded for each tooth, as well as the size and shape of the defect noted.

Methods for recording LEH follow Goodman & Rose (1990), Guatelli-Steinberg (2003), Lukacs (1989), and Miszkiewicz (2015). All teeth were placed under a lamp with the tooth slowly rotated so that light hit at a variety of angles. The smallest discernible LEH defect was recorded; this includes all defects visible with the naked eye, with the hand lens only used to confirm defect type. The distance from the middle of the LEH defect to the CEJ was also measured. If the CEJ was not visible or was broken, then the LEH was still recorded, but no distance was noted. Linear enamel hypoplasia data on South African hominins has already been the subject of extensive research by Guatelli-Steinberg (2003, 2004). Therefore LEH on its own is not the focus of this study. Instead, however, LEH has not been comprehensively compared with other types of hypoplasia and then compared with other species. Therefore, LEH is recorded again here to make sure the same specimens are recorded for each type of defect.

Pitting and localised hypoplasia were both recorded and the defects described, but measurements are not taken due to the often irregular shape of these defects and lack of understanding regarding ageing (Hillson, 2014). Localised hypoplasia was recorded following

M.F. Skinner et al. (2016). The presence of PEH is recorded, and notes on its position described. Additionally, micro-CT scans of some of the *P. robustus* molars are viewed and defect size and number recorded. If pits are part of an LEH defect then the position of the LEH was recorded and pitting noted; however, this pitting is not included as part of the PEH analysis.

Data are presented by tooth count, in which the number of hypoplastic teeth is displayed as a percentage of the total number of observable teeth. Due to the fragmentary nature of the hominin collections this is the best way to get a large enough sample for comparison, but as Lovell & Whyte (1999) note, displaying by per tooth and not per individual allows a broader comparison for both subsamples of different tooth groups and with other populations. Antimeres are treated as separate data points in the overall hypoplasia frequencies. This enlarges the sample and allows maximum recovery of information. Two additional points justify the inclusion of antimeres, first the fact that some defects may be displayed on one antimere and not the other, in particular, localized defects, and secondly the nature of the fossil record in some cases makes assigning antimeres difficult. As the results are presented as a percentage of all teeth affected this also allows the exact frequencies of hypoplasia to be presented and allows better comparisons with other samples.

Instead of rejecting teeth that are worn past a certain point, all teeth that are not broken due to post-mortem damage are included. This will clearly lead to teeth being included that have had enamel defects worn away. However, the alternative of excluding such teeth will also lead to issues, since this will mean an entire sample is made up of individuals that died young i.e. potentially they were more ill, on average, during dental development than the individuals that lived to old age. However, in Chapter 12 we compare the wear scores and enamel hypoplasia frequencies for each species, to see if there is any bias caused by certain samples having more severely worn teeth.

Unless stated, results refer to permanent teeth only. To compare certain defect types, as well as to compare species and sexes, a chi-square test of homogeneity, with significance set at the 0.05 alpha level, was used.

6.3. Results

Table 6.2 displays the frequency of the different types of hypoplasia in the permanent teeth of the different species. The hominins have higher rates of LEH than the extant great apes, with baboons having the lowest frequency (barring *A. sediba* that has a small sample size). There are low levels and very little variation in species for localised hypoplasia and PEH, except for *P. robustus*. There is much more PEH in the *P. robustus* sample than in the other species, with almost three times the rate of *A. africanus* (14.75% and 5.03% respectively). Indeed, *P. robustus* has more teeth with PEH than it does with LEH, which is exceedingly rare in samples of permanent teeth.

Table 6.2. Per tooth frequencies (%) of pitting enamel hypoplasia (PEH), Linear enamel hypoplasia (LEH), and localised hypoplasia for permanent teeth of each species.

Permanent	PEH	LEH	Localised
Chimpanzees	0.65	8.06	0.98
Gorillas	2.89	4.25	0.95
Baboons	0.00	2.07	1.68
H. naledi	0.70	14.79	0.70
A. africanus	5.03	15.08	0.28
P. robustus	14.75	11.51	1.08
Early *Homo*	0.00	8.51	2.13
A. sediba	0.00	0.00	0.00

The high rates of PEH of the permanent teeth of *P. robustus* are due predominately to molars being affected, although the premolars also have high rates compared to the other hominin species (Table 6.3). The three molars are similarly affected with over 20% showing PEH defects for each. Linear enamel hypoplasia is less common in *P. robustus* than either *A. africanus* or *H. naledi*.

Table 6.3. Per tooth frequencies (%) for pitting enamel hypoplasia (PEH) and linear enamel hypoplasia (LEH) for different tooth groups of permanent teeth.

Permanent teeth	***P. robustus***		***A. africanus***		***H. naledi***	
	PEH	LEH	PEH	LEH	PEH	LEH
Anterior teeth	1.75	22.81	2.11	40.00	2.04	24.49
Premolars	7.04	9.86	0.00	6.67	0	23.08
First molars	21.43	5.36	6.90	5.17	0	0
Second molars	21.57	5.88	9.09	6.06	0	0
Third molars	25.58	11.63	10.20	4.08	0	0

Localised hypoplasia is not found on any of the hominin deciduous tooth sample (Table 6.4). This is in contrast with the extant primate sample in which it is relatively common, especially in gorillas (12.93%). In deciduous teeth PEH is rare in all samples except *P. robustus*, in which over 40% of teeth have defects.

Table 6.4. Per tooth frequencies (%) of pitting enamel hypoplasia (PEH) and localised hypoplasia for deciduous teeth of each species.

Deciduous	**Pitting**	**Localised**
Chimpanzees	4.23	5.08
Gorillas	1.39	12.93
Baboons	0.00	3.74
H. naledi	0.00	0.00
A. africanus	5.00	0.00
P. robustus	41.30	0.00
Early *Homo*	14.29	0.00

The PEH defects found on deciduous teeth of *P. robustus* are almost all found on molars (Table 6.5). Both first and second molars are similarly affected, with 54% of first molars and 52% of second molars exhibiting PEH, with the crown often completely covered in PEH (Figure 6.6). In both the permanent and deciduous teeth of *P. robustus*, the severe PEH often covers large areas of the crown and is characteristically made up of numerous relatively uniform small depressions. When pits do not cover the whole crown, they are typically more defined and prevalent toward the occlusal surface. These defects vary little in size or shape,

and hundreds of separate pits are often visible across the crown, with defects in both permanent and deciduous teeth being very similar (Figure 6.6; Table 6.6). In deciduous molars that have antimeres and PEH, both tend to have identical patterns of defects. For example, the right and left second deciduous molars of SK 61 not just have the same general covering of uniform pits but the areas where the most defined defects can be seen is the same. These pits are clearly not post-mortem in nature, and the enamel does not seem to have been reduced (except for the defects themselves) and appears to be of normal density (Figure 6.7). The rate of PEH seems to be similar between *P. robustus* sites. At Kromdraii 16.67% of permanent teeth show PEH, and Swartkrans this figure is 14.34%. Anterior teeth that are associated with permanent molars that have PEH do not show a significant increase in LEH defects, with only 16.67% affected.

Table 6.5. Percentage of deciduous *P. robustus* teeth with pitting enamel hypoplasia (PEH).

Deciduous Teeth	PEH %	Total teeth	Teeth with hypoplasia
All teeth	41.30	46	19
Anterior teeth	8.33	12	1
First molars	53.85	13	7
Second molars	52.38	21	11

Table 6.6. Number and size of *P. robustus* pitting defects. Pit size is in millimetres. L: left; R: right; d: deciduous; M1: first molar; M2: second molar. All mandibular teeth except SK 89.

Specimen	Number of pits	Minimum pit size	Maximum pit size	Average
SK 61: RM1	50+	0.12	0.36	0.23
SK 61: RdM2	80+	0.11	0.28	0.17
SK 61: LdM2	80+	0.12	0.26	0.18
SK 63: RdM2	100+	0.11	0.42	0.23
SK 64: RdM2	300+	0.12	0.21	0.16
SK 89: LM1	100+	0.12	0.26	0.16

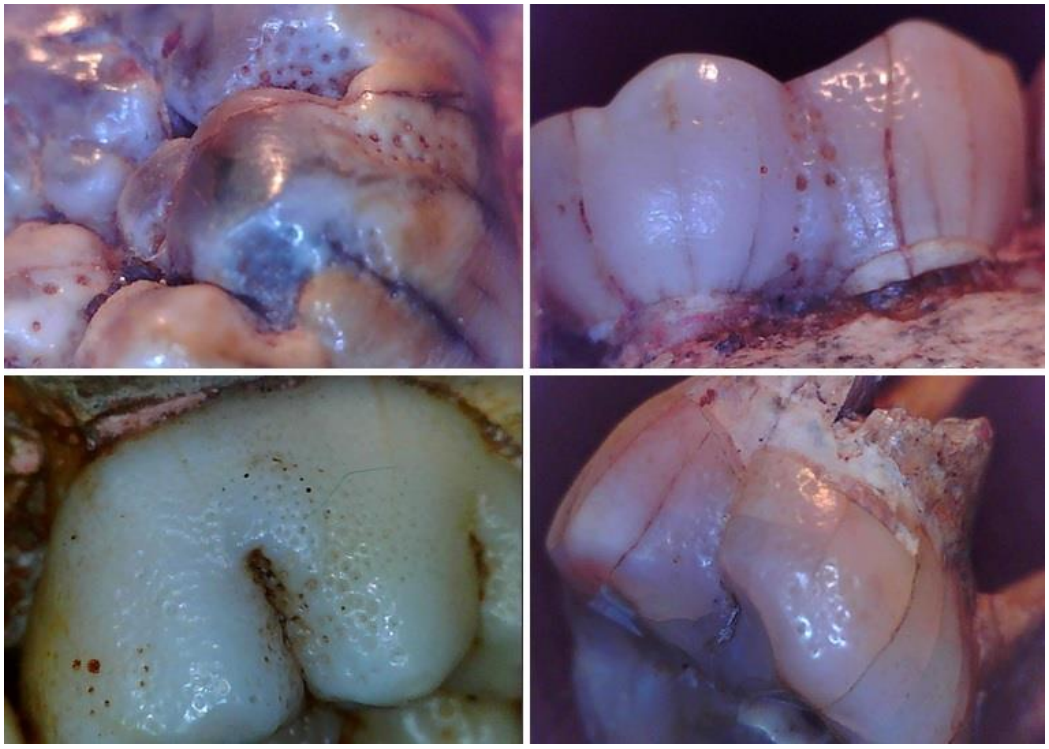

Figure 6.6. Uniform circular pitting enamel hypoplasia on four *P. robustus* teeth. Top left: SK 61; top right: SK 63; bottom left: SK 64; bottom right: SK 90.

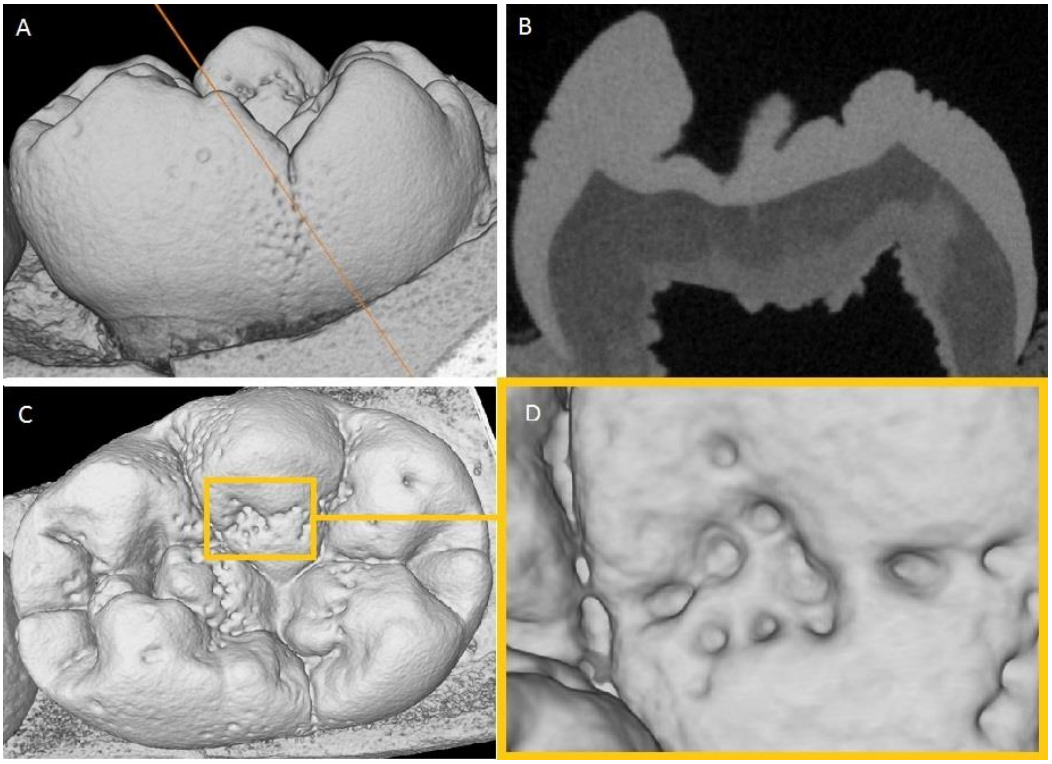

Figure 6.7. Micro-CT scan slices of SK 64 (*P. robustus*), the right mandibular first molar. A) Overview of the lingual surface with the orange line being the position of the slice in B; B) Slice showing pitting on both the lingual and buccal surfaces. C) Occlusal view; D) Close up of pitting on the occlusal surface.

No plane-form defects (not including localised hypoplasia) were recorded in any of the hominin samples. For each species, PEH, LEH and localised hypoplasia frequencies are displayed in Figure 6.8 (permanent teeth) and Figure 6.9 (deciduous teeth). Although *P. robustus* has the lowest frequency of LEH of the hominins, the high rate of PEH means it has the highest overall rate of hypoplasia of any species. When compared directly with the species with the second highest levels of hypoplasia, *A. africanus*, there are no statistically significant differences for LEH (X^2= 1.678, 1 df, p= 0.1953) or overall hypoplasia (X^2= 1.604, 1 df, p= 0.2053). However, there is a statistically significant difference in PEH (X^2= 14.823, 1 df, p= 0.0001). This is also true for deciduous teeth, with *P. robustus* having significantly more PEH defects than *A. africanus* (X^2= 5.824, 1 df, p= 0.0158).

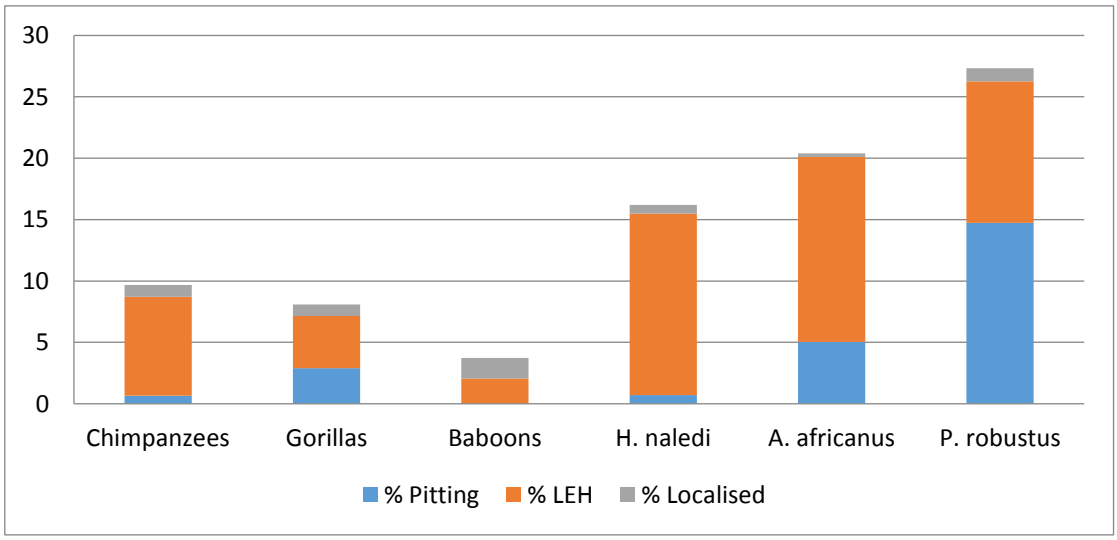

Figure 6.8. Frequency (%) of the different types of hypoplasia on permanent teeth.

The number of teeth with LEH is similar across hominin species, ranging from 12% in *P. robustus* to 15% in *A. africanus*, although the latter shows much higher rates in anterior teeth than both *P. robustus* and *H. naledi* (40%, 23% and 24% respectively). The position in which LEH defects are found on the crown varies substantially between the extant primate and hominin samples. In the hominins LEH is usually a lot closer to the CEJ than in the comparative primates (Means, *P. robustus*: 3.59mm; *A. africanus*: 4.25mm; *H. naledi*: 2.90mm; baboons: 8.30mm; gorillas: 7.05mm; chimpanzees: 5.80mm). This is likely due to crown sizes and developmental timings varying substantially among groups.

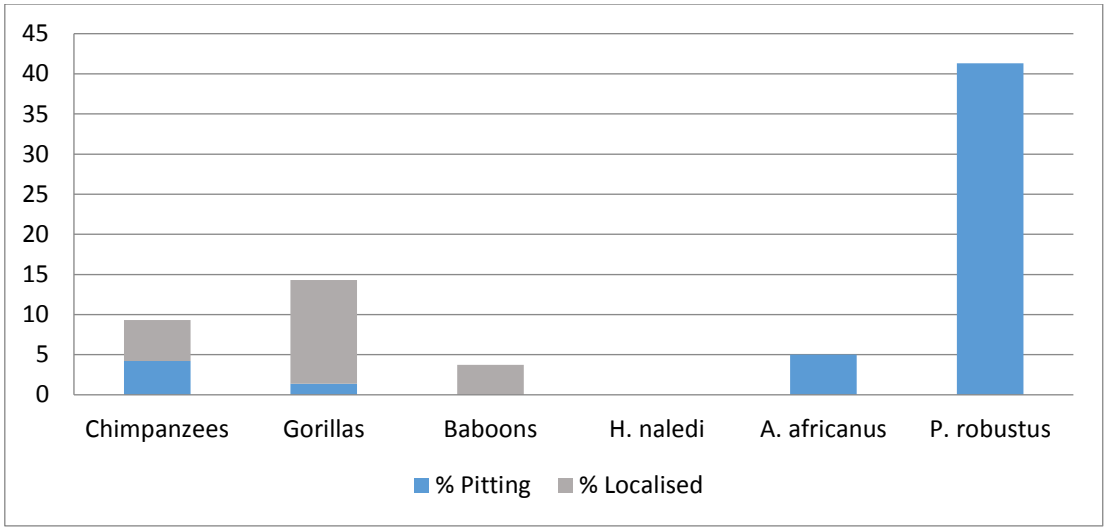

Figure 6.9. Frequency (%) of the pitting enamel hypoplasia and localised hypoplasia on deciduous teeth.

Perhaps surprisingly, when individuals with and without localised hypoplasia are analysed separately, there is more PEH in the group with no localised hypoplasia for both gorillas and chimpanzees. For chimpanzees, in individuals with at least one localised defect, 1.2% of teeth have PEH, whereas for individuals with no localised defects 5.42% of teeth have PEH. For gorillas, the figures are 0% and 5.77% respectively. For both species this is a statistically significant difference (gorillas: X^2= 12.533, 1 df, p= 0.0004; chimpanzees: X^2= 4.416, 1 df, p= 0.0356).

Males have more enamel hypoplasia, of all types, than females in baboons and gorillas, although these differences are not statistically significant (Table 6.7). Chimpanzees, on the other hand, show the opposite trend with localised defects, as well as PEH+LEH, being more common in females. The PEH+LEH difference is statistically significant (X^2= 5.194, 1 df, p= 0.0227). There are no statistically significant differences between the sexes in deciduous teeth, with the rates for both PEH and localised defects very similar (Table 6.8).

Table 6.7. Per tooth frequency (%) of hypoplasia split by sex for permanent teeth of the extant primate samples.

Permanent teeth	**Gorillas**		**Chimpanzees**		**Baboons**	
	Males	Females	Males	Females	Males	Females
Total teeth	793	874	565	1264	484	216
No hypoplasia	717	813	526	1125	462	212
Teeth with localised hypoplasia	11	5	2	16	10	2
% of Localised hypoplasia	**1.39**	**0.57**	**0.35**	**1.27**	**2.07**	**0.93**
Teeth with PEH or LEH	65	56	37	123	12	2
% of PEH/LEH defects	**8.20**	**6.41**	**6.55***	**9.73***	**2.48**	**0.93**

*Chi-square significant at 0.05 level

Table 6.8. Per tooth frequency (%) of hypoplasia split by sexes for deciduous teeth of the extant primate samples.

Deciduous teeth	**Gorillas**		**Chimpanzees**	
	Male	Female	Male	Female
Total teeth	186	241	312	233
No hypoplasia	155	212	282	211
Teeth with localised hypoplasia	25	29	17	11
% of Localised hypoplasia	**13.44**	**12.03**	**5.45**	**4.72**
Teeth with PEH	6	0	13	11
% of PEH defects	**3.23**	**0.00**	**4.17**	**4.72**

6.4. Discussion

Clearly a variety of stresses, illnesses and malnutrition can cause LEH, and likely most PEH defects (Bowman, 1991; Dirks et al., 2002; Guatelli-Steinberg, 2001; Kelley & Bulicek, 2000; Macho et al., 1995; Skinner et al., 2015). The results of this study agree with previous research that has shown higher rates of LEH in great apes than other primates (e.g., Guatelli-Steinberg, 2001; Moggi-Cecchi & Crovella, 1991). With apes being slow growing mammals their teeth are exposed to a longer time span in which teeth may be adversely affected during formation, this is particularly the case for hominins. Thus, individuals likely have to live through disease, nutritional deficiencies and seasonal cycle related stresses to some extent

during this sensitive period (Zihlman et al., 2007). Similarly, *P. robustus* canines have less LEH than both *Australopithecus* and early *Homo*, likely due to the shorter crown, and therefore developmental time, meaning there is less opportunity for a defect to form (Guatelli-Steinberg, 2003, 2004). This is the result also found here, with *P. robustus* having the lowest rate of LEH for any of the hominin samples.

There is much debate over whether PEH is caused by different factors than LEH, or if it is simply a consequence of the tooth and crown position involved (Hillson, 2014). Recent research suggests both may be correct, at least to an extent. For example, PEH is commonly present without LEH, such as in many cases of amelogenesis imperfecta and conditions such as congenital syphilis (Crawford et al., 2007; Hillson, 2014; Lauc et al., 2015). Additionally, there are many examples of the same disturbance events causing PEH on one tooth and LEH on another. Therefore, the answer is clearly going to be a mix of these two viewpoints.

An issue highlighted in this study relates to the idea that PEH only forms on certain areas of a tooth crown and is therefore simply a result of developmental timing. Although much rarer than normal LEH grooves, pitting form LEH was recorded in most species studied. When a pitting LEH defect is present, it is much more likely that another such defect will be present elsewhere on the crown surface, in positions that 'normal' LEH defects commonly occur in other specimens (e.g., Figure 6.10). This would not be expected if such pitting was wholly caused by the position of the crown. That said, certain teeth in this sample were much more likely to show pitting than other teeth, particularly posterior teeth of *P. robustus* and canines of gorillas. These two observations add support to the suggestion that pitting defects are likely caused by a variety of factors, including specific condition/disturbances that only cause pitting, but also systemic generalised stress that may cause PEH or LEH depending on the tooth and tooth position involved. How these different types of PEH can be distinguished may be difficult, however, in certain conditions it is already possible. For example, there are often particular patterns associated with amelogenesis imperfecta (Crawford et al., 2007; Chapter 10). Given the advancement of techniques such as micro-CT scans and SEM in describing enamel defects, this may lead to a better understanding of how and why these different defects form.

Tobias (1967) described the third molars of OH 5, the type specimen of *P. boisei*, as having rugose and irregular enamel. The other teeth of this individual are also affected but by

more defined regular defects, particularly PEH. The irregular and wavy enamel on the third molars in some ways resemble that in some *H. naledi* specimens. No current hypoplasia definitions cover this type of defect, and it is, therefore, hard to know how to characterise them. This 'wrinkly' or 'wavy' appearance was noted in some teeth within the sample of this thesis but seemed particularly common in *H. naledi*. This has not been recorded as a hypoplasia, as it was not part of the methodology, however, photos and notes were taken. This type of abnormality has been described before, and it is not easy to know if it is a type of vertical hypoplasia or simply an effect of the morphology of tooth formation. For example, Xing et al. (2015) at first propose vertical enamel hypoplasia for abnormalities on an incisor, but after viewing CT-scans of the tooth, they conclude that, because the same feature is present at the enamel-dentine junction, that the effect is caused by epithelium folding during development. Therefore many of the instances where 'wavy', 'wrinkled' or 'potential vertical hypoplasia' have been recorded in this thesis are potentially a feature of the crowns morphology rather than being caused by a disturbance to ameloblast secretion, and therefore unlikely caused by periods of stress or illness. However, further research is needed on this type of defect before an aetiology can be proposed. Since such 'wavy' enamel is found much more commonly in certain populations, such as the *H. naledi*, it would be interesting to explore if this can be attributed to dental morphological differences compared to other hominins.

Recent publications have noted unusual vertical groove defects on the incisors of primates (Hannibal et al., 2016; M.F. Skinner et al., 2016). These two papers, published at the same time, highlight similar defects found on the lateral maxillary incisor and are thought to be a developmental defect rather than simply variation in morphology. The primate data for this thesis was recorded before these two publications; however similar unusual defects were recorded. That said, these defects are not similar to 'usual' localised defects and are more likely to be confused with morphological features, therefore given the fact these defects were not specifically looked for during data collection inferences and comparisons with these two studies would be difficult.

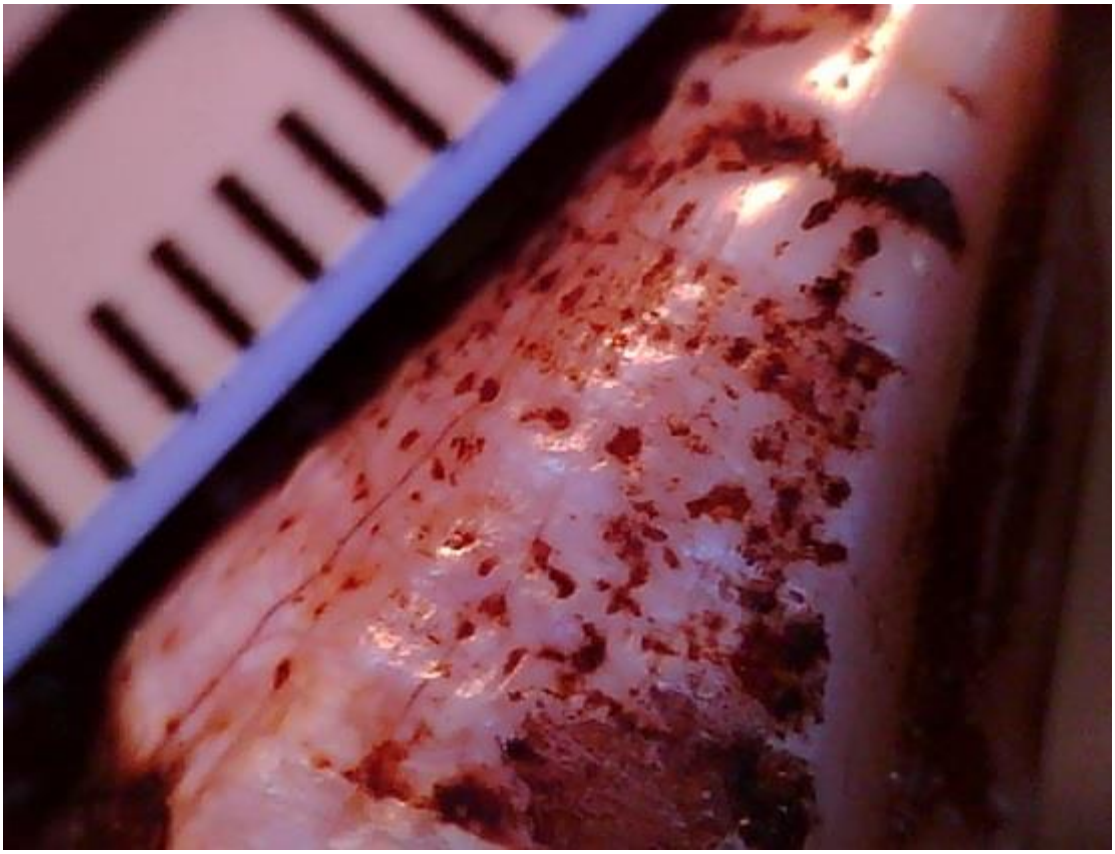

Figure 6.10. Linear enamel hypoplasia in pitting form. Chimpanzee upper left permanent canine, buccal view.

More generally, there are still issues that need to be addressed regarding the aetiology of localised defects. As mentioned, Skinner and colleagues believe localised hypoplasia are caused by contact between the developing tooth and an opposing force, either an adjacent tooth or the surrounding bone, or the lack thereof. Dental crowding is suggested as a potential primary cause (M.F. Skinner et al., 2016). This hypothesis would also explain why localised hypoplasia is often found more often in ill individuals i.e. malnutrition results in a reduced jaw size which results in overcrowding which the creates localised hypoplasia. In their recent paper, M.F. Skinner et al. (2016) suggest there is such a relationship between malnutrition and overcrowding in humans. However, the only paper referenced to support this claim does not clearly show a relationship between malnutrition and overcrowding (Thomaz et al., 2010). As Thomaz et al. (2010) state "An association between low height-for-age (z-score < −1SD) and crowding were only observed in adolescents with a prolonged history of mouth breathing (OR = 3.1). No association was found between underweight and crowding. Malnutrition is related to crowding in permanent dentition among mouth-breathing adolescents". Mouth

breathing increases the likelihood of numerous dental pathologies and the relationship found in this study could, therefore, be the result of complicated relationships between mouth breathing, malnutrition and other pathologies (Newman et al., 2011; Thomaz et al., 2010). A larger sample from a diverse set of populations may show such a relationship, but at present, there is not enough evidence to support a connection between malnutrition, dental overcrowding and localised hypoplasia.

The fact individuals with localised defects on their deciduous canines do not show higher rates of other forms of hypoplasia also suggests general poor health is not directly related to increased localised hypoplasia rates. Certainly, it appears a systemic disturbance during development is not a prerequisite for localised hypoplasia. It is also worth noting; it is the species with the least amount of PEH and LEH in this study that have the highest rate of localised hypoplasia. That said, studies on other mammal species have shown that malnutrition may cause localised enamel defects in certain populations (Dressino & Pucciarelli, 1997; Garat et al., 2006; McCance & Ford, 1961; Tonge & McCance, 1973). Although these studies, on primates, rats and pigs, are not based on wild populations and the animals involved were subject to severe starvation and malnutrition. Therefore in many cases, particularly with localised defects of the deciduous canine, it seems poor health is perhaps not the main driving factor behind high frequencies in certain extant primate species. This is also supported by the lack of sex differences in samples with high rates, such as gorillas and chimpanzees. In particularly, the results of this study also highlight the unlikelihood that localised defects are caused by the same factors as PEH and LEH, as has been suggested (Hillson, 2014). Localised defects are often not related to other types of defects and commonly do not occur on antimeres, therefore a systemic cause on its own seems extremely unlikely. This is also the case with the small amount of localised defects found in the hominin sample, which are found in a variety of tooth types and in no case are they found on antimeres or other teeth in the dentition (Figure 6.11).

Figure 6.11. Localised hypoplasia above a LEH defect (arrow) on the second molar of STW 327 (*A. africanus*).

Tobias (1967) notes that OH 5 has a hypoplastic defect on the left upper canine that is not found on the right canine. He notes that it is similar to a published example found on a baboon right maxillary central incisor (Colyer, 1936). This defect is what we would now describe as localised hypoplasia and it can be clearly seen and compared with similar examples highlighted in this project, found commonly in the gorillas.

Amelogenesis imperfecta has only once been suggested in a fossil hominin (Zilberman et al., 2004). However, given recent research into different types of hypoplasia, there is not enough evidence in this case for such a conclusion (Zanolli et al., 2016). Indeed, given the broad range of how defects can be displayed, it may be tough to diagnosis amelogenesis imperfecta in the fossil record unless a full dentition is available (see Chapter 10).

6.4.1. Localised vs. pitting enamel hypoplasia

Comparisons with other mammals offer potential insight into defect properties. Toxodon teeth show a variety of different types of enamel hypoplasia (Braunn et al., 2014). Interestingly one defect that was common in these fossils is lines of vertical pits. The authors suggest such defects may relate to enamel fold formation. Although not directly applicable to primate teeth, due to Toxodon teeth continuously growing during life, it nonetheless raises the possibility that some PEH defects may in fact not relate to development disturbance or stress, but be more akin to localised hypoplasia. This possibility further blurs the line between what is characterised as PEH and localised hypoplasia. If pitting defects are commonly found in the same regions of the crown in different individuals, especially in areas of undulating

enamel, then it may be possible that defects relate directly to enamel formation rather than a stress experienced by the individual. This is unlikely the case for the pitting found on *P. robustus* given the fact pitting is usually scattered on the crown. There are a few instances in the material studied here in which pitting is present in undulating enamel, particularly on the buccal side near the occlusal surface. The possibility that these are the result of such factors will need to be explored further in extant species.

Therefore the definition of what pitting and localised defects actually mean regarding appearance and aetiology is further complicated. Clearly, as it stands, both can be caused by non-systemic factors, with pitting being caused by disturbance during development but also can be caused by heritable conditions (Crawford et al., 2007; Sundell & Koch, 1984). Localised enamel hypoplasia defects may not be isolated, with multiple defects potentially being displayed on the same crown. Furthermore, localised defects may be present on anti-meres, in adjacent teeth, and those forming at the same time. For example, in gorillas, it is common to have a localised defect on both lower deciduous canines. This means the current classification of localised hypoplasia may no longer be appropriate, especially when comparing species.

An extreme example, but one that illustrates this issue well, is a male chimpanzee from the Powell-Cotton Collection. This juvenile, with a full deciduous dentition, has defects on his maxillary canines, lateral incisors, and right first molar, as well as all mandibular teeth except the deciduous second molars. For most of the teeth, if found in isolation, a researcher would likely record the defects as being localised hypoplasia (Figure 6.12). Some defects may also be recorded as PEH. The fact that so many teeth are affected, and the numerous defects on certain teeth, make a differential diagnosis more complicated. Amelogenesis imperfecta must also be considered. However, the fact an antimere is not affected, and the pattern of the defects are different on each tooth, suggests these defects may not be best described as PEH. This case highlights the major issue in studying enamel hypoplasia in fossil hominins, which predominantly consist of isolated teeth.

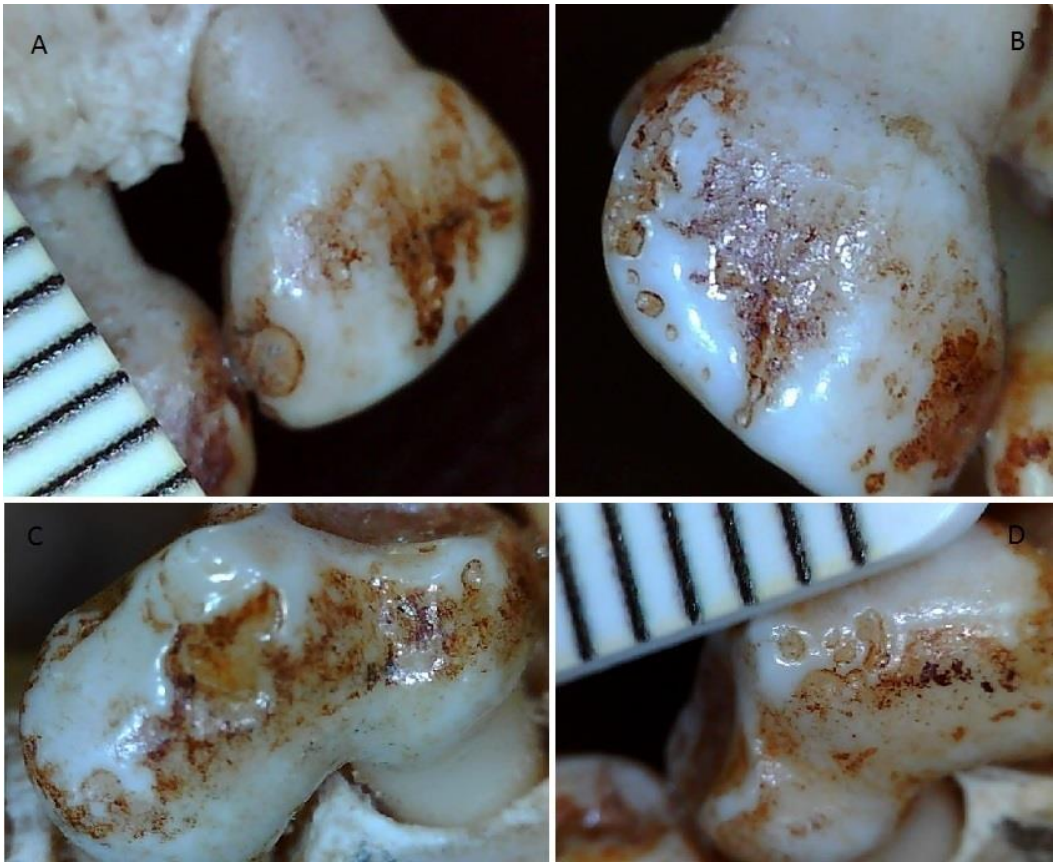

Figure 6.12. M 475, a male chimpanzee displaying non-symmetric localised/pitting hypoplasia on multiple deciduous teeth. A) Upper left lateral incisor; B) Upper right lateral incisor; C) Lower left first molar; D) Lower right first molar. All buccal view.

6.4.2. Paranthropus robustus

Clearly, *P. robustus* has extremely high levels of PEH, particularly on deciduous molars. However, due to the lack of known frequencies in many other hominin samples it is not yet known just how common such pitting is in hominins in general. That said it is clear from the results of this study, as well as comparisons with modern human studies, that the PEH rate found in *P. robustus* is remarkably high (Goodman et al., 1987; Ogilvie et al., 1989; Seow et al., 1992). Identical PEH defects can be seen on the posterior teeth of *P. robustus* specimens not included in this study, from Drimolen and Cooper's, as well as remarkably similar PEH defects on *P. boisei* posterior teeth (Figure 6.13; de Ruiter et al., 2009; Tobias, 1967). The frequency of these defects across sites and time suggests perhaps that the *Paranthropus* genus, in general, shared this tendency for PEH defects.

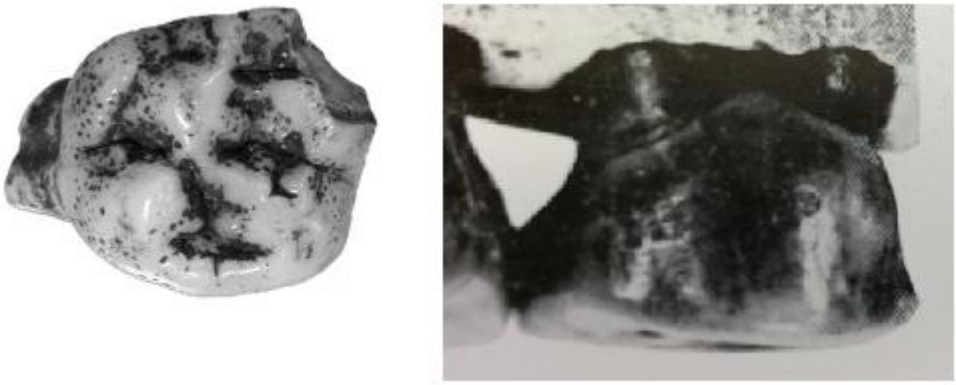

Figure 6.13. Pitting enamel hypoplasia. Left: *P. robustus* from Cooper's Cave (CD 1634; de Ruiter et al., 2009); and *P. boisei* (OH 5; Tobias, 1967).

The extant apes studied here do not have significant rates of PEH. Indeed it is extremely uncommon. In the extant primates that do have PEH, the defects do not usually look similar to those of *P. robustus*. For example, the most similar pitting in the chimpanzee sample is a female juvenile, M 556. This individual has PEH on the maxillary lateral incisors, lower lateral incisors and both sets of deciduous molars (Figure 6.14). Although the pitting in this individual does look similar to examples found in *P. robustus*, the anterior teeth are also affected. This is also the case for other primate examples. Therefore, this raises the question of why *P. robustus* does not also have affected deciduous anterior teeth. This fits with the hypothesis that perhaps the morphology of these teeth makes deciduous molars in particular prone to pitting. That said, in modern humans, differences in developmental timing between the different deciduous teeth mean it is not uncommon to show defects on molars and not anterior teeth. When the developmental timing for deciduous *Paranthropus* teeth is better understood it may be possible to speculate further on this issue.

An isolated Swartkrans tooth, SKX 1756, is not complete but has been attributed to the genus *Homo* (Berger et al., 2015). However, given the broken nature of this specimen, there is doubt over its phylogenetic placement (Grine, 2005). This deciduous molar shows PEH defects that are very similar to that in the *P. robustus* specimens (Figure 6.15). Such defects are not found on any of the other *Homo* specimens, perhaps suggesting this specimen may belong to *P. robustus*.

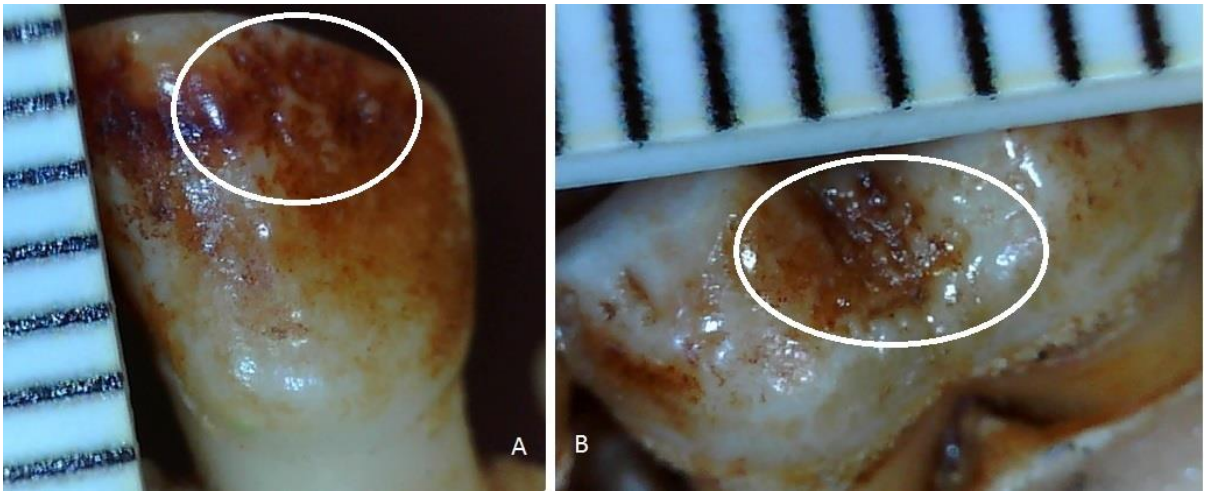

Figure 6.14. Pitting enamel hypoplasia on the deciduous dentition of a female chimpanzee (M 556). A) Left lower lateral incisor; B) Lower left second deciduous molar. Both buccal view.

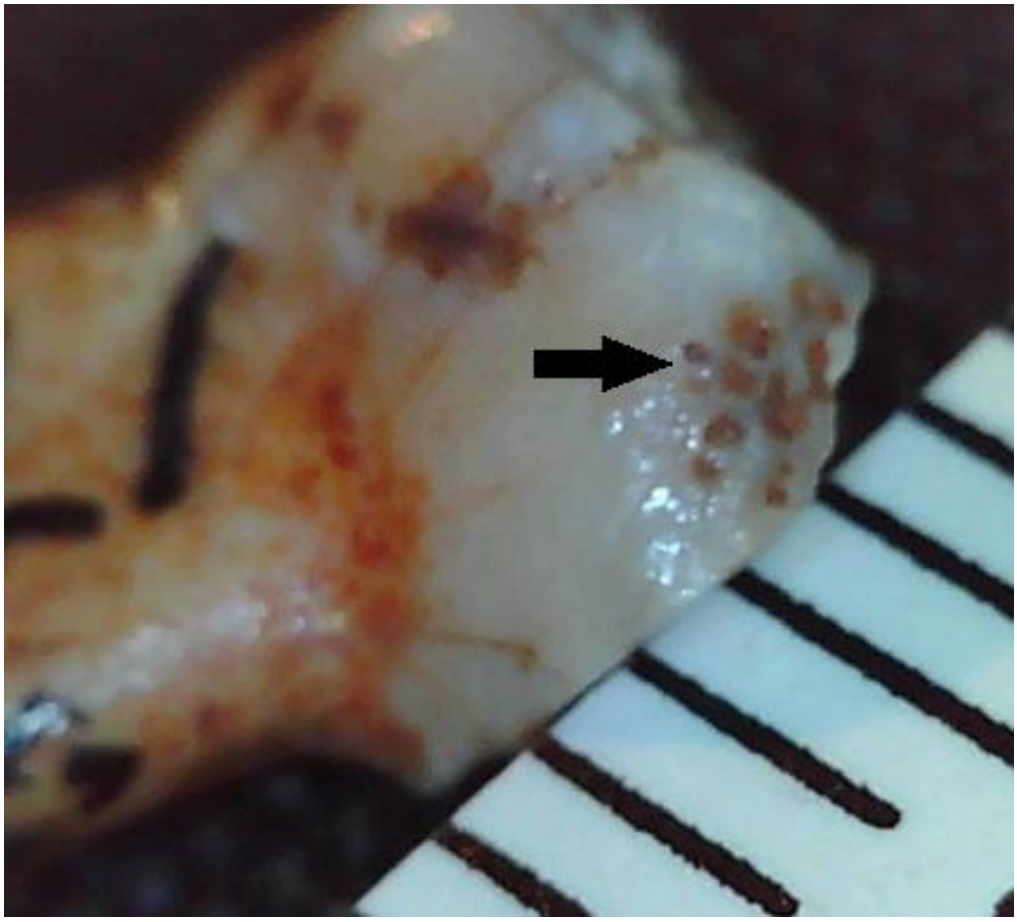

Figure 6.15. SKX 1756: fragmentary deciduous molar assigned to the genus *Homo*. Clear pitting enamel hypoplasia defects (arrow).

The PEH displayed on *P. robustus* molars do not resemble the hypoplastic defects described in many modern clinical studies of primary teeth, including those caused by premature birth, low birth weight, vitamin D deficiency, tuberous sclerosis, congenital syphilis, pseudohypoparathyroidism and epidermolysis bullosa (Aine et al., 2000; Croft et al., 1965; Gaul et al., 2015; Nikiforuk & Fraser, 1979, 1981; Pinhasi et al., 2006; Purvis et al., 1973; Radu & Soficaru, 2016; Seow et al., 1984; Stimmler et al., 1973; Wright et al., 1993). The majority of these conditions are associated with pitting that is usually irregular in shape and distribution. These conditions also tend to affect all teeth and not just molars, and are often associated with other types of dental defects.

PEH defects have also been associated with deficiencies or surpluses of certain compounds. For example, dental fluorosis can create pit like features on the crown of a teeth (Fejerskov et al., 1990). This can manifest in several ways but most commonly in white opaque lines, but the entire crown can have a chalky white appearance (Fejerskov et al., 1990; Xing et al., 2015). If the fluorosis is severe enough then once the tooth has erupted the outer enamel may start to fracture leaving pits or cracks. These pits have edges that are jagged or broken when viewed under a SEM (Thylstrup & Fejerskov, 1978). Another common occurrence with dental fluorosis, particular servere cases, is discolouration. The teeth in this sample do not show jagged or broken edges or discolouration of any of the teeth suggesting fluorosis is unlikely the cause. The fact that it is mainly posterior teeth affected also supports this conclusion. Other toxic compounds such as mercury can also create enamel defects if exposed to high quantities during dental development (Ioannou et al., 2015; Ogden, 2007; Radu & Soficaru, 2016). It is possible therefore that the pitting in *P. robustus* is caused by a specific environmental or dietary component, or lack thereof, although the issue of anterior teeth appearing to be unaffected again makes this hypothesis unlikely.

Ogden et al. (2007) highlight an example of a human sample with high rates of hypoplasia in the deciduous and permanent dentitions from 16th–18th Century London. Superficially some of these defects look similar to the pitting found on the *P. robustus* molars. However, the pitting appears less uniform, and there seems to be a mix of plane form and pitted defects across much of the crown in many teeth. Additionally, the cuspal morphology is affected in many of the teeth, which does not appear to be the case for the *P. robustus* examples.

It is perhaps surprising that out of the 12 permanent anterior teeth associated with posterior teeth with pitting, only two show enamel defects -- both in the form of LEH. Even if the developmental timing differs between the anterior and posterior teeth it is surprising individuals with pitting don't also show defects on anterior teeth. This is because illnesses and malnutrition can often last substantial periods of time and therefore if these defects are caused by a systemic disturbance it would be expected that anterior teeth would also be affected to a certain extent. Although the sample of associated teeth is small, it nonetheless adds further support to the idea that dental morphology, or genetic factors more broadly, are at least partially responsible for the high rate of PEH on posterior teeth of both dentitions.

The example in Hillson (2014) of PEH in a modern human deciduous first molar and associated plane form defects on the second deciduous molar and canine, is suggested as a case of 'neonatal dental hypoplasia', in which a stress at birth causes these severe looking defects. Therefore, a potential hypothesis would be that the *P. robustus* defects are caused at or around birth, perhaps by a specific issue such as an environmental or nutritional stress. If correct, this may have been facilitated by the fact the development of the deciduous molars tended to be at a crucial point of development, in that a pitted defect is more likely to form or be visible. However, there are a couple of issues with this hypothesis: 1) the lack of associated hypoplasia on other deciduous teeth, and 2) the fact that such pitting is also present in permanent molars. A neonatal line can be found in a wide variety of populations affecting the teeth forming at birth, in humans, this means the deciduous dentition and the first permanent molars. It is thought to be due to either a decrease in plasma calcium after birth (Norén et al., 1984; Smith & Avishai, 2005) or disturbances and stresses caused by the birth process itself (Guatelli-Steinberg, 2015; Whittaker & Richards, 1978). Although this hasn't been explored in many fossil hominin materials, if neonatal lines could be found in the deciduous and permanent molars of *P. robustus* teeth with pitting this would allow inferences into timing and perhaps therefore also aetiology. Unfortunately, the only *P. robustus* tooth for which these data are available does not show pitting (Smith et al., 2015). If the deciduous teeth formed PEH in utero this means the defects would relate to nutritional condition of the mother (Lovell & Whyte, 1999; Lukacs, 1992; Seow, 1990). However, it is unclear how many of the defects on deciduous molars of *P. robustus* formed in utero, so it cannot be determined whether the disruption that caused these defects occurred before or after birth.

It has been suggested that a common cause of prenatal enamel hypoplasia is linked to the mother having a calcium deficiency, caused by malnutrition or malabsorption (Lovell & Whyte, 1999). Similarly, defects that are postnatal in origin have also been linked to hypocalcaemia, often caused by insufficient calcium consumption or malabsorption. The defects displayed on the teeth of *P. robustus* are therefore consistent with calcium deficiency. Furthermore, the timing of the majority of the defects and their characteristics would fit with the conclusion that PEH in *P. robustus* is high in deciduous molars due to malabsorption or malnutrition which has led to these defects. That said, further examples in humans are needed to support this.

Superficially, the pitting defects in *P. robustus* resemble certain modern human cases of amelogenesis imperfecta. This set of genetic disorders affects one in every 700 to 14000 humans (Crawford et al., 2007; Sundell & Koch, 1984). Defects usually include scattered pits and plane form hypoplasia but often there is also abnormal colouration, thickness and density of the enamel (Aldred et al., 2003; Chamarthi et al., 2012; Schuurs, 2012; Wright, 1985). A type of amelogenesis imperfecta described as hypoplastic amelogenesis imperfecta (Mehta et al., 2013; Wright, 1993), most closely fits the description of defects in *P. robustus*, in that this variety commonly does not show colouration, thickness and density abnormalities of the enamel (Seow, 1992; Witkop, 1988; Witkop & Sauk, 1976). The pitting defects do in some instances look very similar to examples of this type of amelogenesis imperfecta (e.g., Figure 3 of Rushton, 1964; Ozdemir et al., 2005). However, the lack of defects associated with amelogenesis imperfecta on anterior teeth and the high frequencies of PEH only on deciduous molars suggests a genetic disease is unlikely, at least any type similar to that is found in modern humans today. The large time span involved, the scarcity of these conditions in modern humans, and the lack of other genetic conditions all support this conclusion. It is, however, worth noting that a recent study highlighted a particular genetic mutation that causes enamel defects to be present in posterior but not anterior primary teeth (Kim et al., 2016). Therefore amelogenesis imperfecta as the cause of PEH in *P. robustus*, although unlikely, cannot be ruled out completely.

That said, perhaps *P. robustus* had an increased chance of this type of genetic disorder relative to other hominins. Recent genomic research has begun to explore genetic loci involved in enamel formation, mostly with the aim of understanding formation, but also

for inferences into genetic conditions (Al-Hashimi et al., 2009; Hu et al., 2005; Hu & Yamakoshi, 2003; Ozdemir et al., 2005; Paine et al., 2001). One such locus, *enamelin*, has also been analysed for differences between primate species (Kelley & Swanson, 2008). They hypothesis, using evidence of adaptive *enamelin* evolutionary changes between species, that differences in enamel thickness between primate species is a result of these changes. Areas of this gene show signs of strong positive selection, potentially related to enamel thickness changes (Horvath et al., 2014). Mutations in this same gene are also responsible for the suite of enamel defects known as amelogenesis imperfecta (Crawford et al., 2007; Kelley & Swanson, 2008; Wang et al., 2015). Therefore, it may be possible that the evolution of extremely thick enamel in *Paranthropus* led to an increased chance of other genetic mutations, which often happens in genes subject to high levels of selection. Thus potentially including those that are associated with amelogenesis imperfecta. This hypothesis will be difficult to test. However, a larger sample of primates, incorporating genetic information and enamel thickness data, may show interesting results.

6.5. Conclusions

In sum, the exact cause of the pitting enamel hypoplasia found in *P. robustus* is as yet unknown, however by comparing frequencies and appearance with other species it has been possible to rule out causes and to suggest a few potential aetiologies. Thick enamel, developmental timing, genetic factors, calcium deficiency and acute stress during a crucial period of development are all possible factors. Given that there are substantially fewer defects on anterior teeth and defects are relatively uniform in shape and size on molars across individuals, at present, the hypothesis that fits the data best is a predisposition for molars, both deciduous and permeant, for this 'golf ball' like PEH. However, malnutrition due to a narrow diet or specific disorders such as calcium deficiency, may explain why so many individuals from this one species are affected. General ill health throughout development seems unlikely given the low number of LEH defects compared to other hominins and proportionally greater PEH defects than LEH.

Studies that have only looked at LEH in the South African hominin material have concluded that *A. africanus* may have been under more stress during development due to having higher rates of LEH than *P. robustus*. However, when PEH is included in analysis it is

clear that overall hypoplasia rates are actually greater in the latter species. If it is accepted, which most researchers do, that the majority of PEH is formed by similar disturbances to development as LEH then this would highlight that in fact the opposite may be true, with *P. robustus* experiencing more disturbances whilst teeth are forming. However, this depends on the cause for such high rates of PEH in *P. robustus*.

Localised hypoplasia does not seem to correlate with other types of hypoplasia. Additionally, the sexes seem to be equally affected in the species studied. It is also rare for defects to be displayed on anti-meres. These three observations add further support to the idea that these defects are not systemic in origin and relate to some sort of localised trauma during tooth development.

Chapter 7: Caries

7.1. Introduction

It is often suggested that caries is a modern disease and is scarce or absent in past hominin populations, usually justified by inferring dietary or oral bacterial differences between present day and ancient human samples (Armelagos & Cohen, 1984; Brothwell, 1963; Guatelli-Steinberg, 2016; Hildebolt & Molnar, 1991; Lanfranco & Eggers, 2012; Tillier et al., 1995). However, evidence for the presence of carious lesions in a variety of non-agricultural hominin groups is growing (e.g., Arnaud et al., 2016; Grine et al., 1990; Humphrey et al., 2014; Lacy, 2014; Lanfranco & Eggers, 2012; Trinkaus et al., 2000). In light of this evidence, the South African hominin material was reanalysed, and *H. naledi* recorded for the first time, for the presence of caries; comparisons were then made among all fossil hominins, along with extant primates and recent human samples.

The frequency and location of caries on the dentition vary with diet. Lesions form when specific bacteria demineralize dental tissue through the release of acids as they metabolise sugars and starches (Byun et al., 2004; Larsen et al., 1991). Many different bacteria can be involved, including *Streptococcus mutans* and *Streptococcus sobrinus* (Nishikawara et al., 2007). The genome of some of these bacteria have been sequenced and one in particular, *S. mutans*, has evolved quickly in the last few thousand years, potentially in response to human population growth and agriculture (Cornejo et al., 2013). The acids released form active lesions because the oral pH is lowered over extensive periods (Gussy et al., 2006). Some foods are more cariogenic than others. Those containing high levels of refined carbohydrates and sugars are particularly virulent (Clarkson et al., 1987; Prowse et al., 2008; Rohnbogner & Lewis, 2016). Tough and fibrous foods are linked with low rates of caries since they tend to create a more alkaline oral environment due to high levels of saliva production (Moynihan, 2000; Prowse et al., 2008; Rohnbogner & Lewis, 2016). Meat and dairy products have also been associated with low caries frequency (Moynihan, 2000; Novak, 2015).

Environmental and behavioural influences are also important to consider when researching caries prevalence. The clearest example is that of moderate levels of fluoride in drinking water, which decrease the likelihood of carious lesions developing (Kotecha et al.,

2012; Slade et al., 2013). There may also be protective properties against caries in certain plant species (Moynihan, 2000). Therefore, if certain plant species with these properties are consumed, a population may show surprisingly low levels of carious lesions, even if carbohydrate consumption is high. Many fruits, as well as honey, are cariogenic but the relationship is not always straightforward (Novak, 2015). Nuts and seeds can be highly cariogenic (e.g., Humphrey et al., 2014), although caries rates vary considerably depending on the species involved. The teeth, dental tissues, and crown positions that are most affected in a population can give further insight into the diet, bacteria present and food processing behaviours (Bignozzi et al., 2014; Kelley et al., 1991; Meinl et al., 2010; Novak, 2015; Shen et al., 2004; Takahashi & Nyvad, 2016).

A wide variety of living and fossil species show evidence of caries, including dinosaurs, fishes, bats, bears, and primates (Arnaud et al., 2016; Humphrey et al., 2014; Kear, 2001; Kemp, 2003; Lacy, 2014; Lanfranco & Eggers, 2012; Lovell, 1990; Miles & Grigson, 2003; Palamra et al., 1981; Sala et al., 2007; Trinkaus et al., 2000). Eight carious lesions have already been recorded in the South African hominin fossils, including two in a mandible belonging to an early *Homo* individual, SK 15 (Clement, 1956). The rest are attributed to *P. robustus*, with three on SKX 5023 (Grine et al., 1990), two lesions on SK 55 (Robinson, 1952), and one on SK 13/14 (Robinson, 1952).

7.2. Materials and Methods

Only complete teeth are included in the analysis, with postmortem-damaged teeth excluded. Each tooth was examined macroscopically under good lighting with a 10x hand lens used for clarifying lesions. The frequency of caries is calculated as follows:

[Total number of teeth with caries/total number of teeth observed]*100

Once a carious lesion progressed enough for a cavity to form it is relatively difficult to confuse with postmortem damage. A carious lesion was recorded when there is a clear cavitation; colour changes alone were not recorded. The severity and position of lesions on a tooth were also recorded. Caries severity was scored on a scale of 1 to 4 following Connell and Rauxloh (2003), with (1) enamel destruction only; (2) involvement of dentine but pulp chamber not exposed; (3) destruction of dentine with the pulp chamber exposed; (4) gross destruction with

the crown mostly destroyed. Lesion location is also recorded (distal, buccal, occlusal, lingual, mesial and gross).

The interaction between caries and other dental pathologies is often complex. It has been suggested that because caries can lead to Antemortem Tooth Loss (ATML), correction methods need to be implemented (e.g., Duyar & Erdal, 2003; Kelley et al., 1991; Lukacs, 1995). However, these caries correction methods are not appropriate to implement in this research. Although it is true that the vast majority of missing (i.e., extracted) teeth today are due to caries, in many past populations this would not have been the case. Severe attrition and fractures that exposed the pulp and periodontal disease are all other possible contributors. In particular, given the high rates of wear in fossil hominin specimens, it is likely that most cases of ATML resulted from attrition, not caries. Therefore, following Meinl et al. (2010) and Larsen et al. (1991) no corrective methods were implemented; instead, AMTL frequencies are displayed separately as an independent factor. By not including correction methods direct comparisons with other populations can be made. Additionally, maxilla and mandible fragments are so rare in the fossil record that to include ATML data would have little effect on overall frequencies (Chapter 9).

Micro-CT scans of particular teeth are included in this study to also help clarify if a cavity is carious. Micro-CT scans can differentiate between normal enamel and dentine and areas affected by caries, due to the lower density of areas affected with this pathology (Neves et al., 2010). Using such techniques can clearly make visible the extent of a lesion, even if the cavity on the surface is ambiguous (McErlain et al., 2004). Therefore, if clear areas of demineralised enamel can be seen below the cavity, then it is recorded as caries. As noted in Chapter 8, dental attrition is marked according to Smith (1984) for anterior teeth on a scale of 1 to 8, and Scott (1979) for molars on a scale of 1 to 10. To compare caries rates between chimpanzee males and females (below) a chi-square test of homogeneity with significance set at the 0.05 alpha level was used.

7.3. Results

In addition to the five carious teeth already described in the literature, an additional seven have been added here (Table 7.1). From the seven newly identified teeth, five are from *P. robustus* specimens and two from *H. naledi*. Therefore, there is now a minimum of 16

carious lesions across the available South African fossil hominin material (Table 7.2). The five new *P. robustus* teeth come from three individuals, one of which is thought to belong to the same individual that already had carious lesions described in other teeth (SKX 3601 and SKX 5023; Grine et al., 1990). Therefore, there are three *P. robustus* individuals, one *H. naledi* individual, and one *early Homo* individual that exhibit caries. No carious lesions were found in deciduous teeth, or any tooth belonging to *A. africanus* and *A. sediba*. In sum, there are 16 carious lesions on 12 teeth belonging to five individuals for the South African hominin material (Table 7.2).

Table 7.1. Caries frequency for each species study.

Species	Observable teeth	Carious teeth	% caries
Early *Homo*	44	2	4.55
P. robustus	318	8	2.52
H. naledi	147	2	1.36
A. sediba	16	0	0.00
A. africanus	328	0	0.00
Baboons	760	1	0.13
Chimpanzees	1991	165	8.29
Gorillas	1518	20	1.32

Table 7.2. Hominin specimens with caries. Tooth: first letter, L (left), R (right); second letter, L (lower), U (upper); M1, first molar, P2, second premolar.

Species	Specimen	Tooth	Position	Severity	Lesion #	Described
P. robustus	SK 23	LL M1	Occlusal	1	1	This study
P. robustus	SK 23	RL P2	Occlusal	1	1	This study
P. robustus	SKX 3601	LU M1	Mesial	1	1	This study
P. robustus	SKX 5023	RL M1	Mesial/Distal	2	3	Grine et al. (1990)
P. robustus	SK 55	LU M1	Buccal	1	2	Robinson (1952
P. robustus	SK 13/14	LU M2	Occlusal	2	2	Robinson (1952)
Early *Homo*	SK 15	LL M1	Mesial	2	1	Clement (1956)
Early *Homo*	SK 15	RL M2	Mesial	2	1	Clement (1956)
H. naledi	UW 101-001	RL P2	Distal	2	1	This study
H. naledi	UW 101-001	RL M1	Mesial	2	1	This study
P. robustus	SKW 5	RL M1	Occlusal	1	1	This study
P. robustus	SKW 5	LL M1	Occlusal	1	1	This study

Other potential carious lesions are present in the sample, particularly in *H. naledi*, including: UW 101-525, UW 101-1277 (second molar), UW 101-010, UW 101-516, UW 101-001 (third molar), UW 101-445, UW 101-809, and SKW 33. Micro-CT scans of these teeth are needed to help substantiate the lesions. Additionally, although SKX 3601 is recorded here as having caries, a CT scan would be useful to help rule out post-mortem damage completely.

7.3.1. SK 23

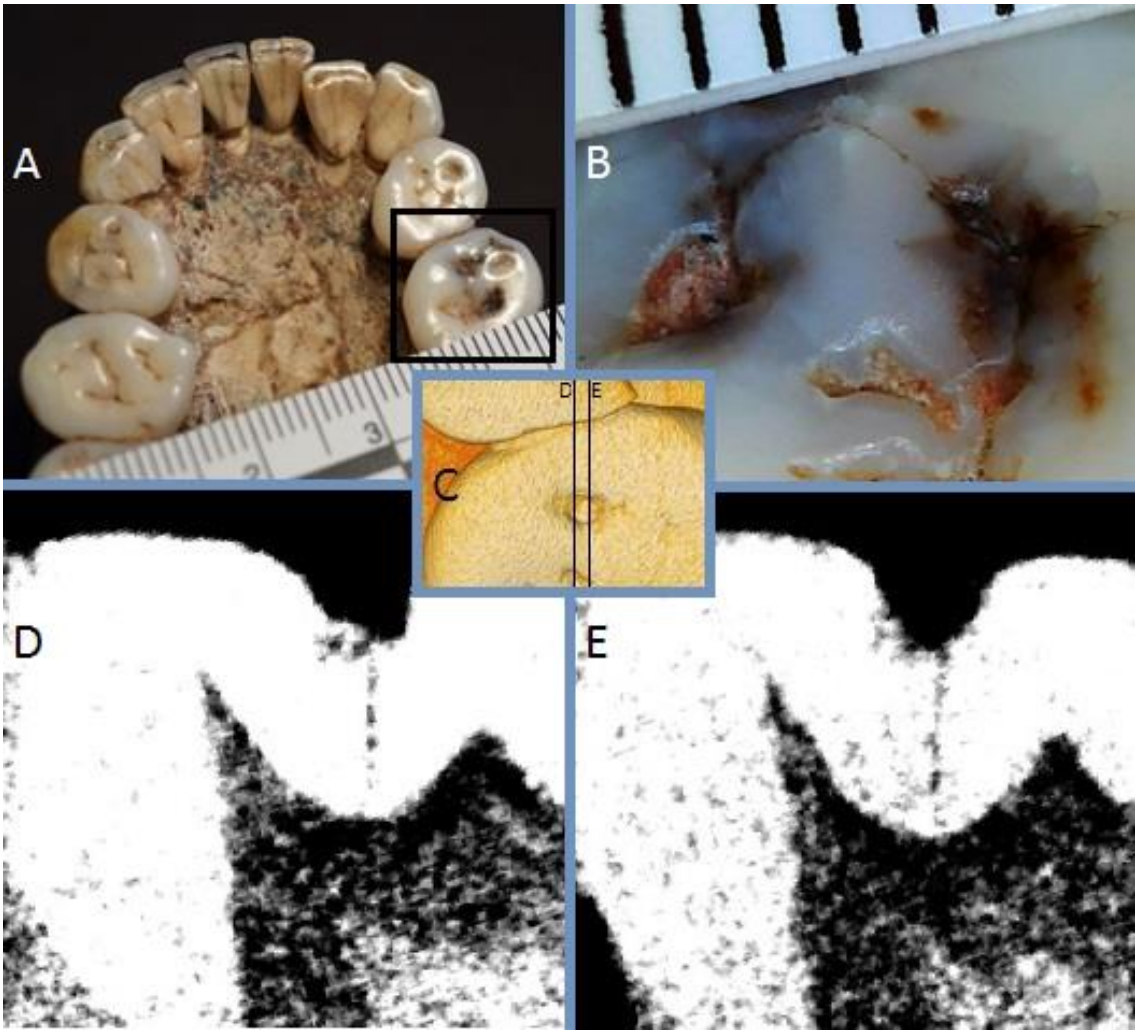

Figure 7.1. Caries in SK 23 (*P. robustus*). A) Occlusal view of mandibular teeth, with the right second premolar highlighted; B) Close-up of the occlusal surface of the right second premolar; C) CT reconstruction with the position of the two slices highlighted; D) CT slice toward the lingual part of the cavity; E) CT slice toward the buccal portion of the cavity.

Several teeth belonging to SK 23 have characteristics of caries on their occlusal surface. In particular, the left first molar and right second premolar have larger and darker coloured fissures on their occlusal surface than do their antimeres (Figure 7.1a). However, due to post-

mortem matrix in these depressions (Figure 7.1b), it is difficult to confirm whether these are carious. A Micro-CT scan of these teeth supports the conclusion that this individual had caries, at least on the right second premolar. Enamel under the depression on the occlusal surface of the right second premolar appears to have patches of less dense material, and a patchy track of less dense enamel extends towards the Enamel-Dentine Junction (EDJ; Figure 7.1d,e). This demineralised enamel near the occlusal surface fits a diagnosis of occlusal caries; the line towards the EDJ is likely also carious in nature, though it may be a crack that formed due to the weakening of tissue by caries. Therefore, the difference compared with its antimere, large size of the depressions, colouration changes, and lower density enamel underneath, are all highly suggestive that a carious lesion had been active in this occlusal fissure.

7.3.2. UW 101-001

Carious lesions on *H. naledi* specimen UW 101-001 are the most severe of all South African hominins (Figure 7.2). They could be severity grade three lesions, however due to sediment in the cavity detailed exploration is not possible and therefore the minimum severity of two is recorded. CT-scans of these teeth could provide further information. However, it is clear that these lesions must have been active for several years since they spread deep into the dentine. It is interesting that no reduction of crown wear is evident on this side of the mouth, so these lesions may not have affected normal mastication.

Figure 7.2. Carious lesions on the mandibular right second premolar (distal) and first molar (mesial). *Homo naledi* (UW 101-001).

7.3.3. SKW 5

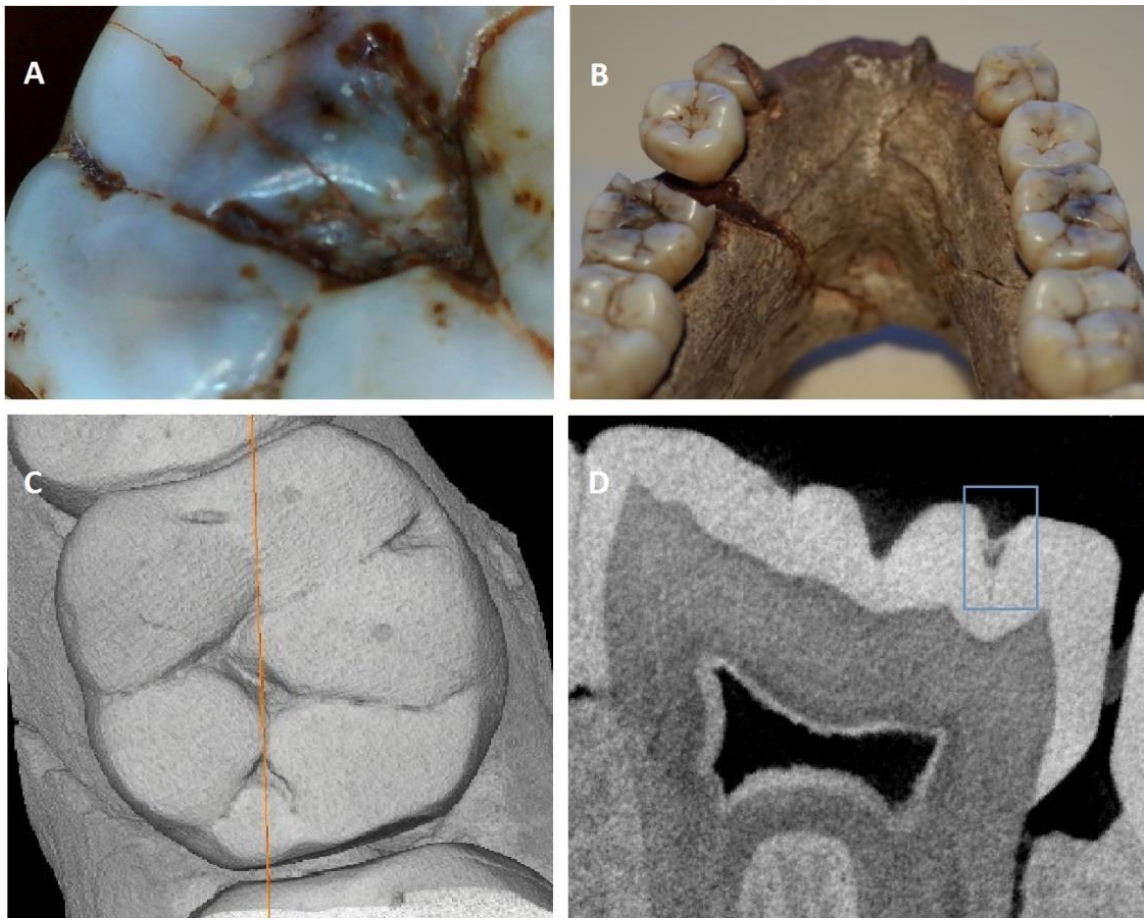

Figure 7.3. Caries on the occlusal surface of SKW 5 (*P. robustus*). A) Occlusal view of the right mandibular first molar; B) mandible displaying the two first molars with likely caries; C) CT reconstruction of the right first molar showing the position of the slice in D; D) CT slice of the right first molar, highlighting the area of demineralised enamel (blue square).

The first molars of SKW 5 (*P. robustus*) appear to have occlusal caries, based on the depth, colouration and pattern within the fissures (Figure 7.3a,b). When a Micro-CT slice of this area is viewed, demineralisation is evident deep into the enamel (Figure 7.3d). However, the low quality of available CT images means that confirmation of the caries severity is needed. Pitting enamel hypoplasia is visible in the adjacent teeth, suggesting this condition may have facilitated caries formation.

7.3.4. SK15

In their book on radiographs of hominin specimens, Skinner and Sperber (1982) make an interesting observation of one of the early *Homo* specimens, SK 15. Substantial interproximal wear is visible relative to little occlusal wear. They do not provide a suggestion for the cause, but do suggest that future research may give insight. They also note interproximal caries in this specimen but do not suggest a link between these two phenomena. Figure 7.4 shows interproximal caries on the right second molar in photo A, and photo B highlights the extent of the interproximal wear.

Figure 7.4. SK15 (early *Homo*). A) right mandibular second molar, mesial caries; B) interproximal and occlusal wear.

7.3.5. Extant primates

The main difference observed between the extant primate sample and hominins are the teeth affected by caries. All carious lesions in the hominin sample are found on posterior teeth, whereas 70% of caries affects anterior teeth in the primates. Particularly common in both gorillas and chimpanzee is the occurrence of carious lesions on interproximal surfaces of the incisors. Significantly more caries are present in the dentitions of female chimpanzees than males (X^2= 20.890, 1 df, p= 0.0001), with five times the number of teeth affected. This difference does not appear to relate to time in occlusion, based on tooth wear (Table 7.3). Although the female sample is composed of individuals with, on average, more worn teeth once wear is accounted for, it is clear that this relationship remains stable (Table 7.3). Females with low and medium wear on their teeth (combined wear score of under 64 for all four first

molars; Scott, 1979) still have significantly more carious teeth, with around five times the caries rate as in males.

Table 7.3. Caries frequencies for male and female chimpanzees. Displayed for all teeth, unworn/little-worn teeth removed, and with heavily worn teeth excluded. Is: incisors; Cs: canines; PMs: premolars.

Sample	Females	Males
Totals (%)		
All teeth	9.30	1.80
All teeth except wear score 1	10.15	2.35
Teeth with medium/low wear*	9.78	2.20
Individuals with caries	44.90	8.33
All Teeth		
Total teeth	1301	334
Carious teeth	121	6
Mean Is, Cs and PMs wear**	3.94	2.61
Mean Molar wear**	4.05	2.70
% carious teeth	9.30	1.80
Wear score 1 taken out		
Total teeth	1192	255
Carious teeth	121	6
Mean Is, Cs and PMs wear**	4.31	3.51
Mean Molar wear**	4.19	2.91
% caries teeth	10.15	2.35
Medium to low wear*		
Total teeth	511	227
Carious teeth	50	5
Mean Is, Cs and PMs wear**	3.52	3.59
Mean Molar wear**	3.28	2.82
% carious teeth	9.78	2.20

*Individuals with a combined wear score of under 64 for all four first molars. Teeth with a wear score of 1 are excluded

*&**Molar wear is calculated using Scott (1979) and all other teeth following Smith (1984)

7.4. Discussion

Carious teeth are more likely to be lost or damaged post-mortem because they are more fragile than non-carious teeth. In support of this statement, all newly reported carious teeth in this study were retained in a jaw. Therefore, it seems likely that caries would have been more common in the fossil hominins than the results of this study suggest.

Recent research has highlighted that carious lesions may form more frequently on different dental tissues, or certain positions on a tooth crown, due to the presence of certain bacteria, pathologies, and dietary components (Bignozzi et al., 2014; Meinl et al., 2010; Novak, 2015; Takahashi & Nyvad, 2016). However, all carious lesions ultimately share an aetiology that is based on the presence of certain cariogenic bacteria and fermentable carbohydrates (Clarkson et al., 1987). Clearly, therefore, cariogenic bacteria were prevalent in fossil hominins.

The frequency of caries on different tooth surfaces has predominantly been linked to the extent and speed of attrition on the occlusal surface. High rates of occlusal caries are associated with low attrition, whereas high levels of interproximal lesions are linked with high attrition (Hillson et al., 2008). However, high rates of interproximal caries may also be related to levels of calculus, in which plaque buildup in these areas may facilitate lesion formation (Tomczyk et al., 2013). Calculus involvement appears to be part of the reason for the high interproximal caries rates in the medieval Gloucester sample. However, there does not seem to be any evidence for calculus associated with caries in the South African hominins, although post-mortem loss may be partly responsible. For example, taphonomic processes may remove calculus deposits, as well as handling and excavation (Chapter 2).

In modern human agricultural groups maxillary teeth tend to display higher rates of caries than mandibular teeth (Caglar et al., 2007; Esclassan et al., 2009; Lunt, 1974; Novak, 2015). It is also more common for caries to be found on posterior teeth than anterior (Novak, 2015; Slaus et al., 1997; Varrela, 1991; Vodanović et al., 2005; Watt et al., 1997). Similarly, the positions most affected are interproximal and occlusal surfaces (Esclassan et al., 2009; Caglar et al., 2007; Slaus et al., 2011; Srejic, 2001; Varrela, 1991; Vodanovic et al., 2012). These differences in location likely reflect the greater complexity of posterior teeth and their larger size, in which more susceptible areas are present for caries to form (Hillson, 2001, 1996). For previously reported carious lesions in fossil hominins, the majority are located on interproximal areas of the tooth crown rather than root surfaces. This is likely due to periodontal attachment loss being uncommon in earlier hominins, which in most cases is a prerequisite for root caries (Bignozzi et al., 2014; Stamm et al., 1990). However, root caries has been shown to be formed commonly by particular bacteria in modern humans (Shen et al., 2005) and may also develop in different acidic conditions than enamel caries (Shen et al., 2004). Therefore the oral environment may also have been the key factor. The fact that fossil

hominins tend to display interproximal carious lesions likely reflects the heavy occlusal wear in these populations, in which fissures and pits on the surface are quickly worn away with wear progressing too fast for lesions to form. The extremely low levels of occlusal caries in the extant primate samples also supports the conclusion that occlusal caries is rare in fossil hominins due to high rates of wear. That said, occlusal caries in *P. robsutus* suggest that tooth wear was not rapid enough to prevent lesions forming, perhaps hinting at a high level of cariogenic food consumption.

Many hominin samples within the last 50,000 years show caries frequencies of around the same or below some of the species studied in this thesis (Figure 7.5). Although Figure 7.5 only contains a handful of samples, it highlights the relatively consistent carious frequency in hominins of around one to five percent. With the advent of agriculture, this figure varies much more dramatically, and some populations had vastly greater rates. A diet high in marine foods and terrestrial meat seems to be the most common reason used to explain low frequencies in recent hominin groups (Kelley et al., 1991; Lacy, 2014; Larsen et al., 1991). Rather than a steady increase, as has been suggested, the rate is rather stable over the last two million years, with only an increase in some agricultural groups. Therefore, the commonly perceived notion that caries is a new disease, in which only recent populations of the genus *Homo* are affected, may be misleading (Armelagos & Cohen, 1984; Brothwell, 1963; Guatelli-Steinberg, 2016; Hildebolt & Molnar, 1991; Lanfranco & Eggers, 2012; Tillier et al., 1995).

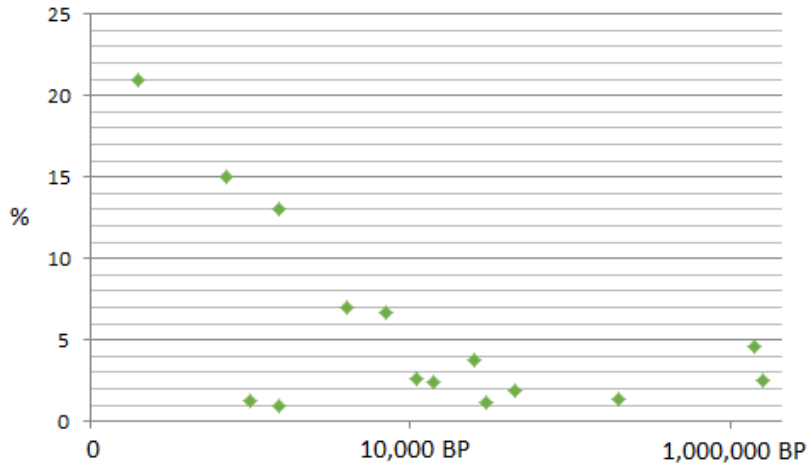

Figure 7.5. Per tooth caries frequencies (%) for various hominin groups, including *H. naledi*, *P. robustus* and early *Homo* (comparative data: Costa, 1980; Esclassan et al., 2009; Kelley et al., 1991; Lacy, 2014; Lanfranco & Eggers, 2012; Larsen et al., 1991; Mant & Roberts, 2015; Meiklejohn et al., 1988; Smith, 1972; Walker et al., 2011). Log-transformed X-axis.

Enamel hypoplasia may influence the formation of carious lesions. Defects may act as a site in which lesions can develop, but also it may speed up the progression of a lesion, as the enamel may be more vulnerable to acid solubility (Hong et al., 2009; Rohnbogner & Lewis, 2016). Therefore, enamel hypoplasia seems likely to be associated with caries in *P. robustus* including specimens SK 55, SK 13/14 and SKW 5. Other pathologies and wear can also create an environment in which caries is more likely to form. For example, caries may develop as a response to unusual occlusion (Calcagno & Gibson, 1991). In populations or species with a moderate amount of caries, presumably, such occurrences will be proportionally more likely to be the cause of the cavity. A good example comes from the lower right third molar of a chimpanzee (M 158). This third molar was still in occlusion at the time of death, making it clear that severe caries on the buccal and occlusal surfaces was caused by the angle it protrudes from the jaw (Figure 7.6). If this tooth had been found isolated, such an interpretation might have been less clear.

Figure 7.6. Chimpanzee (M 158), the right side of the mandible. Occlusal/buccal caries on the third molar (arrow).

An explanation for the high caries rate in *P. robustus* is required. The fact that these individuals have relatively high levels of wear means that caries would be expected to be low, especially on occlusal surfaces (Maat & Van der Velde, 1987; Moynihan, 2000). Therefore,

cariogenic foods may have frequently been consumed, i.e. a similar proportion of such cariogenic foods in the diet of a population without high occlusal wear would likely display much higher rates of caries. The most likely explanation for the high rate of caries in *P. robustus* is the consumption of cariogenic fruits or vegetation matter, although another possible component may be the consumption of honey (Moynihan, 2000). Pitting enamel hypoplasia has also clearly inflated the caries rate by yielding suitable sites for lesions to form. However, cariogenic food and bacteria would have still been a necessary prerequisite for caries formation.

The lack of caries in baboons and *A. africanus* potentially requires a dietary explanation too. It seems unlikely a lack of carious bacteria is on its own responsible, particularly given the frequent occurrence of caries in other hominins and extant primates. As mentioned, some foods can actively limit or prevent caries formation and are associated with low levels of lesions. In particular, tough, hard and fibrous foods can all create a less acidic oral environment due to high concentrations of saliva circulation (Moynihan, 2000; Prowse et al., 2008; Rohnbogner & Lewis, 2016). Meat is also generally thought to be associated with low rates of caries (Novak, 2015). Grit incorporated into the diet can create heavy wear and may therefore also mean caries lesions are less likely to form. Baboons masticate a significant amount of grit but also have an omnivorous diet containing a lot of tough foods such as meat, leaves and roots, likely explaining the lack of caries (Duray, 1992; Moynihan, 2000; Nystrom et al., 2004). Therefore, perhaps *A. africanus* had a similar omnivorous diet to baboons. Occlusal wear and dental chipping frequencies for these two populations are also very similar (Chapter 5). Such a similarity also fits with diverse results for *A. africanus* in isotopic analysis, microwear, and biomechanical analysis, which suggest a varied diet with frequent consumption of tough foods (Nystrom, 2004; Scott, 2005; Sponheimer et al., 2005, 2013; Sponheimer & Lee-Thorp, 1999; Strait et al., 2009; Van Der Merwe et al., 2003).

7.4.1. Extant primates

The unusual interproximal cavities on the incisors of chimpanzees have been noted before, with Kilgore (1989) finding deep depressions in two of the older individuals and colour changes on the interproximal areas in juveniles. Radiographs of the older individuals showed

these voids extended deep into the tooth. Unexpectedly, given this set of observations, they concluded that the cause was severe enamel attrition from stripping foods (Kilgore, 1989). It is not stated how this process would create such narrow, uniform, deep depressions in an interproximal area, or why some individuals only show colouration changes. The descriptions and photos in their Figure 7 (Kilgore, 1989) fit precisely with the observation made in this study. Similarly, Dean et al. (1992) note a few root caries in chimpanzees and no caries in gorillas, for the same sample as this study (Powell Cotton Museum). Interproximal caries on incisors of both species were not recorded, presumably because these lesions were considered non-carious. However, confusingly, earlier research did report these interproximal cavities and recording their occurrence as caries (Coyler, 1936; Schults, 1956), with a longitudinal thin section used as confirmation (Miles & Grigson, 2003). These studies found similarly high rates of caries in chimpanzees to this research, with incisors found to be almost exclusively affected (Coyler, 1936; Schults, 1956). The results of this project support these earlier studies, as the lesions are clearly carious in nature.

The high rate of caries on the anterior teeth of chimpanzees, and to lesser extent gorillas, at first seems unusual, particularly compared with hominins. However, an interesting analogue can be found in modern humans that do not use their posterior teeth. A medieval individual from Poulton (SK 705) has many small (severity 1-2) caries lesions on the interproximal areas of anterior teeth. These anterior teeth are also heavily worn. The reason for these carious lesions is due to many posterior teeth being removed, so those that are left have no counterpart (i.e., isomere) with which to occlude. The individual was therefore almost exclusively using their anterior teeth for processing and masticating food. Therefore, caries found predominately on anterior teeth of chimpanzees may also relate to the ways in which they process food. This hypothesis is supported by behavioural observations of chimpanzees, where they tend to use their muscular lips along with anterior teeth to process fruits (Galdikas, 1982; Ungar, 1994). Such processing may create an acidic microenvironment in the front of the mouth to promote caries development. Clearly, it is mainly females that are affected by this behaviour, which may suggest the processing of foods differs notably between the sexes.

7.5. Conclusion

The results of this chapter show distinct species differences, which likely reflect dietary and processing differences among groups. Caries is not as uncommon as previously thought in earlier members of the hominin lineage. In fact, the fossil hominins in this study had rates similar and, in some cases, higher than modern human groups both pre-and post-agriculture. There are however differences within the hominin sample, most notably the lack of carious teeth in the largest sample, *A. africanus*. This absence of caries may reflect a varied diet with high levels of tough, non-cariogenic foods. Once again *P. robustus* and *A. africanus* have substantial difference results, highlighting probable dietary differences. Given the number of carious teeth of *P. robustus* that have now been documented, a diet containing high amounts of cariogenic fruit/vegetation may be possible. Caries were previously reported in chimpanzees; however, the frequencies have rarely been compared with those in hominins. Chimpanzees have extremely high rates of caries, though not uniform, among tooth types or individuals. The anterior teeth of females have the most caries in this species, which may suggest that food processing of cariogenic foods, such as fruit, is the cause.

Chapter 8: Macro-wear

8.1. Introduction

Wear can be defined as loss of crown enamel and the underlying dentine that is not caused directly by a pathology or defect. It is often split into three broad categories: attrition, abrasion and erosion (Burnett et al., 2013; Grimoud et al., 2012; Kaidonis, 2008). Attrition occurs through the occlusal surfaces of teeth contacting each other, abrasion when other materials are introduced into the mouth, and erosion by acidic removal of dental tissue (Burnett et al., 2013; Deter, 2009; Fiorenza et al., 2011; Smith, 1984). Attrition commonly occurs during mastication but can also be non-masticatory in nature, caused by bruxism. In most circumstances attrition is responsible for the majority of tooth wear (Deter, 2009). Abrasion is most commonly studied with reference to cultural wear grooves or notches, caused by the repeated placement of an item in a specific place in the dentition. Examples include tooth pick grooves in Neanderthals and tooth brushing and cultural tool use in modern humans (Bouchneb & Maureille, 2004; Frayer & Russell, 1987; Hlusko, 2003; Lozano et al., 2013; Shellis & Addy, 2014; Turner & Cacciatore, 1998; Ubelaker et al., 1969; Ungar et al., 2001). Dental erosion is caused by chemical dissolution of dental tissue by acids, and is predominantly caused by specific dietary items or illnesses (Grippo et al., 2004; Indriati & Buikstra, 2001; Oginni et al., 2003; Ritter et al., 2009; Robb et al., 1991; Zero, 1996).

Most important in this research is attrition and abrasion, which provide in-depth detail on an individual's diet and cultural practices (Morse et al., 2013; Shykoluk & Lovell, 2010). In most cases, wear should not be seen as a pathology because it is a normal part of the masticatory process (Carranza et al., 2004; Dirks & Bowman, 2007; Smith, 1991; Williams & Woodhead, 1986). Indeed, occlusal wear in many species is crucial in allowing an efficient masticatory cycle (Ungar & M'Kirera, 2003; Ungar & Williamson, 2000). For example, in certain species wear can adapt the occlusal morphology of the teeth in a dentition so that foods can be processed consistently during the lifetime of an individual (Ungar, 2015).

The wear profile between maxillary and mandibular teeth is formed by the circular motion of the mandible, which contacts the maxillary teeth or dietary items (Grimoud et al., 2012; Hillson, 1996). The severity and pattern of the wear, such as wear direction and teeth

most affected, depends directly on the foods consumed (Lanyon & Sanson, 1986; Lavelle, 1970; Raupp, 1985; Veiberg et al., 2007). It is suggested that hard, tough, and fibrous foods prevent efficient lateral movement of the mandible, instead of maximising sagittal movement that produces horizontal occlusal wear (Fiorenza et al., 2015; Grimoud & Gibbon, 2017). Studying the direction and pattern of wear can, therefore, provide insights into diet, although comparative data is still uncommon in the literature (e.g., Brabant, 1966; Esclassan et al., 2009; Grimoud et al., 2012; Molnar, 1971; Smith, 1984). Additionally, which teeth are most affected by wear can provide information on food processing behaviours (Fiorenza et al., 2015; Hillson, 1996).

Many fossil hominins and hunter-gatherers show a relatively flat occlusal plane (Kaifu et al., 2003; Molnar et al., 1972). The softer diet of agriculturalists creates different wear patterns, with Smith (1984) showing that oblique wear is common. That said, recent research suggests that hunter-gatherer groups and agriculturalists have high variability in wear patterns (Grimoud & Gibbon, 2017; Kaidonis, 2008). At present, it may not be possible to go further than to say that oblique wear may represent a broad, mixed, non-specialised diet in hominins (Eshed et al., 2006; Lev Tov et al., 2003), and if accompanied by low overall wear a diet consisting of soft foods is likely.

Wear has been studied in a variety of primates and is particularly well researched in humans (e.g., Bramblett, 1967; Cuozzo & Sauther, 2006; Deter, 2009; Elgart, 2010; Fiorenza et al., 2015; Grimoud et al., 2017; Janis, 1984; Morse et al., 2013; Teaford, 1982, 1983; Veiberg et al., 2007). Fossil hominins have also been well-studied (Clement & Hillson, 2013; Estebaranz et al., 2009; Fiorenza et al., 2011; Grine, 1986; Tobias, 1967; Ungar et al., 2001; Ungar & Grine, 1991). It was suggested by Robinson (1956) that differences in occlusal wear direction in the mandibular molars of *P. robustus* and *A. africanus*, with more on the lingual side on the former and buccal on the latter, is caused by an increase in arch width in *P. robustus*. This adaptation has been suggested to be a consequence of evolving large posterior teeth in *P. robustus* (Cachel, 1975). Since then other researchers have studied wear patterns in the South African hominin material (e.g., Grine, 1981; Wallace, 1975; Wolpoff, 1973, 1975). Taken as a whole it is evident there is no consensus on differences in both the severity and pattern of attrition (Grine, 1986). That said, except for Wolpoff, all researchers note that

Paranthropus teeth tend to display more even wear between buccal and lingual molar cusps than those of *A. africanus*.

This relationship is explored by comparing how the direction of wear in the maxillary and mandible molars differs between species of hominins and extant primates. Comparisons between tooth types are also made as this offers insights into behaviour and diet. For example, populations that tend to use anterior teeth regularly for processing foods, or as tools, tend to have proportionally more wear on anterior teeth than posterior teeth (Liu et al., 2010; Molnar, 1972, 2008). Therefore, primates that tend to process foods with anterior teeth before consumption should be expected to have higher rates of wear than species which consume significant amounts of easy to process foods.

8.2. Materials and Methods

Broken, obscured and discoloured teeth are excluded from the analysis, because it may not be possible to record the appropriate wear score accurately. If a molar is un-sided or the tooth type debated in the literature, then wear was recorded but the tooth not included in the analysis. The total number of permanent teeth recorded for each species is highlighted in Table 8.1.

Table 8.1. Number of permanent teeth for each sample recorded for occlusal wear.

Species	Observable Teeth
H. naledi	147
A. sediba	16
P. robustus	318
A. africanus	328
Early *Homo*	44
Baboons	880
Chimpanzees	2498
Drills	212
Gorillas	2090
Total	6533

Molars were scored in accordance with the method of Scott (1979), and for all other teeth, the method of Smith (1984) was used. Scott's (1979) method divides teeth into quadrants, in which each quadrant is given a score from 1 to 10. A value of 1 means a tooth is unworn or has negligible wear facets, while a score of 10 describes complete loss of enamel.

Smith's (1984) method is similar but teeth are not divided into quadrants, and the scale is from 1 to 8.

Most hominin teeth are isolated and not associated with a jaw, therefore all teeth were studied separately and not in relation to other teeth in a dentition. Because molars are divided into quadrants, the direction of wear can be estimated. The mean value for each quadrant of each tooth is calculated and then species compared. Notably, lingual and buccal quadrants are compared, as well as anterior and posterior teeth.

8.3. Results

The results highlight the reliability of using wear in reconstructing diet and behaviour, with the comparative primate samples fitting accurately with behavioural studies (Figure 8.1). With the most worn teeth of chimpanzees and baboons being the central incisors, whereas for gorillas it is the first molars. This fits with chimpanzees and baboons using anterior teeth more often to process foods before consumption (Boesch et al., 2002; Isabirye-Basuta, 1989; Macho, 2014; Phillips & Lancelotti, 2014). *Homo naledi* stands out for having the highest wear of all species for both first and second premolars, yet has the lowest wear scores for first and second molars (Table 8.2). The wear pattern of *H. naledi* is the same as baboons and chimpanzees with the central incisors the most worn teeth. *Paranthropus robustus*, on the other hand, has an entirely different wear pattern, with the first molars having the most wear and the anterior teeth, particularly the central incisors, noticeably less worn than the other species (Figure 8.1).

Paranthropus robustus has the flattest molar wear of all the species studied (Table 8.3). The extant primate samples show greater differences between buccal and lingual cusps in both in the upper and lower dentition than the hominins (Figure 8.2 and 8.3). Out of the hominin species, *H. naledi* has the biggest difference between buccal and lingual wear, which, particularly in mandibular teeth, approaches the extant primate samples in steepness. Indeed, some of the *H. naledi* molars show extreme oblique wear (Chapter 5).

Table 8.2. Mean wear scores for each species split by tooth type. Anterior teeth and premolars are recorded following Smith (1984) and molars following Scott (1979).

Species	I1	I2	C	PM1	PM2	M1	M2	M3
Chimpanzees	4.69	4.30	3.67	2.88	2.85	4.01	3.99	3.60
Gorillas	3.86	3.36	3.35	2.61	2.68	4.36	3.67	3.47
Baboons	5.54	5.20	3.29	2.90	2.95	5.01	4.17	3.19
P. robustus	3.32	3.00	2.82	2.83	2.95	4.04	3.64	2.98
A. africanus	3.60	2.85	3.28	2.85	2.88	4.10	3.67	2.89
H. naledi	4.13	2.56	2.96	3.11	3.40	3.92	3.11	3.19

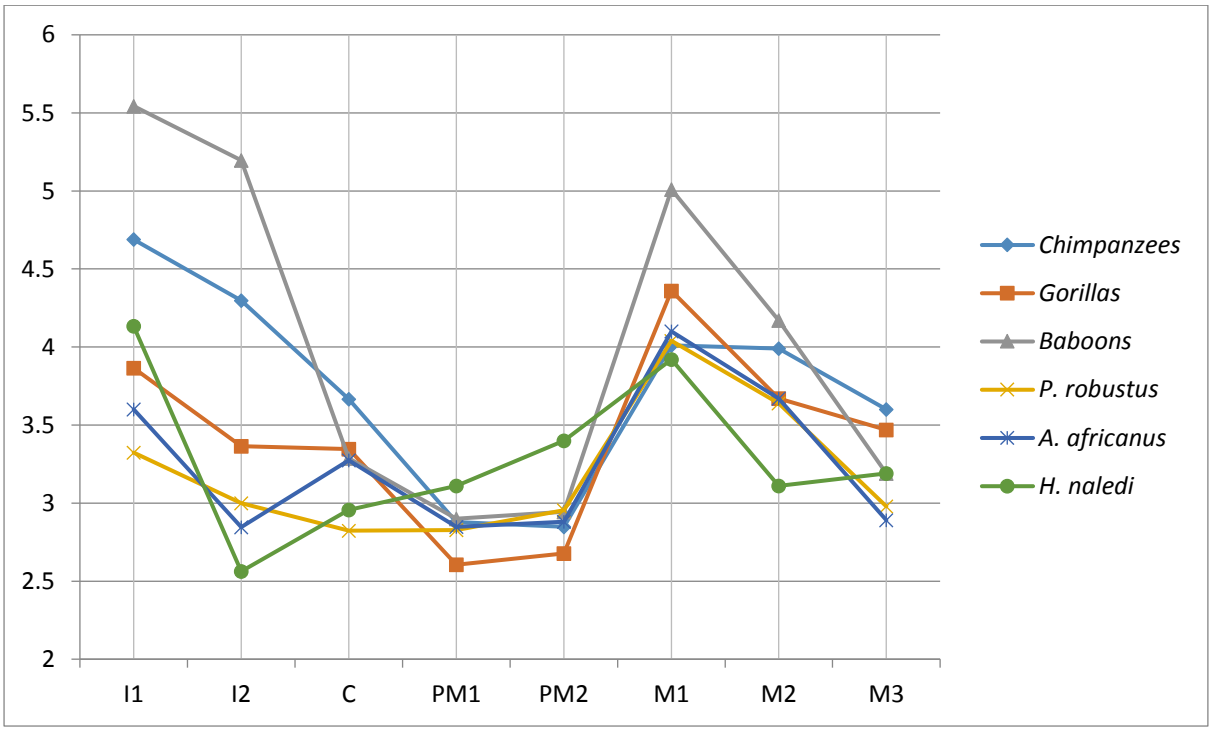

Figure 8.1. Mean wear scores for each species split by tooth, anterior teeth and premolars recorded following Smith (1984) and molars following Scott (1979).

Table 8.3. Mean wear scores for all three permanent molars combined, split by buccal and lingual and by the jaw. Wear score is calculated following Scott (1979).

Species	Upper buccal	Upper Lingual	Lower Buccal	Lower Lingual
Chimpanzees	3.45	4.24	4.36	3.56
Gorillas	3.44	4.43	4.35	3.43
Baboons	3.42	4.5	5.11	3.8
P. robustus	3.72	3.91	3.64	3.28
A. africanus	3.23	3.54	3.88	3.45
H. naledi	3.22	3.65	3.96	3.22

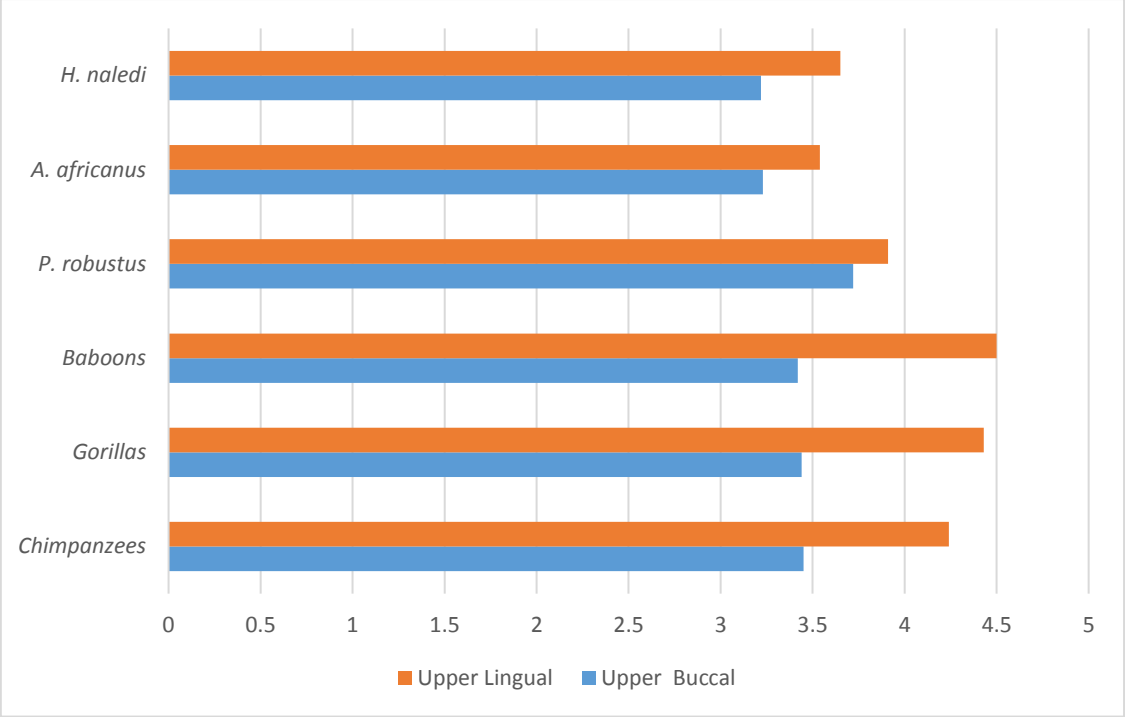

Figure 8.2. Mean wear scores for all upper molars split by buccal and lingual. Wear score is calculated following Scott (1979).

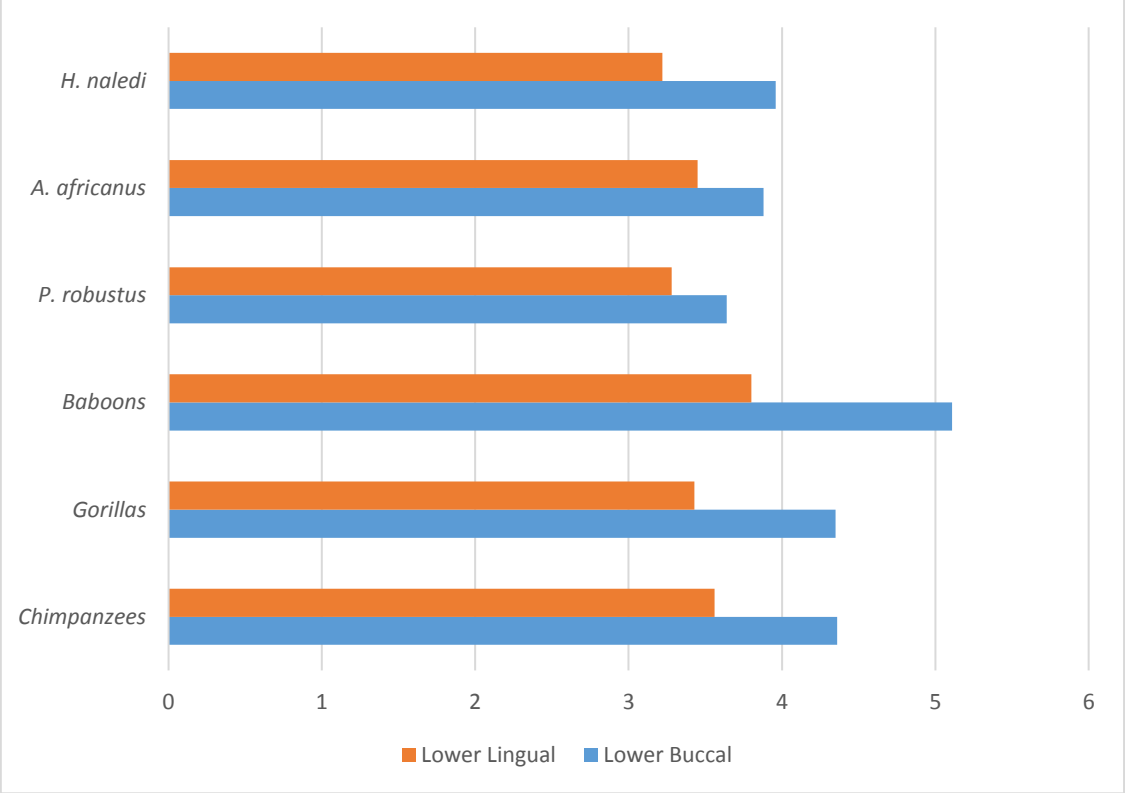

Figure 8.3. Mean wear scores for all lower molars split by buccal and lingual. Wear score is calculated following Scott (1979).

8.4. Discussion

Wallace (1975) looked for different types of wear in the South African hominins, including occupational, ritual, and dietary. He found evidence of only normal dietary wear. Again, that is the same results as found here, with one potential exception. This individual is discussed at the end of this chapter as a case study.

Differences in wear patterns between species will be observable based solely on dental morphology, enamel properties and dental developmental timing (Grimoud et al., 2017; Lavelle, 1970). In diverse primate species, different teeth erupt at different times and therefore individual teeth may be in occlusion comparatively longer in certain species. However, in this research, only the general trends are studied, with anterior vs. posterior and buccal vs. lingual wear strongly related to diet (Burnett et al., 2013; Fiorenza et al., 2011).

Contrary to Robinson's (1956) suggestion, both *A. africanus* and *P. robustus* share the same pattern of wear, as do all other samples in this project. Upper lingual surfaces are on average more worn than buccal surfaces, with the opposite relationship found on lower molars. That said, *P. robustus* has the flattest wear of any species and therefore likely explains why in a smaller sample Robinson found the opposite pattern, i.e. the wear is close to flat, and therefore sub-samples will probably show both directions of wear. Wallace (1975) notes that *P. robustus* tends to have roughly horizontal wear planes in incisors, whereas *A. africanus* wear planes are more angled. Wallace suggests this wear difference may, at least partly, be due to differences in food crushing. Wallace (1975) also suggests the canines and incisors of *P. robustus* may have functioned as an edge to edge occlusion for crushing food items, effectively meaning all teeth were functionally similar with little tearing or slicing with anterior teeth. The results of this study support this conclusion, with the anterior teeth appearing not to be regularly used in processing foods considering the low rate of wear compared with other species. Furthermore, *P. robustus* has similar rates of wear on all anterior teeth and premolars, perhaps also suggesting similar masticatory duties were performed. In sum, the results here agree with previous research that shows *P. robustus* has flat molar wear compared with other hominins (Cachel, 1975; Grine, 1981, 1986; Wallace, 1975).

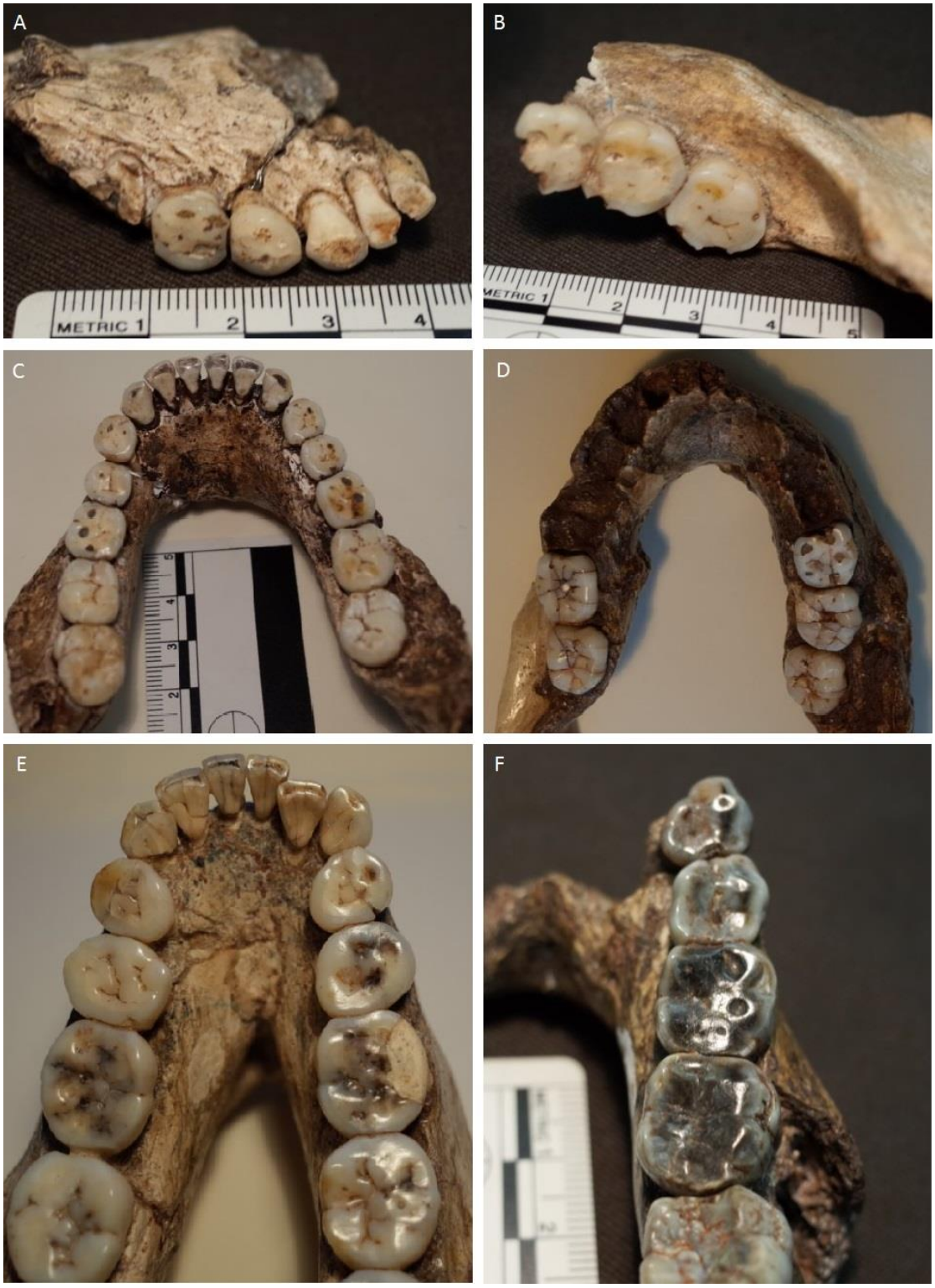

Figure 8.4. Mandibular wear in the different hominin species. A) MH 2 (*A. sediba*) right anterior teeth and premolars B) the same individual as A, right molars; C) UW 101-1261 (*H. naledi*); D) SK 15 (early *Homo*); E) SK 23 (*P. robustus*); F) STW 404 (*A. africanus*).

Wallace (1975) notes that *P. robustus* specimens tend to display lower cusps on molars than those of *A. africanus*. Robinson (1956) made a similar comment regarding the first premolars of *P. robustus*, mentioning their low and bluntly rounded cusps. Robinson also commented that after very little wear the cusps become flat and rounded tubercles. Wallace (1975) built on this by observing that not only do *P. robustus* posterior teeth wear flat, but they do not seem to expose any dentine before they do. In *A. africanus* on the other hand dentine exposure occurs before the cusps of posterior teeth have worn horizontal (Figure 8.4e,f).

It is suggested that the flat occlusal wear observed in *P. boisei* may be the result of eating underground storage organs (Macho, 2016), with comparisons with hunter-gatherer groups used as evidence (Smith, 1984). Tobias (1967) notes that OH 5 (*P. boisei*), shows substantial wear considering the third molars were not fully erupted at the time of death. He comments on the fact that many teeth show cusps completely reduced and large areas of dentine exposed. He then compares this specimen to South African hominins (STS 52a, STS 37, SK 13, SK 52, and SK 49), noting that OH 5 has substantially faster rates of wear. More recent research, using methods such as microwear and isotopic analysis, has suggested the diet of South African hominins differed significantly from that of *P. boisei*, conceivably supporting Tobias's observations.

A specimen assigned to early *Homo*, SK15, has been noted for its wear. Particularly the rather even wear on the buccal and mesial cusps of the lower left first molar, with this type of wear not seen in other Swartkrans teeth (Wallace, 1975; Figure 8.4d). This wear is very similar to that observed in *H. naledi* (Figure 8.4c). However, *H. naledi* does not seem to produce a prominent dentine patch on the mesial lingual quadrant until the three buccal dentine islands are well established (Figure 8.5). This difference in wear may signify slight dietary differences between these species. The wear on the mandibular teeth of *H. naledi* is very similar across individuals and molars, with three dentine islands tending to form on the buccal side before further wear occurs. Similar wear is also seen in other *Homo* specimens, such as a *H. erectus* specimen, KNM-ER 992.

Figure 8.5. *Homo naledi* molar wear. The severity of wear increases from left to right. Specimens from left to right: UW 101-1261; UW 101-377; UW 101-001; LES 1. LES 1 edited from Hawks et al. (2017).

Dentine islands are localised areas in which the dentine has been exposed on the surface through occlusal wear (Figure 8.5). The wear pattern of *A. sediba* is most similar to *H. naledi*, although due to the small number of teeth further comparisons are not possible. However, as in *H. naledi*, MH 2 exhibits three dentine islands on the buccal side of the mandibular first molar and has corresponding heavy wear on the anterior teeth, a pattern very similar to UW 101-1261 (Figure 8.4a,b).

8.4.1. Dietary and behavioural inferences

The flat wear of *P. robustus* fits with the hypothesis that they had a specialised diet, as opposed to regularly consuming a variety of food types (Eshed et al., 2006; Lev-Tov et al., 2003). Additionally, comparisons with human hunter-gatherer groups, as well as orang-utans, suggest a diet of tough or fibrous foods likely created these flat occlusal surfaces (Fiorenza et al., 2015; Grimoud et al., 2017; Kaidonis, 2008; Kaifu et al., 2003; Molnar, 1972; Smith, 1984). Therefore, these results add further support to the idea that *P. robustus* was a specialised feeder, potentially on low-quality vegetable matter. Additionally, the less worn anterior teeth compared to other hominins suggests they were not commonly used for extensive food processing, supporting the conclusion that their diet varied little compared with the other species.

Homo naledi individuals appear to have used their anterior teeth commonly for processing foods, with central incisors displaying the highest mean wear score, a pattern seen in chimpanzees and baboons but not gorillas, *P. robustus* or *A. africanus* (Figure 8.1; Fiorenza et al., 2015; Hillson, 1996). *Homo naledi* also has the steepest molar wear of the hominins (Figures 8.2 and 8.3), potentially relating to high levels of grit in their diets (Chapter 5; Grimoud et al., 2017; Macho, 2016). A varied, non-specialised, diet may also be involved in creating this wear pattern (Eshed et al., 2006). What is clear is the vast difference between *P. robustus* and *H. naledi*, with completely different wear patterns. *Australopithecus africanus* falls between these two species for both molar slope and which teeth have the severest wear. *Australopithecus sediba* and early *Homo* show similar patterns to *H. naledi*, however, the sample for these species are too small for firm conclusions.

There are a few cases of asymmetrical wear. Figure 8.6 shows two such examples. The lateral lower right incisor of SK 23 (*P. robustus*) has erupted inferiorly to its normal position. The surrounding teeth are also crowded. The left and right incisors show different degrees of wear with the right teeth having more dentine exposed (Figure 8.6a). Another specimen with similar differences between sides is STS 52 (*A. africanus*), showing higher wear on the right upper canine than the left, again likely associated with a crowded lower dentition (Figure 8.6b; Oppenheimer, 1964). As a whole, however, no species shows substantially more wear on one side over the other, for any tooth group.

Figure 8.6. Asymmetric wear (arrows) A) SK 23 (*P. robustus*), mandible. B) STS 52a (*A. africanus*), maxilla.

8.5. Conclusions

Overall, the results of this study agree with that of the majority of previous research, in that *P. robustus* clearly has flatter occlusal wear than other South African hominins. Furthermore, *P. robustus* has the flattest wear of all species studied in this thesis, with the extant primates having steeper wear than those of the fossil hominins. Enamel and morphology properties are clearly important, but the results suggest that *P. robustus* diet differed from the other hominins and perhaps hints at a specialised diet of low-quality vegetation. *Homo naledi* likely used their anterior teeth for processing foods more than the other hominin samples, and considering this species has the steepest molar wear of the hominins, likely had a varied diet.

Chapter 9: Abscesses, periodontal disease and antemortem tooth loss

9.1. Introduction

Abscesses, periodontal disease, and Antemortem Tooth Loss (AMTL) are considered together in this chapter. Studying these pathologies concurrently is justified because they are recorded on the alveolar bone of the jaw rather than the tooth, and secondly, they are all limited in terms of numbers of observable specimens. Indeed, only a couple of examples of all three are present in the entire hominin sample, so inferences into diet and behaviour are not possible. Instead, only a limited number interesting examples are presented.

Antemortem tooth loss can be caused by a variety of factors. Most commonly in modern day populations caries is responsible. In the past dental wear would have played a much greater role. However, as the analysis of the individuals from the medieval Gloucester site demonstrates, just because individuals from a site have extremely high rates of caries does not necessarily mean AMTL may stand out (e.g., Caglar et al., 2007; Novak, 2015; Slaus et al., 1997; Vodanović et al., 2005). Other factors are likely important, such as periodontal disease, fractures, and extreme attrition (Caglar et al., 2007; Esclassan et al., 2009; Whittaker et al., 1998). Furthermore, there may be differences in patterns of AMTL between populations. For example, AMTL in posterior teeth of certain populations is more likely to be produced by heavy attrition or caries, whereas for anterior teeth extra-masticatory behaviour is a likely cause (Lukacs, 2007; Molnar, 2011). Therefore, in populations such as hunter-gatherers and fossil hominins in which caries occurrence is relatively rare, and occlusal wear high, the likelihood is that the vast majority of AMTL is not caused by caries.

Periodontal disease develops when certain bacteria accumulate around the gingival margins (Eke et al., 2012; Mikuls et al., 2014; Ogden, 2008). It is widespread and prevalent in mammals and can lead to significant bone destruction (Branch-Mays et al., 2008; Cochran, 2008; Hillson, 1996). For example, in modern day Britain 43% of the population display periodontal disease (Morris et al., 2001). Periodontal disease is often split into gingivitis and periodontitis. Gingivitis only involves soft tissue and therefore cannot be assessed in the fossil record. Both gingivitis and periodontitis are usually painless with the former not necessarily

leading to the latter (Page & Kornman, 2000). Periodontitis is not reversible, as gingivitis can be, and is, therefore, a progressive and irreversible condition (Hajishengallis, 2015). However, periodontitis can be slowed by changes in oral health and diet (Ogden, 2008). The maxilla tends to be more susceptible to periodontal disease than the mandible, likely due to differences in bone thickness but it may also be related to the complexity of the root morphology of maxillary molars (Kerr, 1998). Periodontal disease has been associated with a number of different conditions, including diet (Lieverse, 1999), heart disease (Geismar et al., 2006), respiratory disease (Scannapieco & Ho, 2001), premature birth (Moore, 2002), vitamin D deficiency (Davideau et al., 2004), social status (Borrell et al., 2004), osteoporosis (Jeffcoat et al., 2003), and pregnancy (Katz et al., 2000). Rates have increased through time, with higher periodontal frequencies in Neolithic populations and further increases in the Bronze Age and Medieval periods (Hildebolt & Molnar, 1991; Molnar & Molnar, 1985). Periodontal disease has only rarely been reported in fossil hominins (e.g., Grine, 1981; Grine et al., 1990; Hildebolt & Molnar, 1991).

Abscesses and other voids are commonly found in the mandible and maxilla of humans and other animals (Miles & Grigson, 2003; Sauther et al., 2002). Such voids often develop due to the exposure of a tooth's pulp chamber. Many potential processes, including heavy wear, trauma and caries, can cause the exposure of a pulp chamber (Linn et al., 1987; Nair, 2004). As well as abscesses, other types of voids include cysts, fenestrations and granulomata (Nair, 2004; Ogden, 2008). Although it may seem harmless to call all such voids abscesses, there have been concerns raised about their incorrect use (Dias & Tayles, 1997; Hillson, 2005). The main issue is that the majority of voids may be painless granulomata, with evidence suggesting less than a third may be chronic abscesses (Ogden, 2008). However, given the fact that a granuloma can become a cyst and both of these an abscess, there will be times when a lesion will be hard to decipher in the fossil record. Moreover, in comparative studies, there is much disagreement over what counts as an abscess (Ogden, 2008). Therefore, perhaps the most pertinent figure to present is the total number of teeth with associated voids in the maxilla or mandible.

The frequency of abscessing varies substantially among populations of modern humans, and also differs among species of primates (Dias & Tayles, 1997; Legge, 2012; Lukacs,

1992; Miles & Grigson, 2003; Roney, 1959; Schultz, 1956). Great apes appear to have higher rates than other primates, with the canine the most frequent tooth involved (Schultz, 1956). Legge (2012) looked at the effect tooth breakage may have on abscess rates in chimpanzees but found no relationship between the two. His study also failed to show much difference between the two species of chimpanzees and between sexes, leading him to conclude that occlusal attrition caused by diet was more important than social activity or tooth crown height.

9.2. Methods

The majority of the hominin fossils are isolated teeth, and therefore the sample for this chapter is reduced to only a few well preserved specimens. Substantial bias may be present due to teeth with periodontal disease being supported by less bone and therefore more likely to become detached from the maxilla or mandible. For this reason comparison with the extant primate collection is not made. However, particular cases can give insight into the lifeways of the individual and therefore case studies of certain specimens are presented.

Periodontal disease is commonly recorded if there is a gap larger than 2mm from the CEJ to the alveolar crest. However, such a gap may not necessarily be the result of periodontal disease. Heavy occlusal wear and loss of opposing teeth may lead to further eruption (i.e., supereruption) of a tooth, which may artificially resemble periodontal disease in the archaeological record; however, this possibility has been debated in the literature (Clarke & Hirsch, 1991; Gottlieb & Orban, 1933; Newman & Levers, 1979; Odgen, 2008; Picton, 1957; Sagne & Olsson, 1977). The small sample size studied here requires that the cases of potential periodontal disease are discussed and described separately.

As mentioned in the introduction of this chapter, all voids present in a maxilla or mandible that are associated with a tooth root apex were recorded. A differential diagnosis of each void was conducted, using criteria described by Dias and Tayles (1997) and Ogden (2008). If a lesion is not associated with a root apex, then it is not included here since a systemic disease such as multiple myeloma and metastatic carcinoma is likely responsible (Ogden, 2008). Such an example is not found in the hominin sample; however, instances were observed in the extant primates.

For all samples, the presence of abscesses, periodontal disease and AMTL was based of the criteria outlined by Ogden (2008). For the extant primate sample, frequencies for all variables found in a species are displayed as a percentage of the number of tooth positions with associated mandible or maxilla. A tooth needs to have all the surrounding bone present to be included i.e. past the border with adjacent teeth. Bone loss caused by an abscess, cyst or granuloma around a tooth is not considered as periodontal disease (Hildebolt & Molnar, 1991).

9.3. Results

From the entire hominin sample there are only two abscesses, four potential AMTL's and six teeth with periodontal disease (Table 9.1). There are no cases of these three pathologies in any of the primary dentitions (Table 9.2).

Table 9.1. Frequencies of abscesses, AMTL and periodontal disease for all permanent hominin teeth.

Permanent teeth	Abscess	AMTL	Periodontitis
Teeth observable	450	854	473
Teeth with pathology	2	0-4	6
Total teeth	452	858	479
%	0.44	0-0.47	1.25

Table 9.2. Frequencies of abscesses, AMTL and periodontal disease for all deciduous hominin teeth.

Deciduous teeth	Abscess	AMTL	Periodontitis
Teeth observable	40	86	44
Teeth with pathology	0	0	0
Total teeth	40	86	44
%	0	0	0

Both abscesses and three of the potential AMTL are from the early *Homo* specimen SK 80. The other AMTL is from the *A. africanus* specimen STW 14. These four potential cases of AMTL, however, are all recorded as not possible to determine if the missing tooth was lost ante or postmortem. In STW 14 it looks as if the second premolar has been lost antemortem; however, this could easily not be the case, with potentially post-mortem processes looking superficially like an antemortem loss. Similarly, it is more probable that the teeth of SK 80

were lost postmortem given the lack of alveolar resorption in the tooth sockets. All cases of periodontal disease are on *P. robustus* dentitions, with five teeth from SK 12 and one from SK 65.

Frequencies of AMTL, periodontal disease and abscesses for the comparative primate samples are presented in Table 9.3. Gorillas and chimpanzees have similar rates, but baboons stand out as having extremely low levels of all three pathologies, with only one case of an abscess and three AMTL.

Table 9.3. The frequency of abscesses, AMTL, and periodontal disease in the competitive extant primate samples.

Pathology	Variable	Chimpanzees	Gorillas	Baboons
Abscesses	Teeth without	2414	2060	802
	Teeth with	50	39	1
	Total teeth	2464	2099	803
	%	2.03	1.86	0.12
AMTL	Teeth without	56	2090	790
	Teeth with	2458	16	3
	Total teeth	2514	2106	793
	%	2.23	0.76	0.38
Periodontal disease	Teeth without	2000	1713	797
	Teeth with	423	355	0
	Total teeth	2423	2068	797
	%	17.46	17.17	0.00

9.4. Discussion

There has been much debate surrounding the early *Homo* specimen SK 80, also now commonly referred to as SK 847 due to two other fragments being incorporated (Clarke & Howell, 1972; Grine et al., 1993, 2013; Wolpoff, 1971). This partial maxilla also has much of the frontal cranium present (Figure 9.1a) and has been associated with different mandibles, including SK 45 and SK 74a (Clarke & Howell, 1972; Wolpoff, 1971). Only one anterior tooth remains, with severe wear visible (Figure 9.1c). There are what looks like three abscesses on the buccal surface associated with the maxillary incisors (Figure 9.1d). Two of these superficially look like post-mortem damage; however, this is likely further damage to an already existing void, given the definite abscess associated with the left upper central incisor (Figure 9.1e). The left upper central incisor is not present, likely due to post-mortem factors

(although ATML section above). There is also what is potentially an abscess on the lingual surface, from what was likely a drainage channel associated with right lateral incisor, with this tooth again not being present (Figure 9.1b). However, a CT-scan of this specimen is needed to rule out morphological features. Given the heavy wear on these teeth, the abscesses are almost certainly a result of heavy wear exposing the pulp chamber. The abscesses originate from the apices of the incisor roots and are therefore unlikely to represent a systemic disease such as multiple myeloma (Dias & Tayles, 1997). The voids visible best fit the description of an abscess rather than a cyst or granuloma, with a rounded thickened rim around the void (Figure 9.1e; Dias & Tayles, 1997; Ogden, 2008).

The maxillary teeth of SK 12 (*P. robustus*) show alveolar resorption. The specimen does also show post-mortem damage. However, there appears to be a consistent 3-5mm gap between alveolar bone and the CEJ, which is almost certainly antemortem in nature given the complete alveolar bone in some areas (Figure 9.2). The left upper canine of SK 65 (*P. robustus*) has a sufficient gap antemortem to also count as periodontal disease. Another case of periodontal bone loss has been described in the literature, STS 24 which is assigned to *A. africanus* (Grine, 1981; Grine et al., 1990).

Compared with other pathologies and defects studied in this thesis periodontal disease is perhaps the most unreliable when inferring diet or behaviour. Partly this is due to the issue of continuous eruption masking frequencies, but mainly because of the small number of complete maxilla and mandibles compared with the number of teeth. A further issue is that most human populations are affected by periodontal disease regardless of oral hygiene measures (Clarke & Hirsch, 1991; Newman, 1999; Ogden, 2008). Therefore, it is not adequately understood what effect genetic factors, diet and oral health have on periodontal disease rates at a population level. Consequentially, trying to disentangle meaning from frequencies for fossil species is difficult. For example, it has been suggested that some individuals may be predisposed to periodontitis at an early age, with a possibility that around 10% of individuals may be particularly susceptible and a similar amount being particularly resistant (Anerud et al., 1983; Kerr, 1998; Ogden, 2008).

Figure 9.1. SK 80 (early *Homo*): A) overview of the entire specimen; B) potential lingual abscess; C) heavy wear on left lateral upper incisor; D) buccal abscesses; E) close up of a buccal abscess.

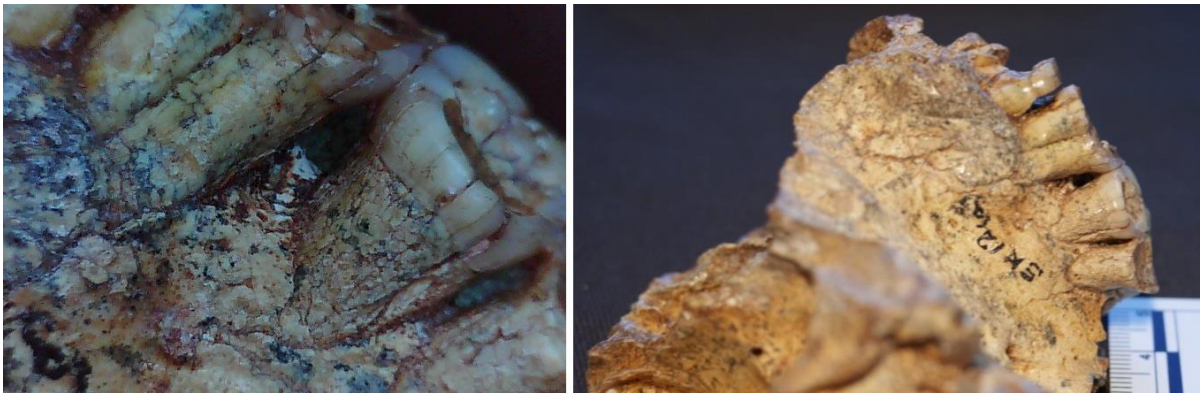

Figure 9.2. SK 12 (*P. robustus*), maxilla showing likely periodontal disease.

9.5. Conclusion

There is a very limited number of instances of abscesses, periodontal disease and AMTL in the fossil record, however, the examples presented here explore a few new examples of dental pathology in South African early hominins. Additionally, the abscesses on the maxilla of SK 80 highlight that this individual used its anterior teeth extensively, to the point that the pulp chamber was exposed on multiple teeth. CT-scans of this specimen may provide further information on the extent and spread of these abscesses. This is the earliest hominin example of a dental abcess and shows that this early *Homo* individual was able to cope with several abscesses at the same time, clearly surviving for a significant amount of time.

Chapter 10: Tertiary dentine

Dentine is exposed on around half of the teeth in this study (Chapter 8). Therefore, studying dentine properties may be just as important as enamel, particularly in populations that exhibit heavy wear. Comparatively few studies have looked at dentine in this way (e.g., Geissler et al., 2015; Pampush et al., 2016). In this chapter tertiary dentine, one aspect of dentine that can be seen macroscopically, is studied. There is often confusion as to what to call this phenomenon. 'Secondary dentine', 'tertiary dentine', 'irregular secondary dentine' and 'reparative dentine' have all been used in the literature to describe this occurrence (e.g., secondary dentine: Ortner, 2003; tertiary dentine: Foster et al., 2013; irregular secondary dentine: Hillson, 2005). Here the descriptions in Foster et al. (2013) is followed and the term tertiary dentine used.

Primary dentine is produced during tooth formation and is succeeded by secondary dentine which is an ongoing slow process (Rutherford et al., 1995). Tertiary dentine forms as protection against insult, caused primarily by excessive wear, microbial infection and caries (Bjørndal, 2001; Fischer et al., 1970; Foster et al., 2013; Mjör & Karlsen, 1970; Ricucci et al., 2014; Stanley et al., 1966; Wennberg et al., 1983). This type of dentine can be formed in two ways. Reactionary, in which new dentine forms from the pre-existing odontoblast, and reparative in which new odontoblast-like cells are formed because the original odontoblasts have died (Ricucci et al., 2014). Tertiary dentin can only be formed when an odontoblast is directly affected by stimuli, and so the position and structure depends on the type and intensity of the force (e.g., occlusal wear and caries). The colour of tertiary dentine is distinct from the surrounding primary dentine, being usually darker in appearance (Hillson, 2005). It is rarely recorded in the literature, and cross-species studies have not been carried out on fossil hominins, although its presence has been noted (e.g., Margvelashvili et al., 2013).

10.1. Materials and Methods

A major issue with comparing frequencies of tertiary dentine is the amount of post-mortem discoloration on many of the hominin teeth. Particularly common is for dentine islands on the occlusal surface to be stained but for the surrounding enamel to appear unaffected. Teeth with such staining are excluded from analysis.

Therefore, only teeth that have dentine exposed on the occlusal surface and are not damaged or obscured by post-mortem damage are included. Tertiary dentine is then marked as present or absent for each tooth. Clear colouration changes that are undoubtedly antemortem in nature are recorded as tertiary dentine (Figure 10.1). Only obvious areas of tertiary dentine that are visible with the naked eye are included.

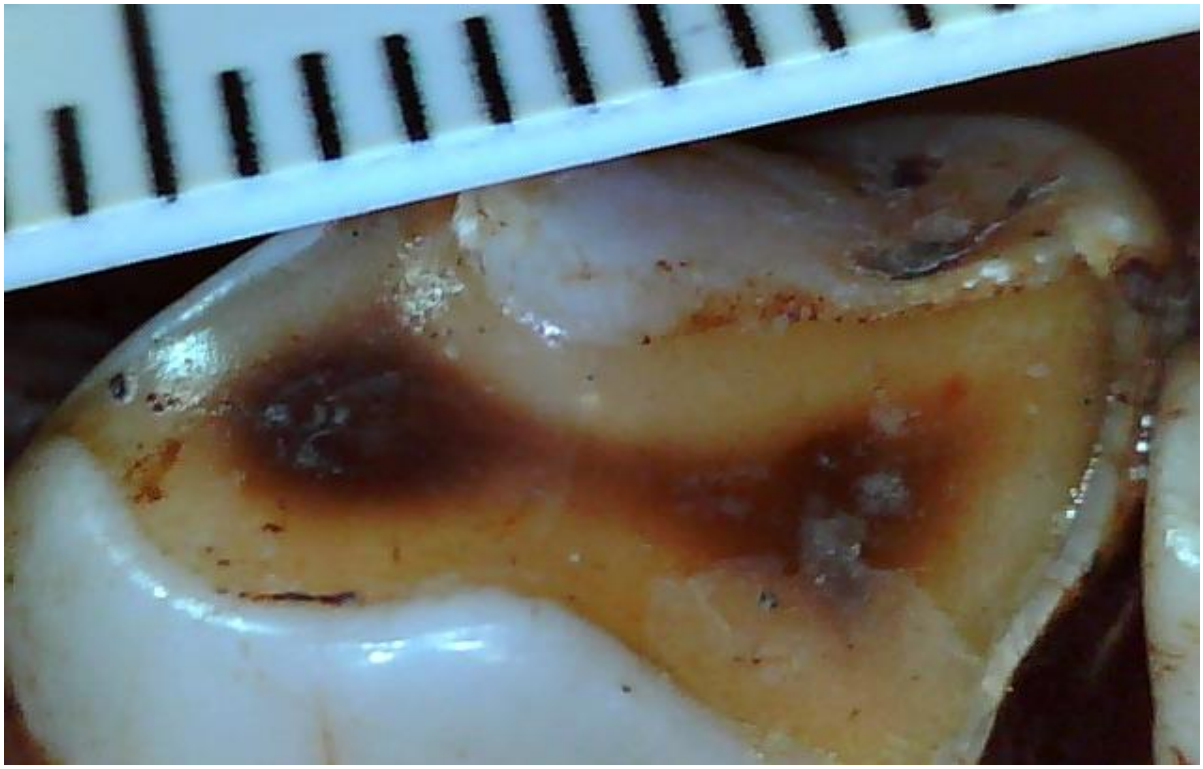

Figure 10.1. Gorilla (M 786) lower left first premolar showing tertiary dentine formation.

10.2. Results

Of the three hominin species with large enough sample sizes to allow comparisons, all have very similar tertiary dentine frequencies (Table 10.1), with no statistically significant differences between them (*P. robustus* vs. *H. naledi*: X^2= 0.326, 1 df, p= 0.5679). The extant primate samples have substantially higher rates, with gorillas standing out with over 90% of teeth with dentine exposed having tertiary dentine. The same order for the extant primate sample is shown in deciduous teeth, with gorillas having the highest frequency followed by chimpanzees and then baboons (Table 10.2).

Table 10.1. The frequency of tertiary dentine on the permanent teeth of the different species.

Species	Tertiary dentine present	No Tertiary dentine	Tertiary dentine %
A. africanus	12	78	13.33
P. robustus	9	66	12.00
H. naledi	5	26	16.13
Baboons	215	321	40.11
Chimpanzees	448	501	47.21
Gorillas	917	67	93.19

Table 10.2. The frequency of tertiary dentine on the deciduous teeth of the comparative primate samples.

Species	Tertiary dentine present	No Tertiary dentine	Tertiary dentine %
Baboons	16	35	31.37
Chimpanzees	113	128	46.89
Gorillas	131	61	68.23

10.3. Discussion

Little is known regarding the processes and properties of tertiary dentine (Ricucci et al., 2014). However, it is known this material forms as a response to stressors, mainly caries and heavy wear (Bjørndal, 2001; Foster et al., 2013; Mjör & Karlsen, 1970; Stanley et al., 1966; Wennberg et al., 1983). Why species differences exist has rarely been explored. The results show that hominins group closely together. The fact that other variables in this thesis vary dramatically among hominin species may suggest the formation of tertiary dentine is more closely linked to phylogeny rather than diet. However, the extant primates studied are substantially different. The differences between the extant primate samples does not appear to be an artefact or a bias of more worn teeth in particular samples, with all species represented by a wide range of wear stages that all cluster around the same mean (Chapter 8). Additionally, the deciduous teeth for the comparative primate sample follow the same pattern as the permanent teeth suggesting genuine species differences. Differences in the speed of wear between species may be an influencing factor with wear that is too fast potentially not allowing enough time for tertiary dentine formation (Fischer et al., 1970; Foster et al., 2013; Mjör & Karlsen, 1970; Ricucci et al., 2014; Stanley et al., 1966; Wennberg et al., 1983). However, it the main factor is likely dentine properties, with certain species,

particularly gorillas, clearly able to lay down tertiary dentine faster and more often. The high speed of tertiary dentine formation in gorillas has potentially evolved as a consequence of their diet to allow continuation of sharp cutting edges. However further research is needed to prove this hypothesis. Additionally, when frequencies for different hominin groups, particularly modern humans, have been researched, further conclusions on what the low rate of tertiary dentine in the fossil hominins means in terms of diet can be further explored.

Chapter 11: Case studies

11.1. Chimpanzee with amelogenesis imperfecta

Amelogenesis imperfecta (AI) is a group of genetic conditions that create enamel defects, affecting one in every 700 to 14,000 humans (Crawford et al., 2007; Sundell & Koch, 1984). A variety of different genetic mutations can be responsible for these heritable diseases (Horvath et al., 2014; Kelley & Swanson, 2008; Smith et al., 2016; Wang et al., 2015). Pitting and plane form enamel hypoplasia is common, but abnormal enamel density, thickness and coloration is also associated with different forms (Aldred et al. 2003; Chamarthi et al., 2012; Huckert et al., 2015; Mardh et al., 2002; Mehta et al., 2013; Ozdemir et al., 2005; Poulter et al., 2014; Schuurs, 2012; Wright, 1985, 1993). To differentiate between different types of AI, clinical, histological and radiographic methods have been used (Crawford et al., 2007; Mehta et al., 2013; Wang et al., 2015).

AI has yet to be implicitly stated to be present on a non-human primate dentition, however research has implicated genetic factors in the formation of severe defects and there are also descriptions of specimens that fit with what is now classified as AI (Jones & Cave, 1960; Miles & Grigson, 2003; Tomes, 1898). Given the large amount of mutations that can cause these heritable conditions in humans it is perhaps surprising that more examples of similar defects in primates have not been described. This likely reflects the much greater sample sizes as well as proportionally more research that is carried out on humans.

11.1.1. Differential diagnosis

Although many of the extant primate and fossil hominin dentitions display enamel defects, with multiple types of hypoplasia commonly occurring (Chapter 6), only one individual displays defects that are consistent with a diagnosis of AI. This specimen, M 299, is an adult female chimpanzee. Pitting enamel hypoplasia is visible on all anterior teeth as well as the maxillary first premolars and both sets of lower premolars (Figure 11.1). There are no visible defects on the molars. Apart from the defects themselves the teeth appear normal in terms of size and morphology.

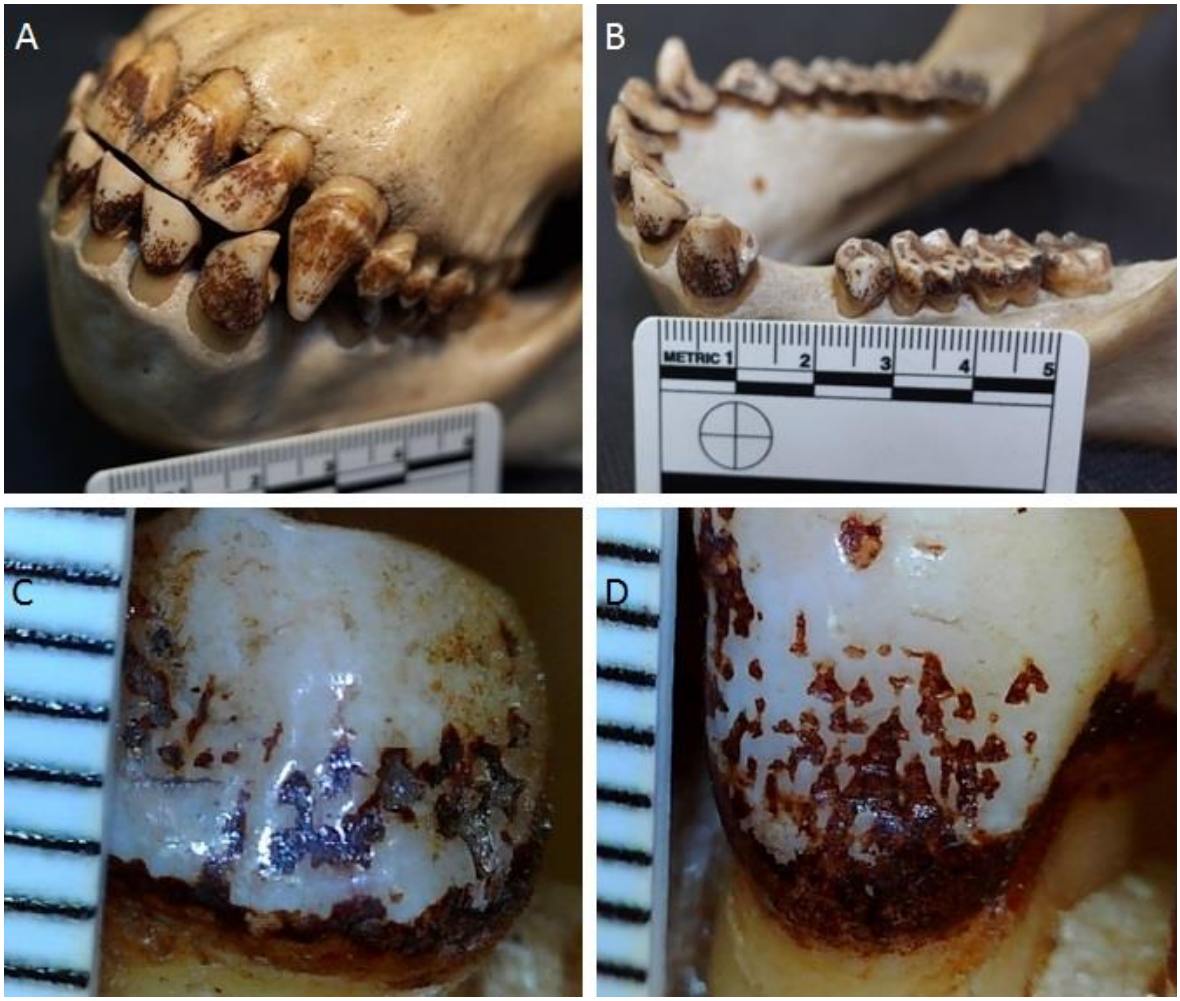

Figure 11.1. Female chimpanzee showing enamel defects and a congenital missing tooth. A) buccal/Labial view of enamel defects on anterior teeth; B) congenital missing left mandibular first premolar; C) buccal view of the lower right first premolar, with clear pitting/plane form defects; D) buccal view of the lower left lateral incisor, with clear pitting/plane form defects.

The pitting is irregular in both shape and size, but small circular depressions are most common (Figure 11.1a,d), with some larger irregular defects that resemble plane form defects (Figure 11.1c). Hypoplasia is most defined on the buccal surface and the cervical half of the crown. The anterior teeth all appear to be equally effected, with roughly the same position and severity of defects (Figure 11.1a,b). There is also a 'mottled' or 'wavey' appearance to the enamel on these teeth. The colour of the enamel appears normal; however, dark post-mortem coloration may mask defects. The mandibular left first premolar is congenitally absent (Figure 11.1b). There is no evidence of caries, ante-mortem enamel fractures, periodontal disease, or severe occlusal attrition. Small amounts of calculus are visible on the posterior mandibular teeth.

The pattern of defects and the teeth affected support a diagnosis of AI for M 299. The uniform pattern of defects on anterior teeth, and complete lack of defects on molars, strongly suggests these defects are not related to a physiological stress during development (Boughner et al., 2015; Crawford et al., 2007; Guatelli-Steinberg, 2015). Additionally, the pattern of defects do not resemble those caused by congenital viruses; vitamin deficiencies, malnutrition, and fluorosis and other mineral contaminants in humans (Hillson, 2014; Ioannou et al., 2015; Thylstrup & Fejerskov, 1978). A congenitally missing tooth adds further support to the diagnosis of AI, as these two conditions are commonly found together (Mehta et al., 2013).

Of the four main AI types that have been commonly categorized in humans, the characteristics on this chimpanzee's dentition match those of the Hypoplastic (Type 1) variety (Mehta et al., 2013; Shivhare et al., 2016; Wright, 1993). In line with this variety there is little or no colouration change visible, no obvious reduction in size or unusual interproximal spacing, and there is clear enamel pitting and plane form defects covering large areas of the crown in multiple teeth. Specifically the defects look indistinguishable from those recorded in humans as Type 1A (Seow, 1992; Witkop, 1988), which is characterized by enamel with pitting that varies from pinpoint to pinhead in size and is found predominantly on the buccal surfaces of permanent teeth (Witkop, 1976). It is also common in this condition for some teeth to show no visible defects (Witkop, 1988), likely explaining why the molars in this individual seem unaffected; however postmortem coloration and ante-mortem wear may conceal defects. This type of AI creates the least amount of dental issues for human patients (Seow, 1992); therefore potentially explaining the relatively good health, except for the defects themselves, of this chimpanzees teeth.

Although other potential cases of AI in primates need to be confirmed, it is suggestive that the range of defects exhibited in non-human primates may be as diverse as in humans (e.g., Tomes, 1898; Miles & Grigson, 2003). It is also likely that many examples have been overlooked in primate skeletal collections, particularly in light of recent DNA advancement showing just how diverse AI defects can be. DNA analysis of this individual as well as additional observations of AI in the dentitions of other primates would further our understanding of these enamel genetic disorders in primates as a whole.

No causes of AI were identified in the hominin material. However, this case study highlights that often whole dentitions are needed to know for certain if defects are caused by AI. Therefore, certain pitting and localised defects that have been described in Chapter 6 may have a genetic origin but given the fragmentary nature of the fossil record diagnosis is not possible.

11.2. *Australopithecus africanus* root grooves

Many human samples have instances of unusual dental wear that was caused by non-masticatory activities requiring repetition of the same action (Milner & Larsen, 1991). In the hominin fossil record one particular type of non-masticatory wear has been commonly described, the so-called toothpick, or interproximal, groove (Boaz & Howell, 1977; Bouchneb & Maureille, 2004; Brown & Molnar, 1990; Castro et al., 1997; Frayer & Russell, 1987; Hlusko, 2003; Lozano et al., 2013; Ricci et al., 2014; Sun et al., 2014; Turner & Cacciatore, 1998; Ubelaker et al., 1969; Ungar et al., 2001). Although there is now a consensus that most of these grooves are caused by the repeated placement of a non-dietary item in these specific locations, there is debate in the literature as to why such behaviour was performed. Removing food particles, grit-laden saliva, therapeutic relief and other pathologies have all been associated with these grooves (Calcagno & Gibson; 1991; Lozano et al., 2013; Turner & Cacciatore, 1998; Wallace, 1974). The objects used may be wide-ranging and difficult to infer from the fossil record, with Kaidonis et al. (2012) describing interproximal grooves in Australian Aborigines created by passing kangaroo tendon between molars.

Grooves are most commonly found in the interproximal areas of posterior teeth; however, similar grooves have been found on anterior teeth (Formicola, 1988; Frayer, 1991; Ungar et al., 2001). Micro striations are often present on surfaces with interproximal grooves, and these tend to be orientated buccal-lingual (Bouchneb & Maureille, 2004; Grine et al., 2000; Hlusko, 2003; Lozano et al., 2013; Ungar et al., 2001). Such striations have also been found on teeth that do not have a clear groove, with researchers suggesting these are likely caused by the same process, but there has not been sufficient time to create a clear depression (Bouchneb & Maureille, 2004; Estalrrich et al., 2016; Grine et al., 2000; Sun et al., 2014). These grooves have been found to affect cementum, enamel, and dentine and commonly are just above, below or on the CEJ.

Other types of lesions can form on the roots of teeth, namely caries and acidic erosion (Grippo et al., 2012). Dental erosion, or corrosion as it is commonly called, of dental tissue has been extensively researched in modern humans (e.g., Chapter 8; Grippo et al., 2004; Oginni et al., 2003; Zero, 1996), as well as archaeological specimens (e.g., Indriati & Buikstra, 2001; Ritter et al., 2009; Robb et al., 1991). Mention of dental erosion in fossil hominin research is however very rare. Wallace (1974) looked for interproximal grooves in South African hominins and found none; similarly Ungar et al. (2001) checked newer finds, and likewise found no evidence for the sort of interproximal grooves found in later hominins.

The teeth described in this case study are STW 270, a mandibular right lateral incisor, and isolated mandibular teeth of STW 213, thought to belong to the same individual (Moggi-Cecchi et al., 2006). Evidence for STW 270 belonging to STW 213 comes from the fact these specimens were found near each other and display similar preservation and wear (Moggi-Cecchi et al., 2006). These specimens were found in Sterkfontein Member 4, in which a minimum of 87 individuals are represented, and are aged 2.8–2.4 Ma (Pickering et al., 2004). Although many researchers see all the hominin material from Member 4 as belonging to *A. africanus*, there has been much debate in the literature as to whether there is justification for the presence of multiple hominin species (e.g., Calcagno et al., 1999; Fornai et al., 2015; Grine, 2013; Lockwood & Tobias, 1999; Moggi-Cecchi, 2003; Wood & Richmond, 2000). However, the specimens studied here do not feature in this debate and are universally seen as *A. africanus*; therefore, it is justifiable to compare these teeth with other canines and incisors thought to belong to *A. africanus*.

Once the unusual depressions on the roots of STW 270 and the right mandibular canine of STW 213 were observed and recorded during data collection, further analysis involving a microscope and higher quality photos were undertaken by Marina Elliott at the University of the Witwatersrand. These observations and photos are used here with Marina's permission.

11.2.1. Differential diagnosis

A shallow and elongated groove starting from the CEJ and extending almost a third of the way down the buccal surface of the root is present on STW 270 (Figure 11.2). The enamel seems unaffected, although there is some slight postmortem damage. The groove spreads

around to the interproximal areas of the cementum and pinches to a point towards the lingual surface (Figure 11.3). The groove is clearly antemortem in nature and not a morphological feature. A smaller depression is visible in the same position on the adjacent right mandibular canine of STW 213 (Figure 11.4).

There are no obvious directional striations; however, a SEM of an impression would certainly be informative. The other teeth belonging to STW 213 do not show similar root grooves; however, there are clear bands on the enamel, of what have been previously described as LEH, on the right premolars and canines. The left teeth show no enamel or root grooves.

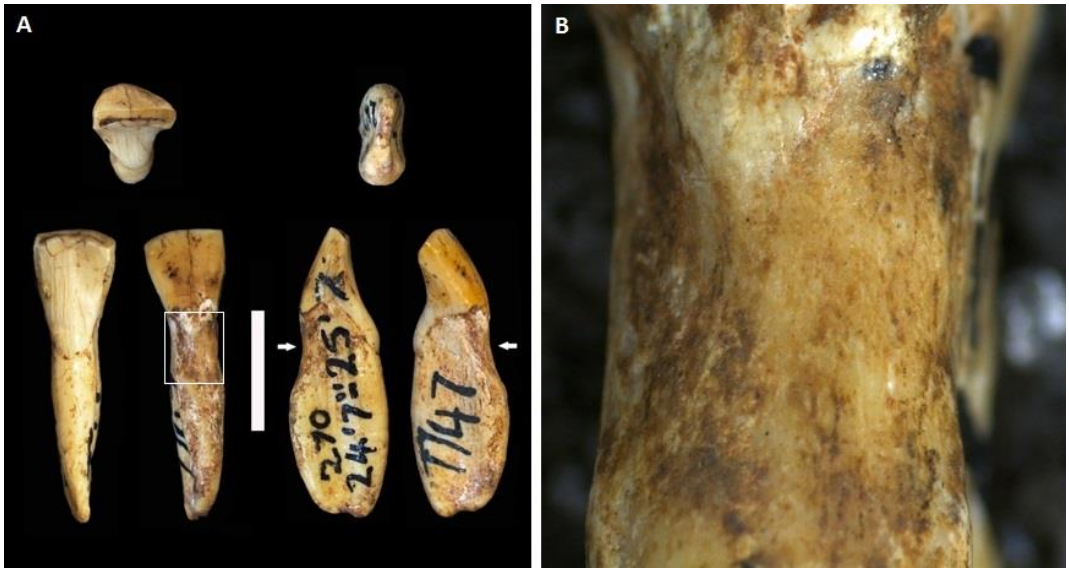

Figure 11.2. STW 270 (*A. africanus*) right mandibular lateral incisor. A) Bottom row from left to right: lingual, labial, mesial, and distal. White arrows and square highlight the location of the groove. The white bar is 1 cm long. B) Close-up of the groove (white square in A), showing no directional striations and a smooth surface.

The only mention of the grooves described above in the literature is in Moggi-Cecchi et al. (2006), in which no inferences into the origin of these antemortem lesions are given. They are instead used to help associate these specimens with each other.

Figure 11.3. STW 270 (*A. africanus*) right mandibular lateral incisor. Distal side, showing the root groove tapering toward the lingual surface.

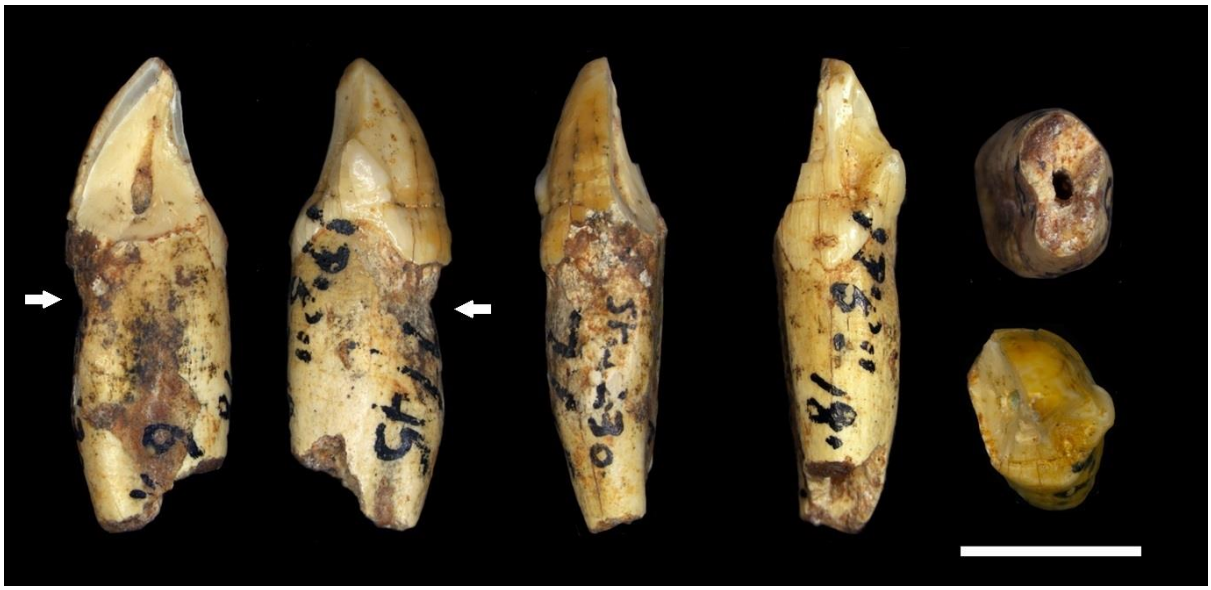

Figure 11.4. STW 213, mandibular right canine. Left to right: mesial, distal, labial, lingual; upper right: root tip; lower right: crown top.

The unusual wear on the root of STW 270 is very similar to a reported cause of cultural wear on a canine of a medieval individual from Ireland (Novak, 2015). The groove has a shallow and elongated groove starting from the CEJ and extending a significant way down the buccal surface of the root. Novak suggests this is caused by habitual activities, noting that it appears something was pulled over the teeth repeatedly and has potentially also removed calculus.

Dental erosion is caused by chemical destruction of dental tissue and has been documented as being caused by a variety of factors, most prominently acidic diets (Grippo et al., 2004; Indriati & Buikstra, 2001; Oginni et al., 2003; Ritter et al., 2009; Robb et al., 1991; Zero, 1996). The lesions on STW 213 and STW 270 look very similar to some examples of erosion. For example, Bader et al. (1993) highlight a case of erosion that looks very similar to that of STW 270, with a large lesion on the buccal root surface and then tapering to a point toward the lingual interproximal area, following the gingiva (Figure 11.5). Additionally, Grippo et al. (2012) highlight an example on an upper canine with the erosion lesion spreading a significant way down the buccal surface of the exposed root (Figure 11.5). The way the lesions on the two *A. africanus* specimen wrap around from the buccal surface onto the interproximal areas is suggestive that it was following the gingiva, making acid erosion the likely cause. If a tool or implement was used, it must have been used regularly on all surfaces, which seems unlikely. Further, the lesions vary in width yet stay remarkably smooth and uniform as they taper towards to lingual on the interproximal surfaces, again suggesting erosion is likely. Ritter et al. (2009) highlight some similar cases of cervical non-carious lesions, finding that they are most common in groups that regularly eat citrus fruits. High occlusal stresses may also be involved (Grippo et al., 2012), although an acidic component is still needed to create the lesions. Although the acid may have originated internally, from regurgitation of gastric acids, this is unlikely since this is usually associated with erosion of lingual tooth surfaces (Loch et al., 2013).

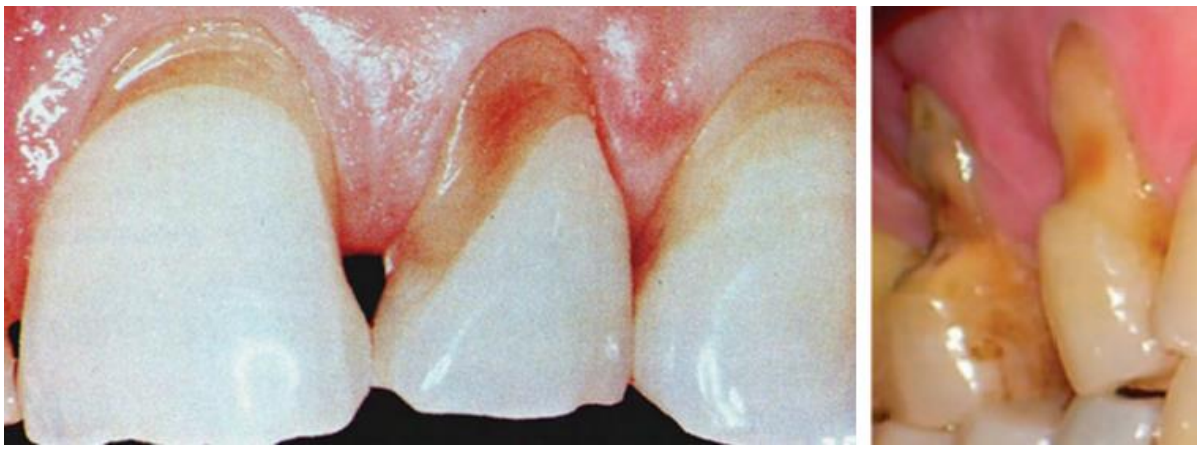

Figure 11.5. Acidic erosion on the root surface of living humans. Left photo: edited from Bader et al. (1993); right photo: edited from Grippo et al. (2012).

The lesions found on the two *A. africanus* teeth look indistinguishable from modern day clinical cases of dental erosion. The grooves also superficially look as if they could have resulted from abrasion through habitual use of an implement. However, the way the lesion wraps around the teeth strongly suggests all root surfaces exposed above the gum line were equally affected, and therefore acidic erosion is the much more likely cause. Additionally, the broad, shallow and smooth surfaces, as well as the lack of clear striations, adds support to this conclusion.

This unique case of erosive wear is the oldest hominin example of such a groove and was likely caused by an acidic diet, such as high concentrations of citric fruits or raw meat, with gingivitis probably facilitating its formation. The supposed LEH bands on only the right premolars and canine teeth suggest two likely possibilities, either the left teeth do not belong to the same individual or these are not, in fact, LEH defects. SEMs of these teeth may resolve this issue. However, one possibility is that these defects also relate, in some way, to the root lesions. Therefore, potentially the whole lower right side of the mouth was affected.

Chapter 12: Discussion, conclusions and future research

12.1. Discussion

Since in depth comparisons and discussions have been presented in each chapter, this section aims to: 1) briefly reiterate the individual findings, and 2) pull together the different variables to yield overall interpretations on the diet and behaviour of each hominin species. Comparisons are also made with other areas of research conducted on the South African hominins to try to reconstruct diet and behaviour. The overall results are highlighted in Table 12.1. The main conclusions that can be drawn when all variables are presented together (Table 12.1), is that *H. naledi* and *P. robustus* are significantly different from each other for the majority of dental pathologies under study. In fact, the only exception to this is tertiary dentine, which may represent a close phylogenetic relationship rather than dietary differences (Chapter 10).

Table 12.1. Frequencies (%) and wear results for the different variables studied. Chipping: % of chipped teeth; X: % multiple chips on chipped teeth; Size: number of small chips divided by large chips; PEH: pitting enamel hypoplasia; LEH: linear enamel hypoplasia; HR: hypoplasia rate (LEH/PEH); TD: % teeth with tertiary dentine; Wear slope: molar wear slope; Most worn: Most worn tooth type in the dentition.

Species	Chipping	X	Size	PEH	LEH	HR	Caries	TD	Slope*	MW
P. robustus	12.77	6.67	1.73	14.75	11.51	0.78	2.52	12	F	M1
A. africanus	21.13	16.07	10.2	5.03	15.08	3.00	0	13.33	I	M1
H. naledi	44.44	50	8.33	0.7	14.79	21.13	1.36	16.13	I/S	I1
Chimpanzees	4.92	2.04	2.27	0.65	8.06	12.40	8.29	47.21	S	I1
Gorillas	11.13	4.14	10.27	2.89	4.25	1.47	1.32	93.19	S	M1
Baboons	25	18.75	5.4	0	2.07	-	0.13	40.11	S	I1

*F: flat; S: steep; I: intermediate

Table 12.2 highlights some potential dietary conclusions that can be made from the caries, wear and chipping results. Up to three of the most appropriate/likely food types were selected based on the results for each variable (Chapters 5, 7 and 8; Burnett et al., 2013; Clement & Hillson, 2013; Constantino et al., 2010; Deter, 2009; Hillson, 2005; Scott & Winn,

2011). Although there is high variability among variables and species, overall it appears that *P. robustus* had a much more specialised diet than *A. africanus* and *H. naledi*. That said, the high rates of caries, and isotopic analysis (see below), for *P. robustus* suggest their diet may not have been as specialised as that of some extant primates.

Table 12.2. Up to three broad dietary categories that best fit each variable. H: hard foods, T: tough foods, F: fruits, V: low-quality vegetation matter, MD: mixed diet, SD: specialised diet.

Species	Caries	Wear	Chipping
P. robustus	F, V	SD, V, T	SD, V, T
A. africanus	T, H, MD	MD, F	H, MD
H. naledi	F, V	MD	H, MD

The enamel hypoplastic defects observed in the comparative modern human sample are far more severe than any other sample studied here, with the potential exception of *P. robustus*. Indeed, the modern human defects sometimes change the occlusal morphology, perhaps suggesting individuals with such severe episodes of disturbance during development were unlikely to survive in earlier hominins (Guatelli-Steinberg, 2015; Ogden et al., 2007; Pindborg, 1970). Other than the modern human samples, some of the most severe enamel defects are on the dentition of a female chimpanzee which likely had amelogenesis imperfecta (Chapter 11).

Carious lesions on seven teeth are described for the first time in this thesis bringing the total number of carious teeth to 12 for the South African hominin material as a whole (Chapter 7). Two of these belong to early *Homo*, eight to *P. robustus* and two to *H. naledi*. It is regularly proposed that caries is a recent disease and is scarce or absent in pre-agricultural hominins (Armelagos & Cohen, 1984; Brothwell, 1963; Guatelli-Steinberg, 2016; Hildebolt & Molnar, 1991; Lanfranco & Eggers, 2012; Tillier et al., 1995). The results of this thesis add to the growing evidence for the presence of carious lesions in a variety of non-agricultural hominin groups (e.g., Arnaud et al., 2016; Grine et al., 1990; Humphrey et al., 2014; Lacy, 2014; Lanfranco & Eggers, 2012; Trinkaus et al., 2000). Rather than a steady increase in caries through time the rate appears to be rather stable over the last two million years, with only an increase in specific groups.

Given the recent discovery of *H. naledi*, there is little in the way of dental literature in which to compare the results of this study (Berger et al., 2015; Cofran et al., 2016; M.M. Skinner et al., 2016). Microwear and isotopic analysis are planned to be performed by other researchers, so further comparisons will be possible. The age of the *H. naledi* material, only 236 ka to 335 ka years before present, raises the intriguing possibility that stone tools and other cultural artefacts found across Africa at this time may not necessarily have been created by *H. sapiens* and their direct ancestors (Berger et al., 2017; Dirks et al., 2017). Presumably given the young age of this material and their possible interaction with other hominins, they could have been processing foods, if not with fire then perhaps with certain tools (Berger et al., 2017). Evidently, *H. naledi* individuals differed regarding diet and behaviour compared with the other hominins considering the extremely high rate of dental chipping (Chapter 5). The presence of severe carious lesions contrasts with the baboon and *A. africanus* samples (Chapter 7). Additionally, *P. robustus* has considerably different wear patterns, hypoplasia rates, and chipping patterns than *H. naledi*. Once additional dietary information has been gathered for these specimens further insight can be gained into why such differences exist.

The high frequency of crown chipping in *H. naledi* stands out compared with the other species studied. Over 50% of *H. naledi* molars have at least one chip, with interproximal areas the most affected (Chapter 5). Many modern human samples with similarly high rates of chipping show different patterns of fractures. Anterior teeth are often more affected, and there are usually higher proportions of large chips, due to fractures commonly caused by non-masticatory behaviour (e.g., Scott & Winn, 2011; Turner & Cadien, 1969). However, there are some human parallels to *H. naledi* (Belcastro et al., 2007; Bonfiglioli et al., 2004; McManamon et al., 1986). Based on these sites with similar chipping patterns, the most likely cause of the high rate of chipping in *H. naledi* is grit incorporated into their diet. The conclusion that that grit is responsible rather than dietary items is also supported by the steep occlusal wear that is present in some of the *H. naledi* molars (Chapter 8; Brace, 1962; Hinton, 1981; Smith, 1984). Additionally, the chipping pattern is similar to that of baboons, in which grit is commonly masticated (Nystrom et al., 2004). How and why grit was regularly digested is not possible to determine. However, the most likely possibility is through the mastication of foods with

adhering grit, such as tubers and other underground storage organs. Carbohydrate-rich tubers could also potentially explain the severe carious lesions in this sample.

It has been suggested (Wallace, 1975) that because samples classified as early *Homo* (e.g., SK 15) have a much lighter build of mandible and thinner enamel, they must have processed much of their food before mastication. The cited author suggests that the job performed by the large teeth of *P. robustus* and *A. africanus* effectively was completed by stone tools in early *Homo*, using the association of stone tools as evidence. Since then it has been pointed out that chimpanzees and humans often deal with the issue of eating hard objects by dismantling them (e.g., seeds or nuts) prior to consumption (Boesch & Boesch, 1982; Wrangham et al., 2003). Daegling et al. (2013) argue that, therefore, food processing is extremely likely to have also been common in fossil hominins. Nothing stands out regarding cultural wear or occlusal attrition on the teeth of early *Homo* studied here. Heavy wear on the anterior teeth is present and likely represents food processing behaviours; however, heavy anterior teeth wear is also found in *H. naledi*, *A. sediba*, baboons, and chimpanzees (Chapter 8).

The small sample size of *A. sediba* also limits comparisons for many of the variables studied here. However, one conclusion that can be made is that the occlusal wear of this species least resembles that of *P. robustus* and is most similar to several *H. naledi* specimens (Chapter 8). The surprising isotopic results for *A. sediba*, with much higher rates of C3 foods than that apparently consumed by *A. africanus* and *P. robustus* (Henry et al., 2012), may, therefore, be interesting to compare with *H. naledi* once isotopic data for this species is available.

Out of all the South African hominin species *P. robustus* has likely received the most attention and debate regarding diet. This attention is because of their large posterior teeth and mandible, along with conflicting conclusions from microwear analysis (Daegling et al., 2013; Grine et al., 2012; Merceron et al., 2004; Scott et al., 2005; Ungar & Grine, 1991). The issue with the large masticatory apparatus is summarised (using *P. boisei* as an example) by Daegling et al. (2013). The bulkier appearance of the *Paranthropus* skull has been proposed to result from a greater reliance on hard foods (Peters, 1987), fibrous and tough foods (DuBrul, 1977), and a non-specialised diet similar to other hominin species but in more significant quantities (Walker, 1981). Indeed, it has been suggested that all three of these

suggestions may cause a similar effect on the cranial morphology (Daegling et al., 2013; Ravosa et al., 2007). This view is challenged by Strait et al. (2009, 2012) who suggest that the dental morphology in *Paranthropus* is not well adapted to processing tough foods and instead favours hard food objects as the evolutionary driving force behind their tooth form and size. Daegling et al. (2013) agree that the teeth of *Paranthropus* are not ideally suited to processing fibrous foods but point out that evolution can only act on features already present. Similar cases have been observed in extant primates in which species with similar diets have evolved a variety of dental solutions to similar stresses (e.g., Daegling & McGraw, 2007; Taylor, 2006). The results of the present study agree with Daegling et al. (2013), with *P. robustus* having a low rate of chipping suggesting infrequent consumption of hard foods (Chapter 5).

Comparisons with other primates may offer insight into the unique morphology of the *P. robustus* dentition. Large premolars in *P. robustus* commonly referred to as molarized, are also found in species that consume large volumes of bamboo, such as bamboo lemurs and giant pandas (Gittleman, 1994; Jernvall et al., 2008). Such a diet has also been suggested for an extinct close relative of orangutangs, *Gigantopithecus blacki*, due to also having molarized premolars (Daegling & Grine, 1994). Therefore a good hypothesis would be that in *P. robustus* large posterior teeth evolved due to a diet high in low quality tough vegetation and not hard foods.

Species that regularly eat hard foods also tend to have relatively large second premolars, although this is not always the case (Daegling et al., 2011). As noted by Daegling et al. (2011), *P. robustus* does not possess large second premolars compared with the first molars, relative to other primate species. It is true that they have very large premolars compared with other hominins but their molars are also very large so that the relative size of the second premolar is not as striking as it first appears. Therefore, the large premolars are potentially just a consequence of the same process that led to large molars, fitting with the suggestion of processing significant amounts of abrasive foods (Daegling et al., 2011).

A popular concept that has often been suggested to explain the large molars of *P. robustus* is "fall-back foods", which broadly refers to material consumed only when favoured foods are unavailable (Constantino & Wright, 2009; Harrison & Marshall, 2011; Lambert, 2009; Marshall & Wrangham, 2007). The term was originally used by primatologists but has also been used to explain dietary trends in hominins (Dominy et al., 2008; Laden &

Wrangham, 2005; Wrangham et al., 2009). Fallback foods are usually seen as lower in nutritional quality or at least tougher/harder than favoured foods (Constantino & Wright, 2009; Lucas et al., 2009). Although it cannot be ruled out that *P. robustus* evolved a large masticatory apparatus to process foods rarely consumed, evidence for such a hypothesis is lacking. Theoretically, such extreme morphology could evolve for this reason, particularly if individuals must rely on such foods for survival in crucial periods; that said, the apparatus cannot evolve to be overly wasteful. Therefore, if *Paranthropus* evolved large masticatory equipment, with molarized premolars and thick enamel, for processing hard foods, it would be expected that such teeth could sustain high levels or sizes of fractures. Therefore, at least occasionally large fractures would be expected, as well as high chipping frequencies compared with species that do not utilise such foods. As has been concluded above, no evidence was found for regular consumption of hard foods, given the very small number of chips on their teeth (Chapter 5). Furthermore, on the whole, hominins have thick enamel compared with other primates, and even in primates with hard food diets it is rare for large fractures to occur (Constantino et al., 2012). Therefore, it seems unlikely that such large masticatory equipment evolved in *Paranthropus* due to a diet of hard objects, either regularly consumed or as fallback foods.

Another suggestion worth mentioning is Liem's paradox, which refers to the observation that the most morphologically-derived cichlids (i.e., a species of fish) have a generalised diet (Liem, 1980; Robinson & Wilson, 1998). This relationship has been extended to hominins with the suggestion that certain species, particularly those in the genus *Paranthropus*, may have evolved distinguishing masticatory characteristics to permit consumption of a broad range of foods, rather than to a particular specialised diet (Wood & Schroer, 2012; Wood & Strait 2004). Such a process as Liem's paradox may certainly be involved in the evolution of the masticatory system of *P. robustus*; however, the results of this study do not support a mixed diet for *P. robustus*, at least compared to the other hominins and baboons (Chapters 5 and 8).

The most striking result of this study for *P. robustus* is the high frequency of PEH compared with the other hominin species as well as the extant primate samples (Chapter 6). Indeed, *P. robustus* shows far higher rates of PEH than almost all modern human samples studied (Goodman et al., 1987; Hillson, 2014; Seow et al., 1992). The PEH in *P. robustus*

predominantly seems to affect the posterior teeth, with only a couple of anterior teeth showing pitting. Over 20% of permanent molars and 50% of deciduous molars have PEH, suggesting these teeth were predisposed to this type of hypoplasia. The PEH on *P. robustus* teeth do not look like the defects published in many modern clinical studies, including those caused by amelogenesis imperfecta, fluorosis, low birth weight, vitamin D deficiency, tuberous sclerosis, congenital syphilis, pseudohypoparathyrgidism and epidermolysis bullosa (Aine et al., 2000; Crawford et al., 2007; Croft et al., 1965; Gaul et al., 2015; Ioannou et al., 2015; Nikiforuk & Fraser, 1979, 1981; Pinhasi et al., 2006; Purvis et al., 1973; Radu & Soficaru, 2016; Seow et al., 1984; Stimmler et al., 1973; Wright et al., 1993). Instead, potential influences for the high rate of PEH in *P. robustus* may include molar morphology, genetic factors, developmental timing, calcium deficiency and acute disturbance during a crucial period of development (Chapter 6). The scarcity of PEH defects on anterior teeth and the uniform nature of the defects on molars across individuals suggest all defects, both on deciduous and permanent teeth, likely resulted from the same aetiology.

Paranthropus robustus and gorillas have much in common with many of the variables under study sharing similar frequencies (Chapters 5, 7 and 8), perhaps due to eating large quantities of low-quality vegetation. In particular, the wear is similar between these two species, with both having similar chipping rates and first molars the most worn. Interestingly the proportion of PEH to LEH is also similar between these two species, although *P. robustus* has much higher overall rates. This similarity is perhaps explained by the lack of a nutritional element, with generalists such as baboons having extremely low levels of PEH. Pitting enamel hypoplasia, therefore, may be related to a lack of dietary breadth, although more research is needed to test this possibility.

Paranthropus boisei has not been studied in depth for many of the present variables, and given its supposed close phylogenetic relationship to *P. robustus*, it is worth comparing. It is often reported that *P. boisei* has high levels of dental fractures, or otherwise chipping is mentioned as a component to back up a statement about this species being a hard food object specialist (e.g., Martínez et al., 2016; Constantino et al., 2010). However, such a study has not been conducted; instead specific instances of chipping have been described, but no frequencies for this species has been published (Tobias, 1967). Therefore, the presence of chipping in *P. boisei* cannot be used as evidence for hard object feeding until a study on its

prevalence has been undertaken; primate species that rarely eat hard foods still have a few examples of chipped teeth (Chapter 5). Fitting with the result of this study, it is therefore probable that the masticatory apparatus in *Paranthropus* as a whole evolved for processing tough vegetation rather than hard foods (Cerling et al., 2011; Rabenold & Pearson, 2011).

Two other similarities can be suggested between *P. robustus* and *P. boisei* from the results of this thesis. Tobias (1967) notes that OH 5 (*P. boisei*), has asymmetric wear on the maxillary anterior teeth, caused by mal-alignment with the mandibular teeth. Such asymmetry and malocclusion have been highlighted in *P. robustus* as well (Chapter 8). Tobias (1967) also notes enamel hypoplasia in *P. boisei* that looks similar to that in *P. robustus* (Chapter 7). However, given the purported close relationship between these species, it may be difficult to infer if similar results are due predominantly to a shared diet or simply a close phylogenic relationship. Further analysis of the *P. boisei* material as well as that on the hypoplastic defects may help clarify this. One suggestion for the high levels of PEH in *P. robustus* is that it may just be a unique morphology for this species, and is not caused by a stress episode like LEH. The issue with this hypothesis is that the enamel hypoplasia is associated with several cases of caries (Chapter 7) and therefore the evolutionary pressures to prevent such pitting would evidently be high. Additionally, wear on these teeth would presumably have been much quicker than if pitting was not present, potentially limiting the life of the tooth. Once *P. boisei* has been studied for PEH, further insight into these defects may be possible.

The debate around the diet of *A. africanus* has been intensive in recent years with microwear, isotopic, biomechanical and chipping data all used to infer different points of view (e.g., Grine et al., 2010; Scott et al., 2005; Sponheimer et al., 2005, 2013; Strait et al., 2009; Van Der Merwe et al., 2003). It appears that the prediction of Strait et al. (2012) is correct in that *A. africanus* does have a high chipping rate, particularly compared with *P. robustus* and the extant great apes studied here (Chapter 5). Additionally, the premolars are the most affected tooth group with almost one third displaying at least one chip. This high rate of chipping fits with the hypothesis by Strait et al. (2009) that facial buttressing features are adaptations to large food processing in the premolar region. However, it should be stressed that very few of these chips are large and so unusually large and hard objects seem unlikely to have been regularly consumed. The evidence from chipping frequencies here does support

at least the conclusion that the premolars were an essential part of the masticatory process for *A. africanus* and perhaps indicates tough and hard foods were regularly masticated in this region of the mouth.

Until recently, it was assumed that *A. africanus* likely had a similar diet to that of extant chimpanzees, though since the use of isotopic and microwear analysis this opinion has changed. Isotopic analysis by Sponheimer and Lee-Thorp (1999) found that individuals were consuming much higher levels of carbon-13–enriched foods meaning that a diet consisting mainly of fruits and leaves was unlikely. Since then more samples have been analysed, and a similar result produced (Sponheimer et al., 2005, 2013; Van Der Merwe et al., 2003). As well as grasses and sedges, the consumption of certain insects and vertebrates could give such an isotopic signal (Sponheimer et al., 2013). Microwear analysis of *A. africanus* teeth is also suggestive of a variable diet that contains tough foods (Scott et al., 2005). Whether the *A. africanus* diet also included a significant amount of hard foods is debated in the literature (Grine et al., 2010; Strait et al., 2009). As noted above, the chipping results from this thesis suggest hard foods were commonly consumed. The fact that this species has the largest sample of teeth, yet no caries also suggests a varied diet or at least one low in cariogenic foods; it also supports the consumption of tough foods. The results of this thesis, therefore, further support the conclusion that *A. africanus* had an extremely mixed diet (Chapters 5, 7, and 8).

Dental pathologies are mostly seen as a nuisance in modern times, but in the past high mortality was often associated with several of these conditions (Calcagno & Gibson, 1991). Therefore, high selective pressures may be placed on populations to prevent dental pathology and excessive wear (Hillson, 1996; Molnar, 1971). Most notably, it has been suggested that evolving smaller teeth may reduce the instances of caries and that large teeth allow further resistance to substantial attrition (Calcagno & Gibson, 1991; Lacy, 2014). Both of these examples seem logical at face value, but evidence for such a hypothesis is debated. An issue with the caries suggestion has been raised by Brace et al. (1991) in that the bulk of the reduction seen in hominin tooth size happens before caries frequencies rise dramatically (in certain populations) in the last 10,000 years. Caries does not seem to increase significantly over the last two million years; therefore it seems unlikely that caries on its own explains the reduction of the jaws seen in the genus *Homo* (Chapter 6).

During the middle of the 20th century, the most prominent hypothesis regarding differences observed between *P. robustus* and *A. africanus* was the dietary hypothesis, as argued by Robinson (e.g., Robinson, 1954). The hypothesis suggests that due to environmental change, these species were specialised in different ecological niches. The robust species (*P. robustus*) focussed on vegetation in forested locations whereas the gracile species (*A. africanus*) were more omnivorous in open habitats. Other hypotheses were subsequently put forward. For example, Cachel (1975) suggests that dental differences between *P. robustus* and *A. africanus* are due to allometry rather than diet. It has also been common to promote the idea of Plio-Pleistocene hominins as mostly carnivorous, and even cannibalistic (e.g., Ardrey, 1961; Dart, 1960; Etkin, 1954; Howell, 1963; Washburn, 1959). As Pickering et al. (2004) point out, opinion began to change when researchers such as Brain (1993) started to highlight that these individuals were likely the prey of large carnivores and therefore not near the top of the food chain. The results of this thesis support the suggestion that *P. robustus* filled a different ecological niche than *A. africanus*, given the vast differences in individual variables (Chapters 5, 6, 7, and 8). The morphology of the crania of *P. robustus* and *A. africanus* also suggests different diets (DuBrul, 1977; Rak, 1983; Strait et al., 2009).

Carbon isotope values between *A. africanus* and *P. robustus* have not been found to be significantly different, with much overlap among species (Sponheimer & Lee-Thorp, 1999, 2003). However, *P. robustus* specimens seem to be less variable (Sponheimer et al., 2005). What can be said for sure is that both *P. robustus* and *A. africanus* are significantly distinct from C3 and C4 specialising bovids, giraffids, chalicotherids, and suids in terms of diet (Sponheimer et al., 2005). Given the differences highlighted between *P. robustus* and *A. africanus* (Chapters 5, 6, 7, and 8*)*, it may be likely that although they had similar levels of C3 and C4 foods in their diet, the actual foods consumed differed substantially.

12.2. Conclusions

12.2.1. Answers to research questions

Can prior studies of diet in these various hominins based on microwear, isotopic analysis etc. be supported or refuted by the study of basic dental pathological indicators?

The results of this study do not contradict previous results based on microwear or isotopic analysis. Indeed, they complement such research and further narrow the possible diets of these hominins. For example, it is known that *P. robustus* and *A. africanus* had a mixed C3 and C4 diet, but based on chipping frequencies it is unlikely that hard food items were a common component, especially in *P. robustus*. Comparisons between results from other dietary reconstructing techniques and those in this study are presented in the discussion (Chapter 12.1).

Can pathology, wear, and enamel defect data suggest differences in diet between the South African hominins?

Yes, clear differences are apparent among the variables studied, particularly in rates and forms of chipping, wear and caries in South African species. This evidence, therefore, highlights clear dietary and/or behavioural differences (Chapters 5, 6, 7 and 8).

Is there a link between the different variables and species? That is, are there certain species which consistently have similar patterns of wear and pathologies?

This seems to be the case for certain species. For example, *A. africanus* has much in common with baboons, with several variables showing similar patterns (Chapters 5, 7, 8 and 12). Similarly, *P. robustus* and gorillas have much in common, especially wear and hypoplasia patterns (Chapters 6 and 8). These results likely reflect dietary and behavioural similarities between these species, with gorillas and *P. robustus* specialising in low-quality vegetation and baboons and *A. africanus* sharing a broader diet (Chapter 12).

Do dental pathology and wear data fit with known ecological information on the extant primate samples, and if so what diet is, therefore, most applicable to the different hominin species?

The answer seems to be ‘yes’ for each extant primate species, with no surprising results in terms of overall frequencies (see variable chapters). This, as well as comparisons with human samples in the literature, suggest that dietary inferences are possible for the fossil hominin species. For example, high levels of environmental grit were likely consumed by *H. naledi*, based on comparisons with baboons and archaeological human samples (Chapter 5). Low levels of hard foods and high levels of low-quality vegetation were likely

present in the diet of *P. robustus*, based on comparisons with gorillas and chimpanzees (Chapters 5, 8 and 12). *Australopithecus africanus* likely had a broad diet with high quantities of tough non-cariogenic foods, similar to that of extant baboons (Chapters 5, 7 and 12).

Are differences observed between the different hominin species substantial enough to infer different ecological niches?

It is difficult to answer this question given the way in which ecological niches are defined. Certainly, there are clear differences in diet and/or behaviour of certain species, for example between *P. robustus* and *H. naledi*. Whether a consequence of a completely different way of life or simply a result of the environment at the time is hard to infer. However, the vast differences in the frequencies of different variables, often covering the entire range of the extant primates, suggests the different South African hominin species differed significantly in terms of food accusation or processing behaviours.

12.2.2. Overall conclusions

From this project as a whole, the main conclusion is that South African hominins vary substantially with regard to frequencies of dental pathologies, developmental defects and wear. Indeed, the variance is often more than that observed among the extant primate species, in which diet and behaviour are known to differ significantly.

Paranthropus robustus has far higher levels of PEH than any other hominin species yet studied. Molars, both deciduous and permanent, are affected. Considering anterior teeth show few defects, molars are likely predisposed to PEH. The high rate of PEH on molars may, therefore, be related to enamel properties, in that the slightest disturbance creates what superficially look like severe defects. However, another possibility is that these defects are genetic in origin or simply a morphological feature resulting from the evolution of large and thick enamelled molars. Specific environmental or dietary influences may also have been involved. Chipping and wear results best support a conclusion that *P. robustus* had a diet low in hard foods; the species may instead have eaten substantial amounts of low-quality tough food items. The presence of caries suggests that dietary items may have also included cariogenic foods such as fruit.

If it is accepted that this 'golf ball' pattern of hypoplasia found commonly in *P. robustus* is unique to *Paranthropus* (Chapter 6), or at least exceedingly rare in other hominins, then SKX 1756 may be better described as *P. robustus*, rather than early *Homo*. The classification of this specimen is debated, and a significant amount of postmortem damage means little is available to use for comparisons. Therefore, this PEH may be useful information to incorporate into assigning a species for this specimen.

The main finding for *H. naledi* is the large amount of antemortem chipping. All individuals that have multiple worn teeth are affected, with half of posterior teeth having one or more chips. The fact that posterior teeth are more affected than anterior, and interproximal areas more so than buccal, suggests a dietary or environmental cause rather than non-masticatory. The most similar chipping patterns found in humans result from the incorporation of grit in their diet. The small size of most chips on *H. naledi* teeth adds further support that grit was masticated. Grit must have been regularly incorporated into their diet; perhaps due directly to environmental factors, however more likely it was included through eating certain foods. Tubers and other underground storage organs can introduce a substantial amount of sediment into the oral cavity if consumed in large quantities. The presence of severe carious lesions may also suggest that cariogenic foods were commonly consumed.

Perhaps what stands out most about *A. africanus* is its tendency not to stand out. It has no carious lesions, the second most hypoplastic defects, unremarkable wear and the third highest level of chipping. All pathologies and wear support a conclusion that this species was a generalised feeder. The lack of caries suggests a non-cariogenic fibrous diet and supports the idea that a variety of foods were consumed. As well as having the most similar caries rate to baboons, *A. africanus* also has the most similar chipping rates to this modern primate. A mixed diet is also supported by isotopic and microwear analysis, in which a mix of foods is the best-supported conclusion for this species. Root grooves were also described on two *A. africanus* teeth, STW 270 and STW 213. These are thought to be adjacent teeth belonging to the same individual. The lesions best fits with an aetiology of erosive wear. These depressions in the roots suggest that an acidic dietary component, such as fruits, may have regularly been consumed. It is also likely this individual suffered from periodontal disease, which facilitated root exposure leading to this isolated pocket of erosion. If correct, then this is the first case

of extensive acidic removal of dental material described in the South African hominin material, with the only comparable examples belonging to recent *Homo.*

Although the extant primate samples were predominantly studied to allow comparisons with the fossil hominins, some interesting results are worth highlighting. Female chimpanzees have five times the amount of carious lesions than do males, due predominantly to interproximal caries on the incisors. This high rate is likely caused by the way they process foods. Male chimpanzees have significantly more chips than females, further demonstrating sex differences in the diet of this species. These differences add further caution to describing diet based on a small sample consisting predominantly of one sex. The first case of amelogenesis imperfecta in an extant primate was also described, which will hopefully lead to further examples of these genetic disorders to be explored in primates in general. The main unexpected finding for gorillas was the rate of tertiary dentine. Almost all teeth with sufficiently worn enamel evidence tertiary dentine formation. Such a high rate may suggest that gorillas have evolved to produce tertiary dentine faster or earlier in response to wear, likely to keep an appropriate cutting edge during mastication.

12.3. Future research

It is proposed that additional studies relating to wear and pathologies be undertaken, to provide further insight into the diet and behaviour of the South African hominins. Studies on other populations and other dietary reconstructing techniques would also allow further comparative material for this study. For example, similar data to that collected here but on other hominin samples, as well as a broader range of primates in general, would provide further comparative information from which further conclusions could be made. A few specific areas of research are explored below.

The use of a Scanning Electron Microscope (SEM) would be extremely informative for a number of different reasons. For STW 270 and STW 213, a SEM of these teeth would allow further conclusions on the cause of the dental erosion and may allow abrasion to be completely ruled out. Additionally, further work on the supposed LEH bands on the right premolars and canine of STW 213 would be interesting, as these grooves do not have corresponding antimeres. It is likely that similar lesions to those found on STW 213 and STW 270 are overlooked in other collections of hominins. Once similar grooves are discovered it

will be possible to further explore the causes for this rare type of wear. Micro CT-scans of different features would also be informative. For example, there are potential carious lesions that may be identified using CT-scans (Chapter 7), pitting enamel hypoplasia could be explored further with more specimen CT-scans (Chapter 6), and enamel property differences between different sections of teeth could be further explored (Chapters 5 and 8; see below).

Differences between chipping rates on right and left teeth were found in *H. naledi*. However, this relationship was not statistically significant (Chapter 5). Further *H. naledi* material has since been described, and there are more individuals to excavate (Hawks et al., 2017). Therefore, when further material is studied, it may allow further investigation into these side differences. Microwear analysis may also provide an interesting insight.

Enamel properties differ among primate species, although little research has explored why such differences exist. For example, the occlusal wear variation between the buccal and lingual halves of posterior teeth is well known, yet surprisingly the opposite pattern is observed for enamel fractures. This means that the surface with the highest wear is the one that shows the least amount of antemortem dental fractures, which would appear counter-intuitive. This relationship seems consistent across all extant primates and fossil hominins in this sample, and it would, therefore, be interesting to explore this relationship. A study that includes enamel property differences such as density and structure, masticatory and behavioural information, along with wear and chipping data would likely show insightful conclusions. Micro CT-scans can be used to analyse a variety of enamel properties, including how enamel density varies over the crown of a tooth (e.g., Swain & Xue, 2009; Ziscovici et al., 2014; He et al., 2011). Masticatory and behavioural information has been published for a variety of extant primates as well as fossil hominins (e.g., Conklin-Brittain et al., 2001; Constantino et al., 2010; Harrison & Marshall, 2011; Rogers et al., 2004; Strait et al., 2009; Taylor, 2006; Wright et al., 2008). Therefore the methodology required to gather the enamel property data is already available and the comparative masticatory and behavioural information already published. Consequently, the study would require analysis of micro-CT scans of different primate species along with chipping and wear data, as well as biomechanical and behavioural information from the literature, for these same species. It will then be possible to see how wear and chipping rates vary in relation to enamel properties and how this relationship varies between species with different diets and dental morphology.

Therefore further insight into why hominin specimens vary in terms of dental wear and chipping would be possible.

As highlighted in this study, the small number of dental fractures found in *P. robustus* compared with other hominins, and extant primates, adds support to suggestions that this species did not specialise in eating hard food objects. Interestingly, the smallest toothed hominin sample studied, *H. naledi*, had the highest fracture rate, four times that of *P. robustus*. Therefore, it would be interesting to build on this to see if there is a correlation between reduction of tooth size and chipping rate. This could be achieved either through collecting more hominin data or through an extensive primate comparative study. For example, *H. naledi* has very simple morphological features and relatively thick enamel. One potential evolutionary suggestion, therefore, could be that perhaps this evolved as a mechanism to protect against high quantities of contaminants or hard foods in their diet. Chipping data is only available for a few species of primates and recording methods are often different (e.g., Constantino et al., 2012; Morse et al., 2013). Therefore, the most appropriate way forward would be to collect more chipping data using the same methods as this thesis but on a larger sample of hominins and extant primates and then to collect tooth size data during data collection, or use already published measurements.

It would also be interesting to explore other fossil primates, such as the baboons found in the South African caves in which hominin material has been discovered (Codron et al., 2005; Pickering et al., 2004; Pickering & Carlson, 2002). In the results of this thesis, there are some similarities between extant baboons and South African hominins, such as chipping rate with *H. naledi* and caries with *A. africanus* (Chapters 5 and 7). Therefore, it would be interesting to explore if wear, pathologies and enamel properties of these fossil baboons may provide further insight into these comparisons.

In sum, it is often noted that dental pathologies are rare in fossil hominins. Moreover, macrowear and enamel defects are given little attention in the literature. However, researchers have begun to show examples of different pathologies, and now a wide variety of lesions and defects have been described in various hominin species. This thesis builds on this research by describing additional carious lesions, abscesses, and instances of periodontal disease. Additionally, rates of tertiary dentine, antemortem chipping and occlusal wear highlight how variable hominin species are, with rates of the latter two encompassing the

entire range of the extant primate samples. In particular the high level of chipping in *H. naledi* and pitting enamel hypoplasia in *P. robustus* stand out, with rates as high as the most extreme cases found in modern humans. These different variables offer intriguing insight into the diet and behaviour of fossil hominins, and between them cover a much broader time scale of an individual's life than specific studies such as microwear or isotopic analysis. The main conclusion from this research is, therefore, the high variability in nearly all variables between hominin species, suggesting significantly different diets were consumed. It is hoped these macroscopic observations of wear, pathologies and defects can be used alongside other dietary reconstructing techniques to allow inferences into the ecological niches these hominins would have filled.

References

Aguirre, E., (1970). Identification de "*Paranthropus*" en Makapansgat. Cronica del XI Congreso Nacional de Arqueologia, 98-124, Merida.

Aine, L., Backström, M. C., Mäki, R., Kuusela, A. L., Koivisto, A. M., Ikonen, R. S., & Mäki, M. (2000). Enamel defects in primary and permanent teeth of children born prematurely. *Journal of Oral Pathology & Medicine*, *29*(8), 403-409.

Albarella, U. (2006). Pig husbandry and pork consumption in medieval England. In: Woolgar, C.M., Serjeantson, D., Waldron, T. (Eds.), *Food in Medieval England*, 72-87. Oxford: Oxford University Press.

Aldred, M. J., Savarirayan, R., & Crawford, P. J. M. (2003). Amelogenesis imperfecta: a classification and catalogue for the 21st century. *Oral Diseases*, *9*(1), 19-23.

Al-Hashimi, N., Sire, J. Y., & Delgado, S. (2009). Evolutionary analysis of mammalian enamelin, the largest enamel protein, supports a crucial role for the 32-kDa peptide and reveals selective adaptation in rodents and primates. *Journal of Molecular Evolution*, *69*(6), 635.

Alvarez, J. A., Rezende, K. M. P., Marocho, S. M. S., Alves, F. B. T., Celiberti, P., & Ciamponi, A. L. (2009). Dental fluorosis: exposure, prevention and management. *Journal of Clinical and Experimental Dentistry*, 1(1), 14-18.

Ånerud, K. E., Robertson, P. B., ÅNerud, H. L., Boysen, H., & Patters, M. R. (1983). Periodontal disease in three young adult populations. *Journal of Periodontal Research*, *18*(6), 655-668.

Ardrey, R. A. (I96I). *Africangenesis*. New York: Atheneum Press.

Armelagos, G. J., & Cohen, M. N. (Eds.). (1984). *Paleopathology at the Origins of Agriculture*. Cambridge: Academic Press.

Arnaud, J., Benazzi, S., Romandini, M., Livraghi, A., Panetta, D., Salvadori, P. A., ... & Peresani, M. (2016). A Neanderthal deciduous human molar with incipient carious infection from the Middle Palaeolithic De Nadale cave, Italy. *American Journal of Physical Anthropology*, 162(2), 370–376.

Backwell, L. R., & d'Errico, F. (2001). Evidence of termite foraging by Swartkrans early hominids. *Proceedings of the National Academy of Sciences*, 98(4), 1358-1363.

Bader, J. D., Levitch, L. C., Shugars, D. A., Heymann, H. O., & McClure, F. (1993). How dentists classified and treated non-carious cervical lesions. *The Journal of the American Dental Association*, *124*(5), 46-54.

Bajaj, D., Nazari, A., Eidelman, N., & Arola, D. D. (2008). A comparison of fatigue crack growth in human enamel and hydroxyapatite. *Biomaterials*, *29*(36), 4847-4854.

Belcastro, G., Rastelli, E., Mariotti, V., Consiglio, C., Facchini, F., & Bonfiglioli, B. (2007). Continuity or discontinuity of the life-style in central Italy during the Roman imperial age-early middle ages transition: Diet, health, and behavior. *American Journal of Physical Anthropology*, *132*(3), 381-394.

Berger, L. (2012). *Australopithecus sediba* and the earliest origins of the genus *Homo*. *Journal of Anthropological Sciences*, *90*, 117-131.

Berger, L. R., & Clarke, R. J. (1995). Eagle involvement in accumulation of the Taung child fauna. *Journal of Human Evolution*, *29*(3), 275-299.

Berger, L. R., De Ruiter, D. J., Churchill, S. E., Schmid, P., Carlson, K. J., Dirks, P. H., & Kibii, J. M. (2010). *Australopithecus sediba*: a new species of *Homo*-like australopith from South Africa. *Science*, *328*(5975), 195-204.

Berger, L. R., De Ruiter, D. J., Steininger, C. M., & Hancox, J. (2003). Preliminary results of excavations at the newly investigated Coopers D deposit, Gauteng, South Africa: preliminary research reports: human origins research in South Africa. *South African Journal of Science*, *99*(5-6), 276-278.

Berger, L. R., Hawks, J., de Ruiter, D. J., Churchill, S. E., Schmid, P., Delezene, L. K., ... & Skinner, M. M. (2015). *Homo naledi*, a new species of the genus *Homo* from the Dinaledi Chamber, South Africa. *Elife*, *4*, e09560.

Berger, L. R., Hawks, J., Dirks, P. H., Elliott, M., & Roberts, E. M. (2017). *Homo naledi* and Pleistocene hominin evolution in subequatorial Africa. *Elife*, *6*, e24234.

Bermudez de Castro, J. M., Arsuaga, J. L., & Perez, P. J. (1997). Interproximal grooving in the Atapuerca-SH hominid dentitions. *American Journal of Physical Anthropology*, *102*(3), 369-376.

Bignozzi, I., Crea, A., Capri, D., Littarru, C., Lajolo, C., & Tatakis, D. N. (2014). Root caries: a periodontal perspective. *Journal of Periodontal Research*, *49*(2), 143-163.

Bjørndal, L. (2001). Presence or absence of tertiary dentinogenesis in relation to caries progression. *Advances in Dental Research*, *15*(1), 80-83.

Boaz, N. T., & Clark Howell, F. (1977). A gracile hominid cranium from Upper Member G of the Shungura Formation, Ethiopia. *American Journal of Physical Anthropology*, *46*(1), 93-108.

Boesch, C., & Boesch, H. (1982). Optimisation of nut-cracking with natural hammers by wild chimpanzees. *Behaviour*, 83(3/4), 265-286.

Boesch, C., Uehara, S., & Ihobe, H. (2002). Variations in chimpanzee-red colobus interactions. In: Boesch, B., Marchant, L. F., Hohmann, G. (Eds.), *Behavioural diversity in chimpanzees and bonobos*. Cambridge: Cambridge University Press, 221-230.

Bombin, M. (1990). Transverse enamel hypoplasia on teeth of South African Plio-Pleistocene hominids. *Naturwissenschaften*, *77*(3), 128-129.

Bonfiglioli, B., Mariotti, V., Facchini, F., Belcastro, M. G., & Condemi, S. (2004). Masticatory and non-masticatory dental modifications in the epipalaeolithic necropolis of Taforalt (Morocco). *International Journal of Osteoarchaeology*, *14*(6), 448-456.

Borrell, L. N., Beck, J. D., & Heiss, G. (2006). Socioeconomic disadvantage and periodontal disease: the Dental Atherosclerosis Risk in Communities study. *American Journal of Public Health*, *96*(2), 332-339.

Bouchneb, L., & Maureille, B. (2004). Sillons d'usure interproximaux: reproduction expérimentale, analyse et application des résultats aux observations sur la lignée néandertalienne. *Bulletins et Mémoires de la Société d'Anthropologie de Paris*, 16 (1-2), 37-48.

Boughner, J. C., Der, J., & Kuykendall, K. L. (2015). A multivariate approach to assess variation in tooth mineralization using free-lived and captive-raised chimpanzees (*P. troglodytes*). *American Journal of Physical Anthropology*, 158(3), 452-462.

Bowman, J. E. (1991). Life history, growth and dental development in young primates: a study using captive rhesus macaques (unpublished PhD thesis), University of Cambridge.

Brabant, H. (1966). Contribution to the knowledge of the pathology of the teeth and jaws among the early populations of Belgium and the Nord region of France. The ossuary of Marville (Meuse, France). *Bulletin du Groupement International Pour la Recherche Scientifique en Stomatologie*, *9*(2), 224.

Brace, C. L. (1962). Cultural factors in the evolution of the human dentition. In Montague, M.F. A. (Ed.), *Culture and the evolution of man*, 343–354. New York: Oxford University Press.

Brace, C. L., Smith, S. L., & Hunt, K. D. (1991). What big teeth you had grandma! Human tooth size, past and present. In: Kelley, M. A., & Larsen, C. S. (Eds.), *Advances in dental anthropology*, 33-57. New York: Wiley-Liss.

Braga, J., & Thackeray, J. F. (2003). Early *Homo* at Kromdraai B: probabilistic and morphological analysis of the lower dentition. *Comptes Rendus Palevol*, *2*(4), 269-279.

Brain, C. K. (1969). The probable role of leopards as predators of the Swartkrans *australopithecines*. *The South African Archaeological Bulletin*, *24*(95/96), 170-171.

Brain, C. K. (1970). New finds at the Swartkrans *australopithecine* site. *Nature*, *225*(5238), 1112-1119.

Brain, C. K. (1983). *The hunters or the hunted? An introduction to African cave taphonomy*. Chicago: University of Chicago Press.

Brain, C. K. (1989). The evidence of bone modification by early hominids in southern Africa. In: Bonnichsen, R., & Sorg, M. H., (Eds.), *Bone Modification*, 291–297. University of Maine, Orono.

Brain, C. K., & Shipman, P. (1993). The Swartkrans bone tools. In: Brain, C.K. (Ed.), *Swartkrans: A cave's chronicle of early man*, 195-215. Transvaal Museum, Pretoria.

Brain, C. K., & Sillent, A. (1988). Evidence from the Swartkrans Cave for the earliest use of fire. *Nature*, *336*(6198), 464-466.

Bramblett, C. A. (1967). Pathology in the Darajani baboon. *American Journal of Physical Anthropology*, *26*(3), 331-340.

Branch-Mays, G. L., Dawson, D. R., Gunsolley, J. C., Reynolds, M. A., Ebersole, J. L., Novak, K. F., ... & Novak, M. J. (2008). The effects of a calorie-reduced diet on periodontal inflammation and disease in a non-human primate model. *Journal of Periodontology*, *79*(7), 1184-1191.

Braunn, P. R., Ribeiro, A. M., & Ferigolo, J. (2014). Microstructural defects and enamel hypoplasia in teeth of *Toxodon Owen*, 1837 from the Pleistocene of Southern Brazil. *Lethaia*, *47*(3), 418-431.

Broadbent, J. M., & Thomson, W. M. (2005). For debate: problems with the DMF index pertinent to dental caries data analysis. *Community Dentistry and Oral Epidemiology*, *33*(6), 400-409.

Bromage, T. G., & Dean, M. C. (1985). Re-evaluation of the age at death of immature fossil hominids. *Nature*, 317(6037), 525-527.

Broom, R. (1936). A new fossil anthropoid skull from South Africa. *Nature*, *138*(3490), 486-488.

Broom, R. (1938). The pleistocene anthropoid apes of South Africa. *Nature*, *142*(3591), 377-379.

Broom, R. (1949). Sterkfontein ape-man Plesianthropus. Pretoria: Transvaal Museum.

Broom, R. (1950). The genera and species of the South African fossil ape-men. *American Journal of Physical Anthropology*, *8*(1), 1-14.

Broom, R., Schepers, G. W. H., & Schepers, G. W. H. (1946). The South African fossil ape-men: the *Australopithecinae* (No. 2). Pretoria: Transvaal Museum.

Brothwell, D. R. (1963). *Digging up bones*. New York: Cornell University Press.

Brothwell, D. R. (1981). *Digging up bones: the excavation, treatment, and study of human skeletal remains*. New York: Cornell University Press.

Brown, P., Sutikna, T., Morwood, M. J., Soejono, R. P., Saptomo, E. W., & Due, R. A. (2004). A new small-bodied hominin from the Late Pleistocene of Flores, Indonesia. *Nature*, 431(7012), 1055-1061.

Brown, T., & Molnar, S. (1990). Interproximal grooving and task activity in Australia. *American Journal of Physical Anthropology*, *81*(4), 545-553.

Bruxelles, L., Clarke, R. J., Maire, R., Ortega, R., & Stratford, D. (2014). Stratigraphic analysis of the Sterkfontein StW 573 *Australopithecus* skeleton and implications for its age. *Journal of Human Evolution*, *70*, 36-48.

Burnett, S. E., Irish, J. D., & Fong, M. R. (2013). 21 Wear's the problem? Examining the effect of dental wear on studies of crown morphology. In: Scott, G.R., Irish, J.D. (Eds). *Anthropological perspectives on tooth morphology: genetics, evolution, variation*, 535–554. Cambridge: Cambridge University Press.

Byun, R., Nadkarni, M. A., Chhour, K. L., Martin, F. E., Jacques, N. A., & Hunter, N. (2004). Quantitative analysis of diverse *Lactobacillus* species present in advanced dental caries. *Journal of Clinical Microbiology*, *42*(7), 3128-3136.

Cachel, S. (1975). A new view of speciation in *Australopithecus*. In: Tuttle, R. H. (Ed.), *Paleoanthropology, morphology and paleoecology*, 183-201. The Hague: Mouton.

Caglar, E., Kuscu, O. O., Sandalli, N., & Ari, I. (2007). Prevalence of dental caries and tooth wear in a Byzantine population (13th CAD) from northwest Turkey. *Archives of Oral Biology*, *52*(12), 1136-1145.

Calcagno, J. M., Cope, D. A., Lacy, M. G., Moggi-Cecchi, J., & Tobias, P. V. (1999). Reinvestigating the number of hominid species in Sterkfontein Member 4. *American Journal of Physical Anthropology*, 28, 101-101.

Calcagno, J. M., & Gibson, K. R. (1991). Selective compromise: evolutionary trends and mechanisms in hominid tooth size. In: Kelley, M. A., & Larsen, C. S.,(Eds), *Advances in Dental Anthropology*, 59-76. New York: Wiley-Liss.

Carlson, K. B., de Ruiter, D. J., DeWitt, T. J., McNulty, K. P., Carlson, K. J., Tafforeau, P., & Berger, L. R. (2016). Developmental simulation of the adult cranial morphology of *Australopithecus sediba*. *South African Journal of Science*, *112*(7-8), 1-9.

Carlson, K. J., Stout, D., Jashashvili, T., De Ruiter, D. J., Tafforeau, P., Carlson, K., & Berger, L. R. (2011). The endocast of MH1, *Australopithecus sediba*. *Science*, *333*(6048), 1402-1407.

Carranza, J., Alarcos, S., Sánchez-Prieto, C. B., Valencia, J., & Mateos, C. (2004). Disposable-soma senescence mediated by sexual selection in an ungulate. *Nature*, *432*(7014), 215-218.

Carter, M. L. (2001). *Sensitivity of stable isotopes (13C, 15N, and 18O) in bone to dietary specialization and niche separation among sympatric primates in Kibale National Park, Uganda*. Doctoral dissertation, University of Chicago, Department of Anthropology.

Cawson, R. A., & Odell, E. W. (2002). *Cawson's Essentials of Oral Medicine and Pathology*. Philadelphia: Churchill Livingstone.

Cerling, T. E., Mbua, E., Kirera, F. M., Manthi, F. K., Grine, F. E., Leakey, M. G., ... & Uno, K. T. (2011). Diet of *Paranthropus boisei* in the early Pleistocene of East Africa. *Proceedings of the National Academy of Sciences*, *108*(23), 9337-9341.

Chai, H., & Lawn, B. R. (2007). A universal relation for edge chipping from sharp contacts in brittle materials: a simple means of toughness evaluation. *Acta Materialia*, *55*(7), 2555-2561.

Chai, H., Lee, J. J. W., & Lawn, B. R. (2011). On the chipping and splitting of teeth. *Journal of the Mechanical Behavior of Biomedical Materials*, *4*(3), 315-321.

Chai, H., Lee, J. J. W., Kwon, J. Y., Lucas, P. W., & Lawn, B. R. (2009). A simple model for enamel fracture from margin cracks. *Acta Biomaterialia*, *5*(5), 1663-1667.

Chamarthi, V., Varma, B. R., & Jayanthi, M. (2012). Amelogenesis imperfecta: A clinician's challenge. *Journal of Indian Society of Pedodontics and Preventive Dentistry*, *30*(1), 70.

Churchill, S. E., Holliday, T. W., Carlson, K. J., Jashashvili, T., Macias, M. E., Mathews, S., ... & Berger, L. R. (2013). The upper limb of *Australopithecus sediba*. *Science*, *340*(6129), 1233477.

Clark, J. D. (1993). Stone artefact assemblages from members 1–3, Swartkrans cave. In: Brain, C. K. (Ed.), *Swartkrans: a cave's chronicle of early man*, 167-194. Pretoria: Transvaal Museum.

Clarke, G. M. (1998). The genetic basis of developmental stability. V. Inter-and intra-individual character variation. *Heredity*, *80*(5), 562-567.

Clarke, N. G., & Hirsch, R. S. (1991). Physiological, pulpal, and periodontal factors influencing alveolar bone. In: Kelley, M. A., & Larsen, C. S. (Eds.), *Advances in dental anthropology*, 241-266. New York: Wiley-Liss.

Clarke, R. J. (1985). Early Acheulean with *Homo habilis* at Sterkfontein. In: Tobias, P. V., Strong, V., & White, H., (Eds.), *Hominid evolution: Past, present and future*, 287-298. Hoboken: Wiley-Blackwell.

Clarke, R. J. (1988). A new Australopithecus cranium from Sterkfontein and its bearing on the ancestry of *Paranthropus*. In: F.E. Grine (Ed.), *Evolutionary History of the "Robust" Australopithecines*, 285-292. New York: Aldine de Gruyter.

Clarke, R. J., & Howell, F. C. (1972). Affinities of the Swartkrans 847 hominid cranium. *American Journal of Physical Anthropology*, *37*(3), 319-335.

Clarke, R. J., Partridge, T. C., Granger, D. E., & Caffe, M. W. (2003). Dating the Sterkfontein fossils. *Science*, 301(5633), 596-597.

Clarkson, B. H., Krell, D., Wefel, J. S., Crall, J., & Feagin, F. F. (1987). In vitro caries-like lesion production by *Streptococcus mutans* and *Actinomyces viscosus* using sucrose and starch. *Journal of Dental Research*, *66*(3), 795-798.

Clement, A., & Hillson, S. (2013). Do larger molars and robust jaws in early hominins represent dietary adaptation? A New Study in Tooth Wear. *Archaeology International*, *16*, 59-71.

Clement, A. J. (1956). Caries in the South African apeman: some examples of undoubted pathological authenticity believed to be 800,000 years old. *British Dental Journal*, *101*, 4-7.

Cochran, D. L. (2008). Inflammation and bone loss in periodontal disease. *Journal of Periodontology*, *79*(8), 1569-1576.

Codron, D., Lee-Thorp, J. A., Sponheimer, M., De Ruiter, D., & Codron, J. (2008). What insights can baboon feeding ecology provide for early hominin niche differentiation? *International Journal of Primatology*, *29*(3), 757-772.

Codron, D., Luyt, J., Lee-Thorp, J. A., Sponheimer, M., De Ruiter, D., & Codron, J. (2005). Utilization of savanna-based resources by Plio-Pleistocene baboons: research letter. *South African Journal of Science*, *101*(5-6), 245-248.

Cofran, Z., Skinner, M. M., & Walker, C. S. (2016). Dental development and life history in *Homo naledi*. *American Journal of Physical Anthropology* 159, 118-118.

Collins Cook, D. (1980). Hereditary enamel hypoplasia in a prehistoric Indian child. *Journal of Dental Research*, *59*(9), 1522-1522.

Conklin-Brittain, N. L., Knott, C. D., & Wrangham, R. W. (2001). The feeding ecology of apes. In: *Conference Proceedings. The Apes: Challenges for the 21st Century*, 167-174. Brookfield: Chicago Zoological Society.

Connell, B., Jones, A. G., Redfern, R., & Walker, D. (2012). *A Bioarchaeological Study of Medieval Burials on the Site of St Mary Spital: Excavations at Spitalfields Market, London E1, 1991-2007*. London: Museum of London Archaeology.

Connell, B., & Rauxloh, P. (2003). A rapid method for recording human skeletal data. *MoL unpub rep*. Available at: http://www.museumoflondon.org.uk/English/Collections/OnlineResources/CHB/AboutUs/WORDdtb.htm.

Conroy, G. C., & Vannier, M. W. (1991). Dental development in South African *australopithecines*. Part II: Dental stage assessment. *American Journal of Physical Anthropology*, *86*(2), 137-156.

Constantino, P. J., Lee, J. J. W., Chai, H., Zipfel, B., Ziscovici, C., Lawn, B. R., & Lucas, P. W. (2010). Tooth chipping can reveal the diet and bite forces of fossil hominins. *Biology Letters*, *6*(6), 826-829.

Constantino, P. J., Markham, K., & Lucas, P. W. (2012). Tooth chipping as a tool to reconstruct diets of great apes (*Pongo, Gorilla, Pan*). *International Journal of Primatology*, *33*(3), 661-672.

Constantino, P. J., & Wright, B. W. (2009). The importance of fallback foods in primate ecology and evolution. *American Journal of Physical Anthropology*, *140*(4), 599-602.

Copeland, S. R., Sponheimer, M., de Ruiter, D. J., Lee-Thorp, J. A., Codron, D., le Roux, P. J., ... & Richards, M. P. (2011). Strontium isotope evidence for landscape use by early hominins. *Nature*, 474(7349), 76-78.

Cornejo, O. E., Lefébure, T., Pavinski Bitar, P. D., Lang, P., Richards, V. P., Eilertson, K., ... & Burne, R. A. (2013). Evolutionary and population genomics of the cavity causing bacteria *Streptococcus mutans*. *Molecular Biology and Evolution*, 30(4), 881-893.

Costa, R. L. (1980). Incidence of caries and abscesses in archeological Eskimo skeletal samples from Point Hope and Kodiak Island, Alaska. *American Journal of Physical Anthropology*, *52*(4), 501-514.

Coyler, J. F. (1936). *Coyler's variations and diseases of the teeth of animals*. John Bale, Sons, and Danielsson, London, UK.

Crawford, P. J., Aldred, M., & Bloch-Zupan, A. (2007). Amelogenesis imperfecta. *Orphanet Journal of Rare Diseases*, 2(1), 17-29.

Crittenden, A. N., Sorrentino, J., Moonie, S. A., Peterson, M., Mabulla, A., & Ungar, P. S. (2017). Oral health in transition: The Hadza foragers of Tanzania. *PLoS One*, *12*(3), e0172197.

Croft, L. K., Witkop, C. J., & Glas, J. E. (1965). Pseudohypoparathyroidism. *Oral Surgery, Oral Medicine, Oral Pathology*, *20*(6), 758-770.

Cunha, E., Rozzi, F. R., Bermúdez de Castro, J. M., Martinón-Torres, M., Wasterlain, S. N., & Sarmiento, S. (2004). Enamel hypoplasias and physiological stress in the Sima de los Huesos Middle Pleistocene hominins. *American Journal of Physical Anthropology*, *125*(3), 220-231.

Cuozzo, F. P., & Sauther, M. L. (2006). Severe wear and tooth loss in wild ring-tailed lemurs (*Lemur catta*): a function of feeding ecology, dental structure, and individual life history. *Journal of Human Evolution*, *51*(5), 490-505.

Cuy, J. L., Mann, A. B., Livi, K. J., Teaford, M. F., & Weihs, T. P. (2002). Nanoindentation mapping of the mechanical properties of human molar tooth enamel. *Archives of Oral Biology*, 47(4), 281-291.

Daegling, D. J., Carlson, K. J., Tafforeau, P., de Ruiter, D. J., & Berger, L. R. (2016). Comparative biomechanics of *Australopithecus sediba* mandibles. *Journal of Human Evolution*, *100*, 73-86.

Daegling, D. J., & Grine, F. E. (1991). Compact bone distribution and biomechanics of early hominid mandibles. *American Journal of Physical Anthropology*, 86(3), 321-339.

Daegling, D. J., & Grine, F. E. (1994). Bamboo feeding, dental microwear, and diet of the Pleistocene ape *Gigantopithecus blacki*. *South African Journal of Science*, *90*(10), 527-532.

Daegling, D. J., Judex, S., Ozcivici, E., Ravosa, M. J., Taylor, A. B., Grine, F. E., ... & Ungar, P. S. (2013). Viewpoints: feeding mechanics, diet, and dietary adaptations in early hominins. *American Journal of Physical Anthropology*, *151*(3), 356-371.

Daegling, D. J., & McGraw, W. S. (2007). Functional morphology of the mangabey mandibular corpus: relationship to dental specializations and feeding behavior. *American Journal of Physical Anthropology*, *134*(1), 50-62.

Daegling, D. J., McGraw, W. S., Ungar, P. S., Pampush, J. D., Vick, A. E., & Bitty, E. A. (2011). Hard-object feeding in sooty mangabeys (*Cercocebus atys*) and interpretation of early hominin feeding ecology. *PLoS One*, *6*(8), e23095.

Dart, R.A., 1925. *Australopithecus africanus*: the man-ape of South Africa. *Nature*, 115, 195-199.

Dart, R. A. (1948). The adolescent mandible of *Australopithecus prometheus*. *American Journal of Physical Anthropology*, *6*(4), 391-412.

Dart, R. A. (1960). The bone tool-manufacturing ability of *Australopithecus prometheus*. *American Anthropologist*, *62*(1), 134-138.

Davideau, J. L., Lezot, F., Kato, S., Bailleul-Forestier, I., & Berdal, A. (2004). Dental alveolar bone defects related to Vitamin D and calcium status. *The Journal of Steroid Biochemistry and Molecular Biology*, *89*, 615-618.

De Groote, I., & Humphrey, L. T. (2016). Characterizing evulsion in the Later Stone Age Maghreb: Age, sex and effects on mastication. *Quaternary International*, *413*, 50-61.

de Ruiter, D. J., DeWitt, T. J., Carlson, K. B., Brophy, J. K., Schroeder, L., Ackermann, R. R., ... & Berger, L. R. (2013). Mandibular remains support taxonomic validity of *Australopithecus sediba*. *Science*, *340*(6129).

de Ruiter, D. J., Pickering, R., Steininger, C. M., Kramers, J. D., Hancox, P. J., Churchill, S. E., ... & Backwell, L. (2009). New *Australopithecus robustus* fossils and associated U-Pb dates from Cooper's cave (Gauteng, South Africa). *Journal of Human Evolution*, *56*(5), 497-513.

Dean, M. C., Beynon, A. D., Thackeray, J. F., & Macho, G. A. (1993). Histological reconstruction of dental development and age at death of a juvenile *Paranthropus robustus* specimen, SK 63, from Swartkrans, South Africa. *American Journal of Physical Anthropology*, *91*(4), 401-419.

Dean, M. C., Jones, M. E., & Pilley, J. R. (1992). The natural history of tooth wear, continuous eruption and periodontal disease in wild shot great apes. *Journal of Human Evolution*, *22*(1), 23-39.

Dean, M. C., & Wood, B. A. (1981). Developing *pongid* dentition and its use for ageing individual crania in comparative cross-sectional growth studies. *Folia Primatologica*, *36*(1-2), 111-127.

DeCasien, A. R., Williams, S. A., & Higham, J. P. (2017). Primate brain size is predicted by diet but not sociality. *Nature Ecology & Evolution*, 1, 0112.

Delgado-Darias, T., Velasco-Vázquez, J., Arnay-de-la-Rosa, M., Martin-Rodriguez, E., & González-Reimers, E. (2006). Calculus, periodontal disease and tooth decay among the prehispanic population from Gran Canaria. *Journal of Archaeological Science*, *33*(5), 663-670.

d'Errico, F., & Backwell, L. R. (2003). Possible evidence of bone tool shaping by Swartkrans early hominids. *Journal of Archaeological Science*, 30(12), 1559-1576.

DeSilva, J. M., Holt, K. G., Churchill, S. E., Carlson, K. J., Walker, C. S., Zipfel, B., & Berger, L. R. (2013). The lower limb and mechanics of walking in *Australopithecus sediba*. *Science*, *340*(6129).

Deter, C. A. (2009). Gradients of occlusal wear in hunter-gatherers and agriculturalists. *American Journal of Physical Anthropology*, *138*(3), 247-254.

Dias, G., & Tayles, N. (1997). 'Abscess cavity'—a misnomer. *International Journal of Osteoarchaeology*, *7*(5), 548-554.

Dirks, P. H., & Berger, L. R. (2013). Hominin-bearing caves and landscape dynamics in the Cradle of Humankind, South Africa. *Journal of African Earth Sciences*, *78*, 109-131.

Dirks, P. H., Berger, L. R., Roberts, E. M., Kramers, J. D., Hawks, J., Randolph-Quinney, P. S., ... & Schmid, P. (2015). Geological and taphonomic context for the new hominin species *Homo naledi* from the Dinaledi Chamber, South Africa. *Elife*, *4*, e09561.

Dirks, P. H., Roberts, E. M., Hilbert-Wolf, H., Kramers, J. D., Hawks, J., Dosseto, A., ... & Hellstrom, J. (2017). The age of *Homo naledi* and associated sediments in the Rising Star Cave, South Africa. *Elife*, *6*, e24231.

Dirks, W., & Bowman, J. E. (2007). Life history theory and dental development in four species of catarrhine primates. *Journal of Human Evolution*, *53*(3), 309-320.

Dirks, W., Reid, D. J., Jolly, C. J., Phillips-Conroy, J. E., & Brett, F. L. (2002). Out of the mouths of baboons: stress, life history, and dental development in the Awash National Park hybrid zone, Ethiopia. *American Journal of Physical Anthropology*, *118*(3), 239-252.

Dobney, K., & Ervynck, A. (2000). Interpreting developmental stress in archaeological pigs: the chronology of linear enamel hypoplasia. *Journal of Archaeological Science*, *27*(7), 597-607.

Dominy, N. J., Vogel, E. R., Yeakel, J. D., Constantino, P., & Lucas, P. W. (2008). Mechanical properties of plant underground storage organs and implications for dietary models of early hominins. *Evolutionary Biology*, *35*(3), 159-175.

Doran, D. M., McNeilage, A., Greer, D., Bocian, C., Mehlman, P., & Shah, N. (2002). Western lowland gorilla diet and resource availability: New evidence, cross-site comparisons, and reflections on indirect sampling methods. *American Journal of Primatology*, *58*(3), 91-116.

Dressino, V., & Pucciarelli, H. M. (1997). Cranial growth in *Saimiri sciureus* (*Cebidae*) and its alteration by nutritional factors: a longitudinal study. *American Journal of Physical Anthropology*, *102*(4), 545-554.

Du Brul, E. L. (1977). Early hominid feeding mechanisms. *American Journal of Physical Anthropology*, *47*(2), 305.

Duray, S. M. (1992). Enamel defects and caries etiology: an historical perspective. Recent contributions to the Study of Enamel Developmental Defects. *Journal of Paleopathology,* (2), 307-319.

Duverger, O., Beniash, E., & Morasso, M. I. (2016). Keratins as components of the enamel organic matrix. *Matrix Biology*, *52*, 260-265.

Duyar, I., & Erdal, Y. S. (2003). A new approach for calibrating dental caries frequency of skeletal remains. *Journal of Comparative Human Biology*, *54*(1), 57-70.

Eke, P. I., Dye, B. A., Wei, L., Thornton-Evans, G. O., & Genco, R. J. (2012). Prevalence of periodontitis in adults in the United States: 2009 and 2010. *Journal of Dental Research*, *91*(10), 914-920.

Elgart, A. A. (2010). Dental wear, wear rate, and dental disease in the African apes. *American Journal of Primatology*, *72*(6), 481-491.

Eliot, M. M., Souther, S. P., Anderson, B. G., & Arnim, S. S. (1934). A study of the teeth of a group of school children previously examined for rickets. *American Journal of Diseases of Children*, *48*(4), 713-729.

Enwonwu, C. O. (1973). Influence of socio-economic conditions on dental development in Nigerian children. *Archives of Oral Biology*, *18*(1), 95-IN15.

Esclassan, R., Grimoud, A. M., Ruas, M. P., Donat, R., Sevin, A., Astie, F., ... & Crubezy, E. (2009). Dental caries, tooth wear and diet in an adult medieval (12th–14th century) population from Mediterranean France. *Archives of Oral Biology*, *54*(3), 287-297.

Eshed, V., Gopher, A., & Hershkovitz, I. (2006). Tooth wear and dental pathology at the advent of agriculture: new evidence from the Levant. *American Journal of Physical Anthropology*, *130*(2), 145-159.

Estalrrich, A., Alarcón, J. A., & Rosas, A. (2016). Evidence of toothpick groove formation in Neandertal anterior and posterior teeth. *American Journal of Physical Anthropology*, 162(4), 747–756.

Estalrrich, A., & Rosas, A. (2013). Handedness in Neandertals from the El Sidrón (Asturias, Spain): evidence from instrumental striations with ontogenetic inferences. *PloS one*, *8*(5), e62797.

Estebaranz, F., Martínez, L. M., Galbany, J., Turbón, D., & Pérez-Pérez, A. (2009). Testing hypotheses of dietary reconstruction from buccal dental microwear in *Australopithecus afarensis*. *Journal of Human Evolution*, *57*(6), 739-750.

Etkin, W. (1954). Social behavior and the evolution of man's mental faculties. *The American Naturalist*, *88*(840), 129-142.

Eversole, L. R. (1984). *Clinical outline of oral pathology: diagnosis and treatment*. Philadelphia: Lea & Febiger publishers.

Fejerskov, O., Manji, F., & Baelum, V. (1990). The nature and mechanisms of dental fluorosis in man. *Journal of Dental Research*, *69*(2), 692-700.

Feuerriegel, E. M., Green, D. J., Walker, C. S., Schmid, P., Hawks, J., Berger, L. R., & Churchill, S. E. (2017). The upper limb of *Homo naledi*. *Journal of Human Evolution*, *104*, 155-173.

Fiore, I., Bondioli, L., Radovčić, J., & Frayer, D. W. (2015). Handedness in the Krapina Neandertals: A re-evaluation. *PaleoAnthropology*, *2015*, 19-36.

Fiorenza, L., Benazzi, S., & Kullmer, O. (2011). Para-masticatory wear facets and their functional significance in hunter–gatherer maxillary molars. *Journal of Archaeological Science*, *38*(9), 2182-2189.

Fiorenza, L., & Kullmer, O. (2013). Dental wear and cultural behavior in Middle Paleolithic humans from the Near East. *American Journal of Physical Anthropology*, *152*(1), 107-117.

Fiorenza, L., & Kullmer, O. (2015). Dental wear patterns in early modern humans from Skhul and Qafzeh: A response to Sarig and Tillier. *Journal of Comparative Human Biology*, *66*(5), 414-419.

Fischer, F. M., El-Kafrawy, A., & Mitchell, D. F. (1970). Studies of tertiary dentin in monkey teeth using vital dyes. *Journal of Dental Research*, *49*(6), 1537-1540.

Formicola, V. (1988). Interproximal grooving of teeth: additional evidence and interpretation. *Current Anthropology*, *29*(4), 663-671.

Fornai, C., Bookstein, F. L., & Weber, G. W. (2015). Variability of *Australopithecus* second maxillary molars from Sterkfontein Member 4. *Journal of Human Evolution*, *85*, 181-192.

Foster, B. L., Nociti, F. H., & Somerman, M. J. (2013). Tooth root development. *Stem Cells in Craniofacial Development and Regeneration*, 153-177.

Fostowicz-Frelik, Ł., & Frelik, G. J. (2010). Earliest record of dental pathogen discovered in a North American Eocene rabbit. *Palaios*, *25*(12), 818-822.

Fox, C. L., & Frayer, D. W. (1997). Non-dietary Marks in the Anterior Dentition of the Krapina Neanderthals. *International Journal of Osteoarchaeology*, *7*(2), 133-149.

Frayer, D. W. (1991). On the etiology of interproximal grooves. *American Journal of Physical Anthropology*, *85*(3), 299-304.

Frayer, D. W., Clarke, R. J., Fiore, I., Blumenschine, R. J., Pérez-Pérez, A., Martinez, L. M., ... & Bondioli, L. (2016). OH-65: The earliest evidence for right-handedness in the fossil record. *Journal of Human Evolution*, *100*, 65-72.

Frayer, D. W., & Russell, M. D. (1987). Artificial grooves on the Krapina Neanderthal teeth. *American Journal of Physical Anthropology*, *74*(3), 393-405.

Galdikas, B. M. (1982). Orangutans as seed dispersers at Tanjung Puling, Central Kalimantan: implications for conservation. In: de Boer, L. E. (Ed.), *The orang Utan: Its biology and conservation*, 285. Berlin: Springer Science & Business Media.

Garat, J. A., Martín, A. E., Pani, M., Holgado, N. R., Meheris, H. E., & González, S. (2006). Orthodontic implications of protein undernutrition in mandibular growth. A cephalometric study in growing rats. *Acta Odontologica Latinoamericana*, *20*(2), 73-78.

Gaul, J. S., Grossschmidt, K., Gusenbauer, C., & Kanz, F. (2015). A probable case of congenital syphilis from pre-Columbian Austria. *Anthropologischer Anzeiger*, *72*(4), 451-472.

Geismar, K., Stoltze, K., Sigurd, B., Gyntelberg, F., & Holmstrup, P. (2006). Periodontal disease and coronary heart disease. *Journal of Periodontology*, *77*(9), 1547-1554.

Geissler, E., Duque, A. C., Pampush, J. D., Daegling, D. J., & Mcgraw, W. S. (2015). Spatial variation of dentine hardness in the molars of three primate taxa. *American Journal of Physical Anthropology*, *156*, 142-142.

Gittleman, J. L. (1994). Are the pandas successful specialists or evolutionary failures? *BioScience*, *44*(7), 456-464.

Gommery, D., Sénégas, F., Thackeray, J. F., Potze, S., Kgasi, L., Claude, J., & Lacruz, R. (2008). Plio-Pleistocene fossils from Femur Dump, Bolt's Farm, Cradle of Humankind World Heritage Site. *Annals of the Transvaal Museum*, *45*(1), 67-76.

Goodman, A. H., Allen, L. H., Hernandez, G. P., Amador, A., Arriola, L. V., Chavez, A., & Pelto, G. H. (1987). Prevalence and age at development of enamel hypoplasias in Mexican children. *American Journal of Physical Anthropology*, *72*(1), 7-19.

Goodman, A. H., & Armelagos, G. J. (1985). The chronological distribution of enamel hypoplasia in human permanent incisor and canine teeth. *Archives of Oral Biology*, *30*(6), 503-507.

Goodman, A. H., Armelagos, G. J., & Rose, J. C. (1980). Enamel hypoplasias as indicators of stress in three prehistoric populations from Illinois. *Human Biology*, 515-528.

Goodman, A. H., Armelagos, G. J., & Rose, J. C. (1984). The chronological distribution of enamel hypoplasias from prehistoric Dickson Mounds populations. *American Journal of Physical Anthropology*, *65*(3), 259-266.

Goodman, A. H., & Rose, J. C. (1990). Assessment of systemic physiological perturbations from dental enamel hypoplasias and associated histological structures. *American Journal of Physical Anthropology*, *33*(S11), 59-110.

Goodman, A. H., & Rose, J. C. (1991). Dental enamel hypoplasias as indicators of nutritional status. In: Kelley, M. A., & Larsen, C. S. (Eds.), *Advances in dental anthropology*. New York: Wiley-Liss, 279-293.

Gottlieb, B., & Orban, B. (1933). Active and passive continuous eruptions of teeth. *Journal of Dental Research*, *13*, 214.

Gould, R. A. (1968). Chipping stones in the outback. *Natural History*, *77*(2), 42-49.

Gracia-Téllez, A., Arsuaga, J. L., Martínez, I., Martín-Francés, L., Martinón-Torres, M., de Castro, J. M. B., ... & Lira, J. (2013). Orofacial pathology in *Homo heidelbergensis*: the case of Skull 5 from the Sima de los Huesos site (Atapuerca, Spain). *Quaternary International*, *295*, 83-93.

Grahnen, H., & Selander, P. (1954). The effect of rickets and spasmophilia on the permanent dentition. I. The effect on the teeth. *Odontologisk Revy*, *5*(1), 7.

Greene, T. R., Kuba, C. L., & Irish, J. D. (2005). Quantifying calculus: a suggested new approach for recording an important indicator of diet and dental health. *Journal of Comparative Human Biology*, 56(2), 119-132.

Grimes, S. T., Brock, F., Rickard, D., Davies, K. L., Edwards, D., Briggs, D. E., & Parkes, R. J. (2001). Understanding fossilization: experimental pyritization of plants. *Geology*, *29*(2), 123-126.

Grimoud, A. M., Gibbon, V. E., & Ribot, I. (2017). Predictive factors for alveolar fenestration and dehiscence. *Journal of Comparative Human Biology*, 68(3), 167–175.

Grimoud, A. M., Roberts, C. A., Boimond, L., Sevin, A., Lucas, S., & Passarrius, O. (2012). Topographical presentation of dental wear as arches in a French mediaeval population. *Archives of Oral Biology*, *57*(6), 841-852.

Grine, F. E. (1981). Trophic differences between 'gracile' and 'robust' australopithecines: a scanning electron microscope analysis of occlusal events. *South African Journal of Science*, *77*(5), 203-230.

Grine, F. E. (1986). Dental evidence for dietary differences in *Australopithecus* and *Paranthropus*: a quantitative analysis of permanent molar microwear. *Journal of Human Evolution*, *15*(8), 783-822.

Grine, F. E. (2005). Early *Homo* at Swartkrans, South Africa: a review of the evidence and an evaluation of recently proposed morphs. *South African Journal of Science*, *101*(1-2), 43-52.

Grine, F. E. (Ed.). (2007). *Evolutionary History of the" robust" Australopithecines*. Piscataway: Transaction Publishers.

Grine, F. E. (2013). The alpha taxonomy of *Australopithecus africanus*. In: Kaye. R. E., John, G. F., Leakey, R. E., (Eds.), *The paleobiology of Australopithecus*, 73-104. Springer-Netherlands.

Grine, F. E., Bromage, T. G., Daegling, D. J., Burr, D. B., & Brain, C. K. (2015). Microbial osteolysis in an Early Pleistocene hominin (*Paranthropus robustus*) from Swartkrans, South Africa. *Journal of Human Evolution*, *85*, 126-135.

Grine, F.E., Delanty, M.M., Wood, B.A. (2013). Variation in mandibular postcanine dental morphology and hominin species representation in Member 4, Sterkfontein, South Africa. In: Reed, K., Fleagle, J., Leakey, R.E.F. (Eds.), *Paleobiology of Australopithecus*, 125-146. New York: Springer.

Grine, F. E., Demes, B., Jungers, W. L., & Cole, T. M. (1993). Taxonomic affinity of the early *Homo* cranium from Swartkrans, South Africa. *American Journal of Physical Anthropology*, *92*(4), 411-426.

Grine, F. E., Gwinnett, A. J., & Oaks, J. H. (1990). Early hominid dental pathology: interproximal caries in 1.5 million-year-old *Paranthropus robustus* from Swartkrans. *Archives of Oral Biology*, *35*(5), 381-386.

Grine, F. E., Henshilwood, C. S., & Sealy, J. C. (2000). Human remains from Blombos Cave, South Africa:(1997–1998 excavations). *Journal of Human Evolution*, *38*(6), 755-765.

Grine, F. E., Judex, S., Daegling, D. J., Ozcivici, E., Ungar, P. S., Teaford, M. F., ... & Walker, A. (2010). Craniofacial biomechanics and functional and dietary inferences in hominin paleontology. *Journal of Human Evolution*, *58*(4), 293-308.

Grine, F. E., Sponheimer, M., Ungar, P. S., Lee-Thorp, J., & Teaford, M. F. (2012). Dental microwear and stable isotopes inform the paleoecology of extinct hominins. *American Journal of Physical Anthropology*, *148*(2), 285-317.

Grippo, J. O., Simring, M., & Coleman, T. A. (2012). Abfraction, Abrasion, Biocorrosion, and the Enigma of Noncarious Cervical Lesions: A 20-Year Perspective. *Journal of Esthetic and Restorative Dentistry*, *24*(1), 10-23.

Grippo, J. O., Simring, M., & Schreiner, S. (2004). Attrition, abrasion, corrosion and abfraction revisited: a new perspective on tooth surface lesions. *The Journal of the American Dental Association*, *135*(8), 1109-1118.

Guatelli-Steinberg, D. (2001). What can developmental defects of enamel reveal about physiological stress in nonhuman primates? *Evolutionary Anthropology: Issues, News, and Reviews*, *10*(4), 138-151.

Guatelli-Steinberg, D. (2003). Macroscopic and microscopic analyses of linear enamel hypoplasia in Plio-Pleistocene South African hominins with respect to aspects of enamel development and morphology. *American Journal of Physical Anthropology*, *120*(4), 309-322.

Guatelli-Steinberg, D. (2015). Micro-to Macroscopic. In: Irish, J. D., & Scott, G. R. (Eds.), *A companion to dental anthropology*, 450. Hoboken: Wiley-Blackwell.

Guatelli-Steinberg, D. (2016). *What Teeth Reveal about Human Evolution*. Cambridge: Cambridge University Press.

Guatelli-Steinberg, D., Larsen, C. S., & Hutchinson, D. L. (2004). Prevalence and the duration of linear enamel hypoplasia: a comparative study of Neandertals and Inuit foragers. *Journal of Human Evolution*, *47*(1), 65-84.

Guatelli-Steinberg, D., & Lukacs, J. R. (1999). Interpreting sex differences in enamel hypoplasia in human and non-human primates: Developmental, environmental, and cultural considerations. *American Journal of Physical Anthropology*, *110*(S29), 73-126.

Guatelli-Steinberg, D., & Skinner, M. (2000). Prevalence and etiology of linear enamel hypoplasia in monkeys and apes from Asia and Africa. *Folia Primatologica*, *71*(3), 115-132.

Guatelli-Steinberg, D., Stinespring-Harris, A., Reid, D. J., Larsen, C. S., Hutchinson, D. L., & Smith, T. M. (2014). Chronology of linear enamel hypoplasia formation in the Krapina Neanderthals. *PaleoAnthropology*, 431, 431-445.

Gussy, M. G., Waters, E. G., Walsh, O., & Kilpatrick, N. M. (2006). Early childhood caries: current evidence for aetiology and prevention. *Journal of Paediatrics and Child Health*, *42*(1-2), 37-43.

Hajishengallis, G. (2015). Periodontitis: from microbial immune subversion to systemic inflammation. *Nature Reviews Immunology*, *15*(1), 30-44.

Hannibal, D. L. (2016). Prevalence of an unusual hypoplastic defect of the permanent maxillary lateral incisor in great apes. *American Journal of Primatology*, 79(2), 1-9.

Harcourt-Smith, W. E. H., Throckmorton, Z., Congdon, K. A., Zipfel, B., Deane, A. S., Drapeau, M. S. M., ... & DeSilva, J. M. (2015). The foot of *Homo naledi*. *Nature Communications*, *6*, 8432.

Harrison, M. E., & Marshall, A. J. (2011). Strategies for the use of fallback foods in apes. *International Journal of Primatology*, *32*(3), 531-565.

Hartnady, P., & Rose, J. C. (1991). Abnormal tooth-loss patterns among Archaic-period inhabitants of the lower Pecos region, Texas. In: Kelley, M. A., & Larsen, C. S. (Eds.), *Advances in dental anthropology*, 267-278. New York: Wiley-Liss.

Hassett, B. R. (2012). Evaluating sources of variation in the identification of linear hypoplastic defects of enamel: a new quantified method. *Journal of Archaeological Science*, *39*(2), 560-565.

Hawks, J., Elliott, M., Schmid, P., Churchill, S. E., de Ruiter, D. J., Roberts, E. M., ... & Feuerriegel, E. M. (2017). New fossil remains of *Homo naledi* from the Lesedi Chamber, South Africa. *Elife*, *6*, e24232.

He, L. H., Fujisawa, N., & Swain, M. V. (2006). Elastic modulus and stress–strain response of human enamel by nano-indentation. *Biomaterials*, 27(24), 4388-4398.

He, L. H., & Swain, M. V. (2008). Understanding the mechanical behaviour of human enamel from its structural and compositional characteristics. *Journal of the Mechanical Behavior of Biomedical Materials*, *1*(1), 18-29.

Henry, A. G., Ungar, P. S., Passey, B. H., Sponheimer, M., Rossouw, L., Bamford, M., ... & Berger, L. (2012). The diet of *Australopithecus sediba*. *Nature*, *487*(7405), 90-93.

Hildebolt, C. F., & Molnar, S. (1991). Measurement and description of periodontal disease in anthropological studies. In: Kelley, M. A., & Larsen, C. S. (Eds.), *Advances in dental anthropology*, 225-240. New York: Wiley-Liss.

Hillson, S. (1996). *Dental anthropology*. Cambridge: Cambridge University Press.

Hillson, S. (2001). Recording dental caries in archaeological human remains. *International Journal of Osteoarchaeology*, *11*(4), 249-289.

Hillson, S. (2005). *Teeth*. Cambridge: Cambridge University Press.

Hillson, S. (2014). *Tooth development in human evolution and bioarchaeology*. Cambridge: Cambridge University Press.

Hillson, S. W. (1992). Dental enamel growth, perikymata and hypoplasia in ancient tooth crowns. *Journal of the Royal Society of Medicine*, *85*(8), 460-466.

Hillson, S., & Bond, S. (1997). Relationship of enamel hypoplasia to the pattern of tooth crown growth: a discussion. *American Journal of Physical Anthropology*, *104*(1), 89-103.

Hillson, S., Grigson, C., & Bond, S. (1998). Dental defects of congenital syphilis. *American Journal of Physical Anthropology*, *107*(1), 25-40.

Hilton-Barber, B., & Berger, L. R. (2004). *Field Guide to the Cradle of Humankind: Sterkfontein, Swartkrans, Kromdraai & Environs World Heritage Site*. South Africa: Struik Publishers.

Hinton, R. J. (1981). Form and patterning of anterior tooth wear among aboriginal human groups. *American Journal of Physical Anthropology*, *54*(4), 555-564.

Hlusko, L. J. (2003). The Oldest Hominid Habit? Experimental Evidence for Toothpicking with Grass Stalks. *Current Anthropology*, *44*(5), 738-741.

Hong, L., Levy, S. M., Warren, J. J., & Broffitt, B. (2009). Association between enamel hypoplasia and dental caries in primary second molars: a cohort study. *Caries Research*, *43*(5), 345-353.

Horvath, J. E., Ramachandran, G. L., Fedrigo, O., Nielsen, W. J., Babbitt, C. C., Clair, E. M. S., ... & Wall, C. E. (2014). Genetic comparisons yield insight into the evolution of enamel thickness during human evolution. *Journal of Human Evolution*, *73*, 75-87.

Howell, F. C. (1963). *African ecology and human evolution*. Piscataway: Transaction Publishers.

Hu, J. C. C., Yamakoshi, Y., Yamakoshi, F., Krebsbach, P. H., & Simmer, J. P. (2005). Proteomics and genetics of dental enamel. *Cells Tissues Organs*, *181*(3-4), 219-231.

Hu, J. C., & Yamakoshi, Y. (2003). Enamelin and autosomal-dominant amelogenesis imperfecta. *Critical Reviews in Oral Biology & Medicine*, *14*(6), 387-398.

Huckert, M., Stoetzel, C., Morkmued, S., Laugel-Haushalter, V., Geoffroy, V., Muller, J., ... & Switala, M. (2015). Mutations in the latent TGF-beta binding protein 3 (LTBP3) gene cause brachyolmia with amelogenesis imperfecta. *Human Molecular Genetics*, 24(11), 3038-3049.

Hughes, A. R., & Tobias, P. V. (1977). A fossil skull probably of the genus *Homo* from Sterkfontein, Transvaal. *Nature*, 265(5592), 310-312.

Humphrey, L. T., De Groote, I., Morales, J., Barton, N., Collcutt, S., Ramsey, C. B., & Bouzouggar, A. (2014). Earliest evidence for caries and exploitation of starchy plant foods in Pleistocene hunter-gatherers from Morocco. *Proceedings of the National Academy of Sciences*, *111*(3), 954-959.

Hylander, W. L. (1988). Implications of in vivo experiments for interpreting the functional significance of "robust" australopithecine jaws. In: Grine, F. E. (Ed.), *Evolutionary history of the "robust" australopithecines*, 55-83. Piscataway: Transaction Publishers.

Indriati, E., & Buikstra, J. E. (2001). Coca chewing in prehistoric coastal Peru: dental evidence. *American Journal of Physical Anthropology*, *114*(3), 242-257.

Infante, P. F., & Gillespie, G. M. (1974). An epidemiologic study of linear enamel hypoplasia of deciduous anterior teeth in Guatemalan children. *Archives of Oral Biology*, *19*(11), 1055-1061.

Ioannou, S., Henneberg, M., Henneberg, R. J., & Anson, T. (2015). Diagnosis of mercurial teeth in a possible case of congenital syphilis and tuberculosis in a 19th century child skeleton. *Journal of Anthropology*, 2015, 1–11.

Irish, J. D., Guatelli-Steinberg, D., Legge, S. S., de Ruiter, D. J., & Berger, L. R. (2013). Dental morphology and the phylogenetic "place" of *Australopithecus sediba*. *Science*, *340*(6129), 1233062.

Isabirye-Basuta, G. (1989). Feeding ecology of chimpanzees in the Kibale Forest, Uganda. In: Heltne, P. G., Marquardt, L. A., (Eds.), *Understanding chimpanzees*. Cambridge: Harvard University Press, 116-127.

Janis, C. M. (1984). Prediction of primate diets from molar wear patterns. In: Chivers, J. Wood, B.A., Bilsborough, A. (Eds.), *Food acquisition and processing in primates*, 331-340. New York: Springer US.

Jeffcoat, M. K., Geurs, N. C., & Lewis, C. E. (2003). Osteoporosis and periodontal bone loss. *Clinical Calcium*, *13*(5), 577-581.

Jernvall, J., Gilbert, C. C., & Wright, P. C. (2008). Peculiar Tooth Homologies of the Greater Bamboo. In: Fleagle, J. G, Gilbert, C. C., (Eds.), *Elwyn Simons: A Search for Origins*. London: Springer.

Johanson, D. C., & Taieb, M. (1976). Plio-pleistocene hominid discoveries in Hadar, Ethiopia. *Nature*, *260*(5549), 293-297.

Jolly, C. J. (1970). The seed-eaters: a new model of hominid differentiation based on a baboon analogy. *Man*, *5*(1), 5-26.

Jones, T. S., & Cave, A. J. E. (1960). Diet, longevity and dental disease in the Sierra Leone chimpanzee. *Journal of Zoology*, 135(1), 147-155.

Jungers, W. L., & Grine, F. E. (1986). Dental trends in the australopithecines: the allometry of mandibular molar dimensions. In: Wood, B. A., Martin, L. B., & Andrews, P. (Eds.), *Major topics in primate and human evolution*, 203-219. New York: Cambridge University Press.

Jungers, W. L., & Kaifu, Y. (2011). On dental wear, dental work, and oral health in the type specimen (LB1) of *Homo floresiensis*. *American Journal of Physical Anthropology*, *145*(2), 282-289.

Kaidonis, J. A. (2008). Tooth wear: the view of the anthropologist. *Clinical Oral Investigations*, *12*(1), 21-26.

Kaidonis, J. A., Ranjitkar, S., Lekkas, D., & Townsend, G. C. (2012). An anthropological perspective: another dimension to modern dental wear concepts. *International Journal of Dentistry*, *2012*, 741405.

Kaifu, Y., Kasai, K., Townsend, G. C., & Richards, L. C. (2003). Tooth wear and the "design" of the human dentition: a perspective from evolutionary medicine. *American Journal of Physical Anthropology*, *122*(S37), 47-61.

Kalthoff, D. C., Rose, K. D., & Von Koenigswald, W. (2011). Dental microstructure in *Palaeanodon* and *Tubulodon* (Palaeanodonta) and bioerosional tunneling as a widespread phenomenon in fossil mammal teeth. *Journal of Vertebrate Paleontology*, *31*(6), 1303-1313.

Kato, A., Kouchi, M., Mochimaru, M., Isomura, A., & Ohno, N. (2011). A geometric morphometric analysis of the crown form of the maxillary central incisor in humans. *Dental Anthropology*, *24*, 1-10.

Katz, J., Chaushu, G., & Sgan-Cohen, H. D. (2000). Relationship of blood glucose level to community periodontal index of treatment needs and body mass index in a permanent Israeli military population. *Journal of Periodontology*, *71*(10), 1521-1527.

Kay, R. F. (1981). The nut-crackers–a new theory of the adaptations of the Ramapithecinae. *American Journal of Physical Anthropology*, *55*(2), 141-151.

Kay, R. F. (1985). Dental evidence for the diet of *Australopithecus*. *Annual Review of Anthropology*, 14(1), 315-341.

Kear, B. P. (2001). Dental caries in an Early Cretaceous ichthyosaur. *Alcheringa*, *25*(4), 387-390.

Keenleyside, A. (2008). Dental pathology and diet at Apollonia, a Greek colony on the Black Sea. *International Journal of Osteoarchaeology*, *18*(3), 262-279.

Kelley, J. L., & Swanson, W. J. (2008). Dietary change and adaptive evolution of enamelin in humans and among primates. *Genetics*, *178*(3), 1595-1603.

Kelley, J., & Bulicek, C. (2000). Identification of a birth cohort in the Miocene hominoid fossil record. *American Journal of Physical Anthropology*, 195-195.

Kelley, M. A., Levesque, D. R., & Weidl, E. (1991). Contrasting patterns of dental disease in five early northern Chilean groups. In: Kelley, M. A., & Larsen, C. S. (Eds.), *Advances in dental anthropology*, 203-213. New York: Wiley-Liss.

Kemp, A. (2003). Dental and skeletal pathology in lungfish jaws and tooth plates. *Alcheringa*, *27*(2), 155-170.

Kerr, N. W. (1998). The prevalence and natural history of periodontal disease in Britain from prehistoric to modern times. *British Dental Journal*, *185*(10), 527-535.

Keyser, A. W., Menter, C. G., Moggi-Cecchi, J., Pickering, T. R., & Berger, L. R. (2000). Drimolen: a new hominid-bearing site in Gauteng, South Africa. *South African Journal of Science*, *96*(4), 193-197.

Kibii, J. M., Churchill, S. E., Schmid, P., Carlson, K. J., Reed, N. D., De Ruiter, D. J., & Berger, L. R. (2011). A partial pelvis of *Australopithecus sediba*. *Science*, *333*(6048), 1407-1411.

Kilgore, L. (1989). Dental pathologies in ten free-ranging chimpanzees from Gombe National Park, Tanzania. *American Journal of Physical Anthropology*, *80*(2), 219-227.

Kim, Y. J., Shin, T. J., Hyun, H. K., Lee, S. H., Lee, Z. H., & Kim, J. W. (2016). A novel de novo mutation in LAMB3 causes localized hypoplastic enamel in the molar region. *European Journal of Oral Sciences*, *124*(4), 403-405.

Kivell, T. L., Deane, A. S., Tocheri, M. W., Orr, C. M., Schmid, P., Hawks, J., ... & Churchill, S. E. (2015). The hand of *Homo naledi*. *Nature Communications*, *6*, 8431.

Kivell, T. L., Kibii, J. M., Churchill, S. E., Schmid, P., & Berger, L. R. (2011). *Australopithecus sediba* hand demonstrates mosaic evolution of locomotor and manipulative abilities. *Science*, *333*(6048), 1411-1417.

Koch, M. J., Bührer, R., Pioch, T., & Schärer, K. (1999). Enamel hypoplasia of primary teeth in chronic renal failure. *Pediatric Nephrology*, *13*(1), 68-72.

Kono, R. T. (2004). Molar enamel thickness and distribution patterns in extant great apes and humans: new insights based on a 3-dimensional whole crown perspective. *Anthropological Science*, *112*(2), 121-146.

Koritzer, R. T. (1977). An anthropological approach to the study of dental pathology. In: Dahlberg, A. A., & Graber, T. M. (Eds.), *Orofacial Growth and Development*, 283-300. *The Hague: Mouton*.

Kotecha, P. V., Patel, S. V., Bhalani, K. D., Shah, D., Shah, V. S., & Mehta, K. G. (2012). Prevalence of dental fluorosis & dental caries in association with high levels of drinking water fluoride content in a district of Gujarat, India. *Indian Journal of Medical Research*, *135*(6), 873-877.

Krenz-Niedbała, M., & Kozłowski, T. (2013). Comparing the chronological distribution of enamel hypoplasia in Rogowo, Poland (2nd century AD) using two methods of defect timing estimation. *International Journal of Osteoarchaeology*, *23*(4), 410-420.

Kreshover, S. J. (1960). Metabolic disturbances in tooth formation. *Annals of the New York Academy of Sciences*, *85*(1), 161-167.

Kuczumow, A., Cukrowska, E., Stachniuk, A., Gawęda, R., Mroczka, R., Paszkowicz, W., ... & Backwell, L. (2010). Investigation of chemical changes in bone material from South African fossil hominid deposits. *Journal of Archaeological Science*, *37*(1), 107-115.

Kuman, K., & Clarke, R. J. (2000). Stratigraphy, artefact industries and hominid associations for Sterkfontein, Member 5. *Journal of Human Evolution*, *38*(6), 827-847.

Lacruz, R. S. (2007). Enamel microstructure of the hominid KB 5223 from Kromdraai, South Africa. *American Journal of Physical Anthropology*, *132*(2), 175-182.

Lacy, S. A. (2014). Oral health and its implications in late Pleistocene Western Eurasian humans. St. Louis: Washington University.

Laden, G., & Wrangham, R. (2005). The rise of the hominids as an adaptive shift in fallback foods: plant underground storage organs (USOs) and australopith origins. *Journal of Human Evolution*, *49*(4), 482-498.

Lambert, J. E. (2009). Summary to the symposium issue: Primate fallback strategies as adaptive phenotypic plasticity—scale, pattern, and process. *American Journal of Physical Anthropology*, *140*(4), 759-766.

Lanfranco, L.P., Eggers, S., 2012. Caries through time: an anthropological overview. In: Li, M. (Ed.), *Contemporary Approach to Dental Caries*. INTECH Open Access Publisher.

Lanyon, J. M., & Sanson, G. D. (1986). Koala (*Phascolarctos cinereus*) dentition and nutrition. II. Implications of tooth wear in nutrition. *Journal of Zoology*, *209*(2), 169-181.

Larsen, C. S. (2015). Bioarchaeology: interpreting behavior from the human skeleton. Cambridge: Cambridge University Press.

Larsen, C. S., Shavit, R., & Griffin, M. C. (1991). Dental caries evidence for dietary change: an archaeological context. In: Kelley, M. A., & Larsen, C. S. (Eds.), *Advances in dental anthropology*, 179-202. New York: Wiley-Liss.

Lauc, T., Fornai, C., Premužić, Z., Vodanović, M., Weber, G. W., Mašić, B., & Šikanjić, P. R. (2015). Dental stigmata and enamel thickness in a probable case of congenital syphilis from XVI century Croatia. *Archives of Oral Biology*, *60*(10), 1554-1564.

Lavelle, C. L. B. (1970). Analysis of attrition in adult human molars. *Journal of Dental Research*, *49*(4), 822-828.

Lawn, B. R., Lee, J. J. W., Constantino, P. J., & Lucas, P. W. (2009). Predicting failure in mammalian enamel. *Journal of the Mechanical Behavior of Biomedical Materials*, *2*(1), 33-42.

Lebel, S., Trinkaus, E., Faure, M., Fernandez, P., Guérin, C., Richter, D., ... & Wagner, G. A. (2001). Comparative morphology and paleobiology of Middle Pleistocene human remains from the Bau de l'Aubesier, Vaucluse, France. *Proceedings of the National Academy of Sciences*, *98*(20), 11097-11102.

Ledogar, J. A., Smith, A. L., Benazzi, S., Weber, G. W., Spencer, M. A., Carlson, K. B., ... & Richmond, B. G. (2016). Mechanical evidence that *Australopithecus sediba* was limited in its ability to eat hard foods. *Nature Communications*, *7*, 10596.

Lee-Thorp, J., & Sponheimer, M. (2003). Three case studies used to reassess the reliability of fossil bone and enamel isotope signals for paleodietary studies. *Journal of Anthropological Archaeology*, *22*(3), 208-216.

Lee-Thorp, J. A., Sponheimer, M., & Luyt, J. (2007). Tracking changing environments using stable carbon isotopes in fossil tooth enamel: an example from the South African hominin sites. *Journal of Human Evolution*, 53(5), 595-601.

Legge, S. S. (2012). Dentoalveolar abscess variation among three groups of chimpanzees (*Pan troglodytes schweinfurthii, Pan troglodytes troglodytes*, and *Pan paniscus*). *International Journal of Paleopathology*, *2*(1), 1-9.

Leonard, W. R., Robertson, M. L., Snodgrass, J. J., & Kuzawa, C. W. (2003). Metabolic correlates of hominid brain evolution. *Comparative Biochemistry and Physiology Part A: Molecular & Integrative Physiology*, 136(1), 5-15.

Lev-Tov, N., Smith, P., & Gopher, A. (2003). Dental evidence for dietary practices in the Chalcolithic period: the findings from a burial cave in Peqi'in (northern Israel). *Paleorient*, 29, 121-134.

Liem, K. F. (1980). Adaptive significance of intra-and interspecific differences in the feeding repertoires of cichlid fishes. *American Zoologist*, *20*(1), 295-314.

Lieverse, A. R. (1999). Diet and the aetiology of dental calculus. *International Journal of Osteoarchaeology*, *9*(4), 219-232.

Lillie, M. C. (1996). Mesolithic and Neolithic populations of Ukraine: indications of diet from dental pathology. *Current Anthropology*, *37*(1), 135-142.

Lillie, M. C., & Richards, M. (2000). Stable isotope analysis and dental evidence of diet at the Mesolithic–Neolithic transition in Ukraine. *Journal of Archaeological Science*, 27(10), 965-972.

Linn, J., Srikandi, W., Clarke, N. G., & Smith, T. (1987). Radiographic and visual assessment of alveolar pathology of first molars in dry skulls. *American Journal of Physical Anthropology*, 72(4), 515-521.

Littleton, J., & Frohlich, B. (1989). An analysis of dental pathology and diet on historic Bahrain. *Paléorient*, 15, 59-75.

Littleton, J., & Townsend, G. (2005). Linear enamel hypoplasia and historical change in a central Australian community. *Australian Dental Journal*, 50(2):101-107.

Liu, W., Zhang, Q. C., Wu, X. J., & Zhu, H. (2010). Tooth wear and dental pathology of the Bronze–Iron Age people in Xinjiang, Northwest China: Implications for their diet and lifestyle. *Journal of Comparative Human Biology*, *61*(2), 102-116.

Loch, C., Grando, L. J., Schwass, D. R., Kieser, J. A., Fordyce, R. E., & Simões-Lopes, P. C. (2013). Dental erosion in South Atlantic dolphins (*Cetacea:* Delphinidae): a macro and microscopic approach. *Marine Mammal Science*, *29*(2), 338-347.

Lockwood, C. A., Kimbel, W. H., & Lynch, J. M. (2004). Morphometrics and hominoid phylogeny: support for a chimpanzee–human clade and differentiation among great ape subspecies. *Proceedings of the National Academy of Sciences of the United States of America*, *101*(13), 4356-4360.

Lodge, E., Ross, C., Ortmann, S., & MacLarnon, A. M. (2013). Influence of diet and stress on reproductive hormones in Nigerian olive baboons. *General and Comparative Endocrinology*, 191, 146-154.

Lockwood, C. A., & Tobias, P. V. (1999). A large male hominin cranium from Sterkfontein, South Africa, and the status of *Australopithecus africanus*. *Journal of Human Evolution*, *36*(6), 637-685.

Lous, I. (1970). Masticatory system used as a tool. *Tandlaegebladet*, *74*(1), 1-10.

Lovell, N. C. (1990). Skeletal and dental pathology of free-ranging mountain gorillas. *American Journal of Physical Anthropology*, *81*(3), 399-412.

Lovell, N. C., & Whyte, I. (1999). Patterns of dental enamel defects at ancient Mendes, Egypt. *American Journal of Physical Anthropology*, *110*(1), 69-80.

Lozano, M., Subirà, M. E., Aparicio, J., Lorenzo, C., & Gómez-Merino, G. (2013). Toothpicking and periodontal disease in a Neanderthal specimen from Cova Foradà site (Valencia, Spain). *PloS one*, *8*(10), e76852.

Lucas, P. W., Constantino, P. J., Chalk, J., Ziscovici, C., Wright, B. W., Fragaszy, D. M., ... & Lee, P. K. (2009). Indentation as a technique to assess the mechanical properties of fallback foods. *American Journal of Physical Anthropology*, *140*(4), 643-652.

Lucas, P., Constantino, P., Wood, B., & Lawn, B. (2008). Dental enamel as a dietary indicator in mammals. *BioEssays*, *30*(4), 374-385.

Lukacs, J. R. (1989). Dental paleopathology: methods for reconstructing dietary patterns. *Reconstruction of Life from the Skeleton*, *1*, 261-286.

Lukacs, J. R. (1992). Dental paleopathology and agricultural intensification in South Asia: new evidence from Bronze Age Harappa. *American Journal of Physical Anthropology*, *87*(2), 133-150.

Lukacs, J. R. (1995). The 'caries correction factor': a new method of calibrating dental caries rates to compensate for antemortem loss of teeth. *International Journal of Osteoarchaeology*, *5*(2), 151-156.

Lukacs, J. R. (1996). Sex differences in dental caries rates with the origin of agriculture in South Asia. *Current Anthropology*, *37*(1), 147-153.

Lukacs, J. R. (1999). Enamel hypoplasia in deciduous teeth of great apes: Do differences in defect prevalence imply differential levels of physiological stress? *American Journal of Physical Anthropology*, *110*(3), 351-363.

Lukacs, J. R. (2001). Enamel hypoplasia in the deciduous teeth of great apes: variation in prevalence and timing of defects. *American Journal of Physical Anthropology*, *116*(3), 199-208.

Lukacs, J. R. (2007). Dental trauma and antemortem tooth loss in prehistoric Canary Islanders: prevalence and contributing factors. *International Journal of Osteoarchaeology*, *17*(2), 157-173.

Lunt, D. A. (1974). The prevalence of dental caries in the permanent dentition of Scottish prehistoric and mediaeval populations. *Archives of Oral Biology*, *19*(6), 431-437.

Maat, G. J., & Van der Velde, E. A. (1987). The caries-attrition competition. *International Journal of Anthropology*, *2*(4), 281-292.

Macho, G. (2016). The implications of morphology, mechanics, and microstructure of teeth for understanding dietary drivers in human evolution. In: Lee-Thorp, J., Katzenberg, M. A., (Eds.), *The Oxford Handbook of the Archaeology of Diet*. Oxford: Oxford University Press.

Macho, G. A. (1995). The significance of hominid enamel thickness for phylogenetic and life-history reconstruction. In Cecchi, J. M. (Ed.), *Aspects of Dental Biology: Palaeontology, Anthropology, and Evolution*, 51-68. Florence: Angelo Pontecorbi.

Macho, G. A. (2014). Baboon feeding ecology informs the dietary niche of *Paranthropus boisei*. *PloS one*, *9*(1), e84942.

Macho, G. A. (2015). 1. Can Extant Primates Serve as Models to Determine the Dietary Ecology of Hominins? The Case of *Paranthropines*. *Trends in Biological Anthropology,* 1, 1.

Macho, G. A., & Shimizu, D. (2009). Dietary adaptations of South African australopiths: inference from enamel prism attitude. *Journal of Human Evolution*, *57*(3), 241-247.

Macho, G. A., & Spears, I. R. (1999). Effects of loading on the biochemical behavior of molars of *Homo*, *Pan*, and *Pongo*. *American Journal of Physical Anthropology*, *109*(2), 211-227.

Mahoney, E., Holt, A., Swain, M., & Kilpatrick, N. (2000). The hardness and modulus of elasticity of primary molar teeth: an ultra-micro-indentation study. *Journal of Dentistry*, *28*(8), 589-594.

Mann, A. (1988). The nature of Taung dental maturation. *Nature*, *333*(6169), 123-123.

Manning, J. T., & Chamberlain, A. T. (1993). Fluctuating asymmetry, sexual selection and canine teeth in primates. *Proceedings of the Royal Society of London B: Biological Sciences*, *251*(1331), 83-87.

Mant, M., & Roberts, C. (2015). Diet and dental caries in post-medieval London. *International Journal of Historical Archaeology*, *19*(1), 188-207.

Marchewka, J., Skrzat, J., & Wróbel, A. (2014). Analysis of the enamel hypoplasia using micro-CT scanner versus classical method. *Anthropologischer Anzeiger*, *71*(4), 391-402.

Mårdh, C. K., Bäckman, B., Holmgren, G., Hu, J. C. C., Simmer, J. P., & Forsman-Semb, K. (2002). A nonsense mutation in the enamelin gene causes local hypoplastic autosomal dominant amelogenesis imperfecta (AIH2). *Human Molecular Genetics*, *11*(9), 1069-1074.

Margvelashvili, A., Zollikofer, C. P., Lordkipanidze, D., Peltomäki, T., & de León, M. S. P. (2013). Tooth wear and dentoalveolar remodeling are key factors of morphological variation in the Dmanisi mandibles. *Proceedings of the National Academy of Sciences*, *110*(43), 17278-17283.

Marshall, A. J., & Wrangham, R. W. (2007). Evolutionary consequences of fallback foods. *International Journal of Primatology*, *28*(6), 1219.

Martínez, L. M., Estebaranz-Sánchez, F., Galbany, J., & Pérez-Pérez, A. (2016). Testing dietary hypotheses of east African hominines using buccal dental microwear data. *PloS one*, 11(11), e0165447.

McCance, R. A., Ford, E. H. R., & Brown, W. A. B. (1961). Severe undernutrition in growing and adult animals. *British Journal of Nutrition*, *15*(02), 213-224.

McErlain, D. D., Chhem, R. K., Bohay, R. N., & Holdsworth, D. W. (2004). Micro-computed tomography of a 500-year-old tooth: technical note. *Canadian Association of Radiologists Journal*, *55*(4), 242-245.

McManamon, F. P., Bradley, J. W., & Magennis, A. L. (1986). *The IndianNeck Ossuary (No. 17)*. Division of Cultural Resources, North Atlantic Regional Office, National Park Service, US Dept. of the Interior.

McNulty, K. P., Frost, S. R., & Strait, D. S. (2006). Examining affinities of the Taung child by developmental simulation. *Journal of Human Evolution*, *51*(3), 274-296.

Mehta, D. N., Shah, J., & Thakkar, B. (2013). Amelogenesis imperfecta: Four case reports. *Journal of Natural Science, Biology, and Medicine*, 4(2), 462-465.

Meiklejohn, C., Baldwin, J., & Schentag, C. (1988). Caries as a probable dietary marker in the Western European Mesolithic. In: Kennedy, B. V., LeMoine, G. M., (Eds), *Diet and Subsistence: Current Archaeological Perspectives*, 273-279. Calgary: University of Calgary Archaeologic Association.

Meinl, A., Rottensteiner, G. M., Huber, C. D., Tangl, S., Watzak, G., & Watzek, G. (2010). Caries frequency and distribution in an early medieval Avar population from Austria. *Oral Diseases*, *16*(1), 108-116.

Mellanby, M. (1929). *Diet and the teeth: an experimental study. Part I. Dental structure in dogs*. London: Medical Research Council (H.M.S.O.).

Merceron, G., Viriot, L., & Blondel, C. (2004). Tooth microwear pattern in roe deer (*Capreolus capreolus L.*) from Chizé (Western France) and relation to food composition. *Small Ruminant Research*, *53*(1), 125-132.

Mikuls, T. R., Payne, J. B., Yu, F., Thiele, G. M., Reynolds, R. J., Cannon, G. W., ... & Reimold, A. (2014). Periodontitis and Porphyromonas gingivalis in patients with rheumatoid arthritis. *Arthritis & Rheumatology*, *66*(5), 1090-1100.

Miles, A. E. W., & Grigson, C. (2003). *Colyer's Variations and Diseases of the Teeth of Animals*. Cambridge: Cambridge University Press.

Milner, G. R., & Larsen, C. S. (1991). Teeth as artifacts of human behavior: intentional mutilation and accidental modification. In: Kelley, M. A., & Larsen , C. S. (Eds.), *Advances In Dental Anthropology*, 357-378. Hoboken: Wiley-Liss.

Miszkiewicz, J. J. (2015). Linear Enamel Hypoplasia and Age-at-Death at Medieval (11th–16th Centuries) St. Gregory's Priory and Cemetery, Canterbury, UK. *International Journal of Osteoarchaeology*, *25*(1), 79-87.

Mitani, J. C., Call, J., Kappeler, P. M., Palombit, R. A., & Silk, J. B. (Eds.). (2012). *The evolution of primate societies*. Chicago: University of Chicago Press.

Mjör, I. A., & Karlsen, K. (1970). The interface between dentine and irregular secondary dentine. *Acta Odontologica Scandinavica*, *28*(3), 363-376.

Moggi-Cecchi, J. (2000). Enamel hypoplasia in South African early hominids: A reappraisal. *American Journal of Physical Anthropology*, 30(1), 230-231.

Moggi-Cecchi, J. (2003). The elusive 'second species' in Sterkfontein Member 4: the dental metrical evidence: research articles: human origins research in South Africa. *South African Journal of Science*, 99(5-6), 268-270.

Moggi-Cecchi, J., & Crovella, S. (1991). Occurrence of enamel hypoplasia in the dentitions of simian primates. *Folia Primatologica*, *57*(2), 106-110.

Moggi-Cecchi, J., Grine, F. E., & Tobias, P. V. (2006). Early hominid dental remains from Members 4 and 5 of the Sterkfontein Formation (1966–1996 excavations): catalogue, individual associations, morphological descriptions and initial metrical analysis. *Journal of Human Evolution*, 50(3), 239-328.

Moggi-Cecchi, J., Menter, C., Boccone, S., & Keyser, A. (2010). Early hominin dental remains from the Plio-Pleistocene site of Drimolen, South Africa. *Journal of Human Evolution*, *58*(5), 374-405.

Moggi-Cecchi, J., Pacciani, E., & Pinto-Cisternas, J. (1994). Enamel hypoplasia and age at weaning in 19th-century Florence, Italy. *American Journal of Physical Anthropology*, *93*(3), 299-306.

Moggi-Cecchi, J., Tobias, P. V., & Beynon, A. D. (1998). The mixed dentition and associated skull fragments of a juvenile fossil hominid from Sterkfontein, South Africa. *American Journal of Physical Anthropology*, *106*(4), 425-465.

Molnar, P. (2008). Dental wear and oral pathology: possible evidence and consequences of habitual use of teeth in a Swedish Neolithic sample. *American Journal of Physical Anthropology*, *136*(4), 423-431.

Molnar, P. (2011). Extramasticatory dental wear reflecting habitual behavior and health in past populations. *Clinical Oral Investigations*, *15*(5), 681-689.

Molnar, S. (1971). Human tooth wear, tooth function and cultural variability. *American Journal of Physical Anthropology*, *34*(2), 175-189.

Molnar, S., & Molnar, I. (1985). Observations of dental diseases among prehistoric populations of Hungary. *American Journal of Physical Anthropology*, *67*(1), 51-63.

Molnar, S., Barrett, M. J., Brian, L., Brace, C. L., Brose, D. S., Dewey, J. R., ... & Kennedy, K. A. (1972). Tooth wear and culture: A survey of tooth functions among some prehistoric populations. *Current Anthropology*, *13*(5), 511-526.

Moore, S. (2002). *The relationship between maternal periodontal disease and adverse pregnancy outcome*. Doctoral dissertation, King's College London (University of London).

Morris, A. J., Steele, J., & White, D. A. (2001). Adult dental health survey: The oral cleanliness and periodontal health of UK adults in 1998. *British Dental Journal*, *191*(4), 186-192.

Morse, P. E., Daegling, D. J., McGraw, W. S., & Pampush, J. D. (2013). Dental wear among cercopithecid monkeys of the Taï forest, Côte d'Ivoire. *American Journal of Physical Anthropology*, *150*(4), 655-665.

Moynihan, P. (2000). Foods and factors that protect against dental caries. *Nutrition Bulletin*, *25*(4), 281-286.

Nair, P. N. R. (2004). Pathogenesis of apical periodontitis and the causes of endodontic failures. *Critical Reviews in Oral Biology & Medicine*, 15(6), 348-381.

Neves, A. D. A., Coutinho, E., Cardoso, M. V., Jaecques, S. V., & Van Meerbeek, B. (2010). Micro-CT based quantitative evaluation of caries excavation. *Dental Materials*, *26*(6), 579-588.

Newcomb Jr, W. W. (2010). *The Indians of Texas: from prehistoric to modern times*. Austin: University of Texas Press.

Newman, H. N. (1999). Attrition, eruption, and the periodontium. *Journal of Dental Research*, *78*(3), 730-734.

Newman, H. N., & Levers, B. G. (1979). Tooth eruption and function in an early Anglo-Saxon population. *Journal of the Royal Society of Medicine*, *72*(5), 341.

Newman, M. G., Takei, H., Klokkevold, P. R., & Carranza, F. A. (2011). *Carranza's clinical periodontology*. London, W.B. Saunders Company.

Nikiforuk, G., & Fraser, D. (1979). Etiology of enamel hypoplasia and interglobular dentin: the roles of hypocalcemia and hypophosphatemia. *Metabolic Bone Disease and Related Research*, *2*(1), 17-23.

Nikiforuk, G., & Fraser, D. (1981). The etiology of enamel hypoplasia: a unifying concept. *The Journal of Pediatrics*, *98*(6), 888-893.

Nishikawara, F., Nomura, Y., Imai, S., Senda, A., & Hanada, N. (2007). Evaluation of cariogenic bacteria. *European Journal of Dentistry*, 1(1), 31-39.

Norén, J. G., Odelius, H., Rosander, B., & Linde, A. (1984). SIMS analysis of deciduous enamel from normal full-term infants, low birth weight infants and from infants with congenital hypothyroidism. *Caries Research*, *18*(3), 242-249.

Novak, M. (2015). Dental health and diet in early medieval Ireland. *Archives of Oral Biology*, *60*(9), 1299-1309.

Nystrom, P., Phillips-Conroy, J. E., & Jolly, C. J. (2004). Dental microwear in *anubis* and hybrid baboons (*Papio hamadryas, sensu lato*) living in Awash National Park, Ethiopia. *American Journal of Physical Anthropology*, *125*(3), 279-291.

Ogden, A. (2008). Advances in the palaeopathology of teeth and jaws. In: Pinhasi, R., & Mays, S. (Eds.), *Advances in Human Palaeopathology*, 283-307. Hoboken: John Wiley & Sons.

Ogden, A. R., Pinhasi, R., & White, W. J. (2007). Gross enamel hypoplasia in molars from subadults in a 16th–18th century London graveyard. *American Journal of Physical Anthropology*, *133*(3), 957-966.

Ogilvie, M. D., Curran, B. K., & Trinkaus, E. (1989). Incidence and patterning of dental enamel hypoplasia among the Neandertals. *American Journal of Physical Anthropology*, *79*(1), 25-41.

Oginni, A. O., Olusile, A. O., & Udoye, C. I. (2003). Non-carious cervical lesions in a Nigerian population: Abrasion or abfraction? *International Dental Journal*, *53*(5), 275-279.

Oppenheimer, A. (1964). Tool use and crowded teeth in Australopithecinae. *Current Anthropology*, *5*(5), 419-421.

Ortner, D. J. (2003). *Identification of pathological conditions in human skeletal remains*. Cambridge: Academic Press.

Ozdemir, D., Hart, P. S., Firatli, E., Aren, G., Ryu, O. H., & Hart, T. C. (2005). Phenotype of ENAM mutations is dosage-dependent. *Journal of Dental Research*, *84*(11), 1036-1041.

Page, R. C., & Kornman, K. S. (2000). The pathogenesis of human periodontitis: an introduction. *Periodontology*, *14*(1), 9-11.

Paine, M. L., White, S. N., Luo, W., Fong, H., Sarikaya, M., & Snead, M. L. (2001). Regulated gene expression dictates enamel structure and tooth function. *Matrix Biology*, *20*(5), 273-292.

Palamra, J., Phakey, P. P., Rachinger, W. A., & Orams, H. J. (1981). The ultrastructure of enamel from the kangaroo *Macropus giganteus*: Tubular enamel and its' black spots' defect. *Australian Journal of Zoology*, *29*(4), 643-652.

Pampush, J. D., Spradley, J. P., Morse, P. E., Harrington, A. R., Allen, K. L., Boyer, D. M., & Kay, R. F. (2016). Wear and its effects on dental topography measures in howling monkeys (*Alouatta palliata*). *American Journal of Physical Anthropology*, *161*(4), 705-721.

Pap, I., Tillier, A. M., Arensburg, B., Weiner, S., & Chech, M. (1995). First scanning electron microscope analysis of dental calculus from European Neanderthals: Subalyuk, (Middle Paleolithic, Hungary). *Bulletins et Mémoires de la Société d'Anthropologie de Paris*, *7*(1), 69-72.

Patterson Jr, D. K. (1984). *A diachronic study of dental palaeopathology and attritional status of prehistoric Ontario pre-Iroquois and Iroquois populations*. Toronto: Archaeological Survey of Canada.

Patterson, B. D., Neiburger, E. J., & Kasiki, S. M. (2003). Tooth breakage and dental disease as causes of carnivore–human conflicts. *Journal of Mammalogy*, *84*(1), 190-196.

Pechenkina, E. A., Benfer, R. A., & Zhijun, W. (2002). Diet and health changes at the end of the Chinese Neolithic: the Yangshao/Longshan transition in Shaanxi province. *American Journal of Physical Anthropology*, *117*(1), 15-36.

Pedersen, P. O. (1944). Taendernes tilstand hos 2–6 aarige børn. *Tandlaegebladet*, *48*, 485-565.

Pederson, P. O., & Rasch, G. (1947). The tooth morphologic basis of caries recording. *Odontol Tidskr*, *45*(1).

Peters, C. R. (1987). Nut-like oil seeds: Food for monkeys, chimpanzees, humans, and probably ape-men. *American Journal of Physical Anthropology*, *73*(3), 333-363.

Pfretzschner, H. U. (2004). Fossilization of Haversian bone in aquatic environments. *Comptes Rendus Palevol*, *3*(6), 605-616.

Phillips, C., & Lancelotti, C. (2014). Chimpanzee diet: phytolithic analysis of feces. *American Journal of Primatology*, 76(8), 757-773.

Pickering, R., Dirks, P. H., Jinnah, Z., De Ruiter, D. J., Churchill, S. E., Herries, A. I., ... & Berger, L. R. (2011). *Australopithecus sediba* at 1.977 Ma and implications for the origins of the genus *Homo*. *Science*, *333*(6048), 1421-1423.

Pickering, T. R. (2001). Taphonomy of the Swartkrans hominid postcrania and its bearing on issues of meat-eating and fire management. In: Stanford, C. B., & Bunn, H. T. (Eds.), *Meat-Eating and Human Evolution*, 33-51. Oxford: Oxford University Press.

Pickering, T. R., & Carlson, K. J. (2002). Baboon bone mineral densities: implications for the taphonomy of primate skeletons in South African cave sites. *Journal of Archaeological Science*, *29*(8), 883-896.

Pickering, T. R., Domínguez-Rodrigo, M., Egeland, C. P., & Brain, C. K. (2004). Beyond leopards: tooth marks and the contribution of multiple carnivore taxa to the accumulation of the Swartkrans Member 3 fossil assemblage. *Journal of Human Evolution*, 46(5), 595-604.

Pickering, T. R., Dominguez-Rodrigo, M., Egeland, C. P., & Brain, C. K. (2005). The contribution of limb bone fracture patterns to reconstructing early hominid behaviour at Swartkrans Cave (South Africa): archaeological application of a new analytical method. *International Journal of Osteoarchaeology*, *15*(4), 247.

Pickering, T. R., Domínguez-Rodrigo, M., Egeland, C. P., Brain, C. K., & Kuman, K. (2007). Carcass foraging by early hominids at Swartkrans Cave (South Africa): a new investigation of the zooarchaeology and taphonomy of Member 3. In: Pickering, T. R., Schick, K. D., & Toth, N. P. (Eds.), *breathing life into fossils: taphonomic studies in honor of CK ("Bob") Brain*, 233-254. Gosport: Stone Age Institute Press.

Pickering, T. R., Egeland, C. P., Domínguez-Rodrigo, M., Brain, C. K., & Schnell, A. G. (2008). Testing the "shift in the balance of power" hypothesis at Swartkrans, South Africa: hominid cave use and subsistence behavior in the early Pleistocene. *Journal of Anthropological Archaeology*, *27*(1), 30-45.

Pickering, T. R., White, T. D., & Toth, N. (2000). Brief communication: Cutmarks on a Plio-Pleistocene hominid from Sterkfontein, South Africa. *American Journal of Physical Anthropology*, *111*(4), 579-584.

Picton, D. C. A. (1957). Calculus, wear and alveolar bone loss in the jaws of sixth-century Jutes. *Dental Practice, 7*, 301-303.

Pindborg, J. J. (1970). *Pathology of the dental hard tissues*. Philadelphia: W. B. Saunders Company.

Pineda-Munoz, S., Lazagabaster, I. A., Alroy, J., & Evans, A. R. (2016). Inferring diet from dental morphology in terrestrial mammals. *Methods in Ecology and Evolution*, 8, 481–491.

Pinhasi, R., Shaw, P., White, B., & Ogden, A. R. (2006). Morbidity, rickets and long-bone growth in post-medieval Britain—a cross-population analysis. *Annals of Human Biology*, *33*(3), 372-389.

Pisanty, S., & Garfunkel, A. (1977). Familial hypoparathyroidism with candidiasis and mental retardation. *Oral Surgery, Oral Medicine, Oral Pathology*, *44*(3), 374-383.

Poulter, J. A., El-Sayed, W., Shore, R. C., Kirkham, J., Inglehearn, C. F., & Mighell, A. J. (2014). Whole-exome sequencing, without prior linkage, identifies a mutation in LAMB3 as a cause of dominant hypoplastic amelogenesis imperfecta. *European Journal of Human Genetics*, *22*(1), 132-135.

Prowse, T. L., Saunders, S. R., Schwarcz, H. P., Garnsey, P., Macchiarelli, R., & Bondioli, L. (2008). Isotopic and dental evidence for infant and young child feeding practices in an imperial Roman skeletal sample. *American Journal of Physical Anthropology*, *137*(3), 294-308.

Purvis, R. J., MacKay, G. S., Cockburn, F., Barrie, W. M., Wilkinson, E. M., Belton, N. R., & Forfar, J. O. (1973). Enamel hypoplasia of the teeth associated with neonatal tetany: a manifestation of maternal vitamin-D deficiency. *The Lancet*, *302*(7833), 811-814.

Rabenold, D., & Pearson, O. M. (2011). Abrasive, silica phytoliths and the evolution of thick molar enamel in primates, with implications for the diet of *Paranthropus boisei*. *PLoS One*, *6*(12), e28379.

Radu, C., & Soficaru, A. D. (2016). Dental developmental defects in a subadult from 16th–19th centuries Bucharest, Romania. *International Journal of Paleopathology*, *15*, 33-38.

Rak, Y. (1983). *The Australopithecine Face*. New York: Academic Press.

Randolph-Quinney, P. S., Williams, S. A., Steyn, M., Meyer, M. R., Smilg, J. S., Churchill, S. E., ... & Berger, L. R. (2016). Osteogenic tumour in *Australopithecus sediba*: Earliest hominin evidence for neoplastic disease. *South African Journal of Science*, *112*(7-8), 1-7.

Raupp, M. J. (1985). Effects of leaf toughness on mandibular wear of the leaf beetle, *Plagiodera versicolora*. *Ecological Entomology*, *10*(1), 73-79.

Ravosa, M. J., Kunwar, R., Stock, S. R., & Stack, M. S. (2007). Pushing the limit: masticatory stress and adaptive plasticity in mammalian craniomandibular joints. *Journal of Experimental Biology*, *210*(4), 628-641.

Raymond, H. (1971). Is the new composite cranium from Swartkrans a small robust australopithecine? *Nature*, 230(5293), 398-401.

Reed, K. E., Kitching, J. W., Grine, F. E., Jungers, W. L., & Sokoloff, L. (1993). Proximal femur of *Australopithecus africanus* from member 4, Makapansgat, South Africa. *American Journal of Physical Anthropology*, *92*(1), 1-15.

Reid, D. J., & Dean, M. C. (2000). Brief communication: the timing of linear hypoplasias on human anterior teeth. *American Journal of Physical Anthropology*, *113*(1), 135-139.

Ricci, S., Capecchi, G., Boschin, F., Arrighi, S., Ronchitelli, A., & Condemi, S. (2014). Toothpick use among epigravettian humans from Grotta Paglicci (Italy). *International Journal of Osteoarchaeology*, 26(2), 281-289.

Ricucci, D., Loghin, S., Lin, L. M., Spångberg, L. S., & Tay, F. R. (2014). Is hard tissue formation in the dental pulp after the death of the primary odontoblasts a regenerative or a reparative process? *Journal of Dentistry*, *42*(9), 1156-1170.

Ricucci, D., Pascon, E. A., Ford, T. R. P., & Langeland, K. (2006). Epithelium and bacteria in periapical lesions. *Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology, and Endodontology*, 101(2), 239-249.

Riede, F., & Wheeler, J. M. (2009). Testing the 'Laacher See hypothesis': tephra as dental abrasive. *Journal of Archaeological Science*, *36*(10), 2384-2391.

Rightmire, G. P., & Deacon, H. J. (2001). New human teeth from Middle Stone Age deposits at Klasies River, South Africa. *Journal of Human Evolution*, *41*(6), 535-544.

Rightmire, G. P., Deacon, H. J., Schwartz, J. H., & Tattersall, I. (2006). Human foot bones from Klasies River main site, South Africa. *Journal of Human Evolution*, *50*(1), 96-103.

Ripamonti, U. (1988). Paleopathology in *Australopithecus africanus*: A suggested case of a 3-million-year-old prepubertal periodontitis. *American Journal of Physical Anthropology*, *76*(2), 197-210.

Ritter, A. V., Grippo, J. O., Coleman, T. A., & Morgan, M. E. (2009). Prevalence of carious and non-carious cervical lesions in archaeological populations from North America and Europe. *Journal of Esthetic and Restorative Dentistry*, *21*(5), 324-334.

Robb, N. D., Cruwys, E., & Smith, B. G. N. (1991). Regurgitation erosion as a possible cause of tooth wear in ancient British populations. *Archives of Oral Biology*, *36*(8), 595-602.

Robinson, B. W., & Wilson, D. S. (1998). Optimal foraging, specialization, and a solution to Liem's paradox. *The American Naturalist*, *151*(3), 223-235.

Robinson, J. T. (1952). Some hominid features of the ape-man dentition. *Journal of the Dental Association of South Africa*, 7: 102–13.

Robinson, J. T. (1954). Prehominid dentition and hominid evolution. *Evolution*, 8(4), 324-334.

Robinson, J. T. (1956). The dentition of the *Australopithecinae*: Maxillary molars. *Transvaal Museum Memoirs*, 9(1), 81-100.

Robinson, J. T. (1963). Australopithecines, culture and phylogeny. *American Journal of Physical Anthropology*, 21(4), 595-605.

Robles, M., Ruiz, M., Bravo Pérez, M., González, E., & Peñalver, M. (2013). Prevalence of enamel defects in primary and permanent teeth in a group of schoolchildren from Granada (Spain). *Oral Medicine and Pathology*, 18(2), 187-193.

Rogers, M. E., Abernethy, K., Bermejo, M., Cipolletta, C., Doran, D., Mcfarland, K., ... & Tutin, C. E. (2004). Western gorilla diet: a synthesis from six sites. *American Journal of Primatology*, *64*(2), 173-192.

Rohnbogner, A., & Lewis, M. (2016). Dental caries as a measure of diet, health, and difference in non-adults from urban and rural Roman Britain. *Dental Anthropology*, 29(1), 16-31.

Roney, J. G. (1959). Palaeopathology of a California archaeological site. *Bulletin of the History of Medicine*, *33*, 97-109.

Rossi, M., Casali, F., Romani, D., Bondioli, L., Macchiarelli, R., & Rook, L. (2004). MicroCT Scan in paleobiology: application to the study of dental tissues. *Nuclear Instruments and Methods in Physics Research Section B: Beam Interactions with Materials and Atoms*, *213*, 747-750.

Rushton, M. A. (1964). Hereditary enamel defects. *Proceedings of the Royal Society of Medicine*, *57*(1), 53-58.

Rutherford, B., Spångberg, L., Tucker, M., & Charette, M. (1995). Transdentinal stimulation of reparative dentine formation by osteogenic protein-1 in monkeys. *Archives of Oral Biology*, *40*(7), 681-683.

Sagne, S., & Olsson, G. (1977). Studies of the periodontal status of a medieval population. *Dentomaxillofacial Radiology*, *6*(1), 46-52.

Sala, N., Cuevas, J. & López, N. (2007). Estudio paleopatológico de una hemimandíbula de Tethytragus (Artiodactyla, Mammalia) del Mioceno Medio de Somosaguas (Pozuelo de Alarcón, Madrid). *Colóquios de Paleontología*, 57, 7-14.

Sarnat, B. G., & Schour, I. (1941). Enamel hypoplasia (chronologic enamel aplasia) in relation to systemic disease: a chronologic, morphologic and etiologic classification. *The Journal of the American Dental Association*, 28(12), 1989-2000.

Sauther, M. L., Sussman, R. W., & Cuozzo, F. (2002). Dental and general health in a population of wild ring-tailed lemurs: a life history approach. *American Journal of Physical Anthropology*, *117*(2), 122-132.

Scannapieco, F. A., & Ho, A. W. (2001). Potential associations between chronic respiratory disease and periodontal disease: analysis of National Health and Nutrition Examination Survey III. *Journal of Periodontology*, *72*(1), 50-56.

Schatz, D., Alfter, G., & Göz, G. (2001). Fracture resistance of human incisors and premolars: morphological and patho-anatomical factors. *Dental Traumatology*, *17*(4), 167-173.

Scheutzel, P., & Ritter, W. (1989). Alterations of teeth and jaws in children with chronic renal failure. *Deutsche Zahnarztliche Zeitschrift*, *44*(2), 115-118.

Schmid, P., Churchill, S. E., Nalla, S., Weissen, E., Carlson, K. J., de Ruiter, D. J., & Berger, L. R. (2013). Mosaic morphology in the thorax of *Australopithecus sediba*. *Science*, *340*(6129), 1234598.

Schoeninger, M. J., Moore, J., & Sept, J. M. (1999). Subsistence strategies of two "savanna" chimpanzee populations: The stable isotope evidence. *American Journal of Primatology*, 49(4), 297-314.

Schollmeyer, K. G., & Turner, C. G. (2004). Dental caries, prehistoric diet, and the pithouse-to-pueblo transition in southwestern Colorado. *American Antiquity*, 69(3), 569-582.

Schour, I., & Massler, M. (1945). The Effects of Dietary Deficiencies Upon the Oral Structures. III. *The Journal of the American Dental Association*, *32*(15), 1022-1030.

Schultz, A. H. (1956). The occurrence and frequency of pathological and teratological conditions and of twinning among non-human primates. *Primatolo Gia*, 965-1014.

Schuurs, A. (2012). *Pathology of the hard dental tissues*. Hoboken: John Wiley & Sons.

Schwartz, J. H., & Tattersall, I. (2003). *The human fossil record, craniodental morphology of genus Homo (Africa and Asia), Vol. 2*. New York: Wiley.

Scott, E. C. (1979). Dental wear scoring technique. *American Journal of Physical Anthropology*, *51*(2), 213-217.

Scott, G. R., & Winn, J. R. (2011). Dental chipping: contrasting patterns of microtrauma in Inuit and European populations. *International Journal of Osteoarchaeology*, *21*(6), 723-731.

Scott, R. S., Ungar, P. S., Bergstrom, T. S., Brown, C. A., Grine, F. E., Teaford, M. F., & Walker, A. (2005). Dental microwear texture analysis shows within-species diet variability in fossil hominins. *Nature*, *436*(7051), 693-695.

Seow, W. K. (1990). Enamel hypoplasia in the primary dentition: a review. *Journal of Dentistry for Children*, *58*(6), 441-452.

Seow, W. K. (1992). Clinical diagnosis and management strategies of amelogenesis imperfecta variants. *Pediatric Dentistry*, *15*(6), 384-393.

Seow, W. K., Brown, J. P., Tudehope, D. A., & O'Callaghan, M. (1984). Dental defects in the deciduous dentition of premature infants with low birth weight and neonatal rickets. *Pediatric Dentistry*, *6*(2), 88-92.

Shellis, R. P., & Addy, M. (2014). The interactions between attrition, abrasion and erosion in tooth wear. *Erosive Tooth Wear*, *25*, 32-45.

Shen, S., Samaranayake, L. P., & Yip, H. K. (2004). In vitro growth, acidogenicity and cariogenicity of predominant human root caries flora. *Journal of Dentistry*, *32*(8), 667-678.

Shen, S., Samaranayake, L. P., & Yip, H. K. (2005). Coaggregation profiles of the microflora from root surface caries lesions. *Archives of Oral Biology*, *50*(1), 23-32.

Shivhare, P., Shankarnarayan, L., Gupta, A., & Sushma, P. (2016). Amelogenesis Imperfecta: A Review. *Journal of Advanced Oral Research*, *7*(1), 1-6.

Shykoluk, N. L., & Lovell, N. C. (2010). Technical note: Enhancement of Scott's molar wear scoring method. *American Journal of Physical Anthropology*, *143*(3), 482-487.

Silberman, S. L., Trubman, A., Duncan, W. K., & Meydrech, E. F. (1991). Prevalence of primary canine hypoplasia of the mandibular teeth. *Pediatric Dentistry*, *13*, 356-60.

Silva, A. M., Gil, P., Soares, J., & da Silva, C. T. (2016). Short report: Evidence of non-masticatory dental use in Bronze Age individuals exhumed from the Necropolis of Casas Velhas (Portugal). *Bulletin of the International Association for Paleodontology*, *10*(1), 31-38.

Skinner, M. F., Hadaway, W., Dickie, J., (1994). Effects of ethnicity, nutrition and birth month on localised enamel hypoplasia of the primary canine. J*ournal of Dentistry for Children,* 61:109 –113.

Skinner, M. (1996). Developmental stress in immature hominines from Late Pleistocene Eurasia: evidence from enamel hypoplasia. *Journal of Archaeological Science*, *23*(6), 833-852.

Skinner, M. F. (1986). An enigmatic hypoplastic defect of the deciduous canine. *American Journal of Physical Anthropology*, *69*(1), 59-69.

Skinner, M. F., & Hung, J. T. W. (1989). Social and biological correlates of localized enamel hypoplasia of the human deciduous canine tooth. *American Journal of Physical Anthropology*, *79*(2), 159-175.

Skinner, M. F., & Newell, E. A. (2003). Localized hypoplasia of the primary canine in bonobos, orangutans, and gibbons. *American Journal of Physical Anthropology*, *120*(1), 61-72.

Skinner, M. F., & Sperber, G. H. (1982). *Atlas of radiographs of early man*. New York: Alan R. Liss, Inc.

Skinner, M. F., Dupras, T. L., & Moya-Sola, S. (2015). Periodicity of linear enamel hypoplasia among Miocene Dryopithecus from Spain. *Journal of Paleopathology*, 7(3), 195-222.

Skinner, M. F., Skinner, M. M., Pilbrow, V. C., & Hannibal, D. L. (2016). An enigmatic hypoplastic defect of the maxillary lateral incisor in recent and fossil orangutans from Sumatra (*Pongo abelii*) and Borneo (*Pongo pygmaeus*). *International Journal of Primatology*, *37*(4-5), 548-567.

Skinner, M. M., Lockey, A. L., Gunz, P., Hawks, J., & Delezene, L. K. (2016). Enamel-dentine junction morphology and enamel thickness of the Dinaledi dental collection. A*merican Journal of Physical Anthropology*, 159, 293-293.

Slade, G. D., Sanders, A. E., Do, L., Roberts-Thomson, K., & Spencer, A. J. (2013). Effects of fluoridated drinking water on dental caries in Australian adults. *Journal of Dental Research*, *92*(4), 376-382.

Šlaus, M., Bedić, Ž., Rajić Šikanjić, P., Vodanović, M., & Domić Kunić, A. (2011). Dental health at the transition from the Late Antique to the early Medieval period on Croatia's eastern Adriatic coast. *International Journal of Osteoarchaeology*, *21*(5), 577-590.

Slaus, M., Pećina-Hrncević, A., & Jakovljević, G. (1997). Dental disease in the late Medieval population from Nova Raca, Croatia. *Collegium Antropologicum*, *21*(2), 561-572.

Smith, B. H. (1984). Patterns of molar wear in hunter–gatherers and agriculturalists. *American Journal of Physical Anthropology*, *63*(1), 39-56.

Smith, B. H. (1991). *Standards of human tooth formation and dental age assessment*. New York: Wiley-Liss.

Smith, C. E., Murillo, G., Brookes, S. J., Poulter, J. A., Silva, S., Kirkham, J., ... & Mighell, A. J. (2016). Deletion of amelotin exons 3–6 is associated with amelogenesis imperfecta. *Human Molecular Genetics*, 25(16), 3578–3587.

Smith, F. N., & Lang, N. P. (1977). Lymphocyte blastogenesis to plaque antigens in human periodontal disease. *Journal of Periodontal Research*, *12*(4), 310-317.

Smith, P. (1972). Diet and attrition in the Natufians. *American Journal of Physical Anthropology*, *37*(2), 233-238.

Smith, P., & Avishai, G. (2005). The use of dental criteria for estimating postnatal survival in skeletal remains of infants. *Journal of Archaeological Science*, *32*(1), 83-89.

Smith, T. M., & Tafforeau, P. (2008). New visions of dental tissue research: tooth development, chemistry, and structure. *Evolutionary Anthropology: Issues, News, and Reviews*, *17*(5), 213-226.

Smith, T. M., Tafforeau, P., Le Cabec, A., Bonnin, A., Houssaye, A., Pouech, J., ... & Menter, C. G. (2015). Dental ontogeny in Pliocene and early Pleistocene hominins. *PloS one*, 10(2), e0118118.

Sognnaes, R. F. (1956). Histologic evidence of developmental lesions in teeth originating from Paleolithic, prehistoric, and ancient man. *The American Journal of Pathology*, *32*(3), 547-577.

Sponheimer, M., Alemseged, Z., Cerling, T. E., Grine, F. E., Kimbel, W. H., Leakey, M. G., ... & Wynn, J. G. (2013). Isotopic evidence of early hominin diets. *Proceedings of the National Academy of Sciences*, *110*(26), 10513-10518.

Sponheimer, M., & Lee-Thorp, J. A. (1999). Isotopic evidence for the diet of an early hominid, *Australopithecus africanus*. *Science*, *283*(5400), 368-370.

Sponheimer, M., Lee-Thorp, J., de Ruiter, D., Codron, D., Codron, J., Baugh, A. T., & Thackeray, F. (2005). Hominins, sedges, and termites: new carbon isotope data from the Sterkfontein valley and Kruger National Park. *Journal of Human Evolution*, *48*(3), 301-312.

Sponheimer, M., Passey, B. H., De Ruiter, D. J., Guatelli-Steinberg, D., Cerling, T. E., & Lee-Thorp, J. A. (2006). Isotopic evidence for dietary variability in the early hominin *Paranthropus robustus*. *Science*, 314(5801), 980-982.

Spradley, J. P., Glander, K. E., & Kay, R. F. (2016). Dust in the wind: How climate variables and volcanic dust affect rates of tooth wear in Central American howling monkeys. *American Journal of Physical Anthropology*, *159*(2), 210-222.

Srejić, M. D. (2001). Dental paleopathology in a Serbian medieval population. *Anthropologischer Anzeiger*, 59(2), 113-122.

Stamm, J. W., Banting, D. W., & Imrey, P. B. (1990). Adult root caries survey of two similar communities with contrasting natural water fluoride levels. *The Journal of the American Dental Association*, *120*(2), 143-149.

Stanley, H. R., White, C. L., & McCray, L. (1966). The rate of tertiary (reparative) dentine formation in the human tooth. *Oral Surgery, Oral Medicine, Oral Pathology*, *21*(2), 180-189.

Steininger, C., Berger, L. R., & Kuhn, B. F. (2008). A partial skull of *Paranthropus robustus* from Cooper's Cave, South Africa: research letter. *South African Journal of Science*, *104*(3-4), 143-146.

Stimmler, L., Snodgrass, G. J. A. I., & Jaffe, E. (1973). Dental defects associated with neonatal symptomatic hypocalcaemia. *Archives of Disease in Childhood*, *48*(3), 217-220.

Stojanowski, C. M., Johnson, K. M., & Kathleen, S. (2015). Indicators of idiosyncratic behavior in the dentition. In: Irish, J. D. & Scott, G. R. (Eds.), *A companion to dental anthropology*, 337-395. Hoboken: Wiley-Blackwell.

Strait, D. S., Weber, G. W., Constantino, P., Lucas, P. W., Richmond, B. G., Spencer, M. A., ... & Wood, B. A. (2012). Microwear, mechanics and the feeding adaptations of *Australopithecus africanus*. *Journal of Human Evolution*, 62(1), 165-168.

Strait, D. S., Weber, G. W., Neubauer, S., Chalk, J., Richmond, B. G., Lucas, P. W., ... & Grosse, I. R. (2009). The feeding biomechanics and dietary ecology of *Australopithecus africanus*. *Proceedings of the National Academy of Sciences*, *106*(7), 2124-2129.

Sun, C., Xing, S., Martín-Francés, L., Bae, C., Liu, L., Wei, G., & Liu, W. (2014). Interproximal grooves on the Middle Pleistocene hominin teeth from Yiyuan, Shandong Province: New evidence for tooth-picking behavior from eastern China. *Quaternary International*, *354*, 162-168.

Sundell, S., & Koch, G. (1984). Hereditary amelogenesis imperfecta. I. Epidemiology and clinical classification in a Swedish child population. *Swedish Dental Journal*, *9*(4), 157-169.

Swain, M. V., & Xue, J. (2009). State of the art of Micro-CT applications in dental research. *International Journal of Oral Science*, *1*(4), 177.

Takahashi, N., & Nyvad, B. (2016). Ecological hypothesis of dentin and root caries. *Caries Research*, *50*(4), 422-431.

Tayles, N., Domett, K., & Nelsen, K. (2000). Agriculture and dental caries? The case of rice in prehistoric Southeast Asia. *World Archaeology*, *32*(1), 68-83.

Taylor, A. B. (2006). Feeding behavior, diet, and the functional consequences of jaw form in orangutans, with implications for the evolution of *Pongo*. *Journal of Human Evolution*, *50*(4), 377-393.

Teaford, M. F. (1982). Differences in molar wear gradient between juvenile macaques and langurs. *American Journal of Physical Anthropology*, *57*(3), 323-330.

Teaford, M. F. (1983). Differences in molar wear gradient between adult macaques and langurs. *International Journal of Primatology*, *4*(4), 427-444.

Teaford, M. F., & Glander, K. E. (1996). Dental microwear and diet in a wild population of mantled howling monkeys (*Alouatta palliata*). *Adaptive Radiations of Neotropical Primates*, 433-449.

Teaford, M. F., & Robinson, J. G. (1989). Seasonal or ecological differences in diet and molar microwear in *Cebus nigrivittatus*. *American Journal of Physical Anthropology*, *80*(3), 391-401.

Ten Cate, A. R., & Nanci, A. (1994). Structure of the oral tissues. In: Ten Cate, A. R. (Ed.), *Oral Histology: Development, Structure, Function*, 45–57. St. Louis: Mosby Year Book.

Thomas, C., Sloane, B., & Phillpotts, C. (1997). *Excavations at the priory and hospital of St Mary Spital, London*. London: Museum of London Archaeology Service.

Thomas, H. C. (2000). *Mechanical behavior of materials*. New York: Mc Graw-Hill Publishing.

Thomaz, E. B., Cangussu, M. C. T., Silva, A. A. M. D., & Assis, A. M. O. (2010). Is malnutrition associated with crowding in permanent dentition? *International Journal of Environmental Research and Public Health*, *7*(9), 3531-3544.

Thylstrup, A., & Fejerskov, O. (1978). Clinical appearance of dental fluorosis in permanent teeth in relation to histologic changes. *Community Dentistry and Oral Epidemiology*, *6*(6), 315-328.

Tillier, A. M., Arensburg, B., Rak, Y., & Vandermeersch, B. (1995). Middle Palaeolithic dental caries: new evidence from Kebara (Mount Carmel, Israel). *Journal of Human Evolution*, *29*(2), 189-192.

Tobias, P. V. (1965). *Australopithecus*, *Homo habilis*, tool-using and tool-making. *The South African Archaeological Bulletin*, *20*(80), 167-192.

Tobias, P. V. (1967). *Olduvai Gorge (Vol. 2)*. Cambridge: Cambridge University Press.

Tobias, P. V. (1978). The earliest Transvaal members of the genus Homo with another look at some problems of hominid taxonomy and systematics. *Zeitschrift für Morphologie und Anthropologie*, 69(3), 225-265.

Tobias, P. V. (1991). *Olduvai Gorge IV: The skulls, endocasts and teeth of Homo habilis*. Cambridge: Cambridge University Press.

Tobias, P. V., & Hughes, A. R. (1969). The New Witwatersrand University excavation at Sterkfontein: progress report, some problems and first results. *The South African Archaeological Bulletin*, 95/96(24), 158-169.

Tomczyk, J., Szostek, K., Komarnitki, I., Mańkowska-Pliszka, H., & Zalewska, M. (2013). Dental caries and chemical analyses in reconstruction of diet, health and hygienic behaviour in the Middle Euphrates valley (Syria). *Archives of Oral Biology*, *58*(6), 740-751.

Tomes, C. S. (1898). Partial suppression of teeth in a very hairy monkey (*Colobus caudatus*). *Transactions Odontoid Soiety Great Britain*, 30, 30-35.

Tonge, C. H., & McCance, R. A. (1973). Normal development of the jaws and teeth in pigs, and the delay and malocclusion produced by calorie deficiencies. *Journal of Anatomy*, *115*(1), 1-22.

Towle, I., Irish, J. D., & De Groote, I. (2017). Behavioral inferences from the high levels of dental chipping in *Homo naledi*. *American Journal of Physical Anthropology*, https://doi.org/10.1002/ajpa.23250.

Trinkaus, E., Smith, R. J., & Lebel, S. (2000). Dental caries in the Aubesier 5 Neandertal primary molar. *Journal of Archaeological Science*, *27*(11), 1017-1021.

Turner, C. G. (1979). Dental anthropological indications of agriculture among the Jomon people of central Japan. X. Peopling of the Pacific. *American Journal of Physical Anthropology*, *51*(4), 619-635.

Turner, C. G. I., & Cacciatore, E. (1998). Interproximal tooth grooves in Pacific Basin, East Asian, and new world populations. *Anthropological Science*, *106*, 85-94.

Turner, C. G., & Cadien, J. D. (1969). Dental chipping in Aleuts, Eskimos and Indians. *American Journal of Physical Anthropology*, *31*(3), 303-310.

Turner, C. G., Nichol, C. R., & Scott, G. R. (1991). Scoring procedures for key morphological traits of the permanent dentition: the Arizona State University dental anthropology system. In: Kelley, M. A., & Larsen, C. S. (Eds.), *Advances in dental anthropology*. New York: Wiley-Liss, *24*, 13-31.

Tutin, C. E. (1996). Ranging and social structure of lowland gorillas in the Lopé Reserve, Gabon. In: Toshisada, N., McGrew, W. C., Marchant, L. F. (Eds.), *Great ape societies*. Cambridge: Cambridge University Press, 58-70.

Ubelaker, D. H., Phenice, T. W., & Bass, W. M. (1969). Artificial interproximal grooving of the teeth in American Indians. *American Journal of Physical Anthropology*, *30*(1), 145-149.

Ungar, P. (2004). Dental topography and diets of *Australopithecus afarensis* and early *Homo*. *Journal of Human Evolution*, 46(5), 605-622.

Ungar, P. S. (1994). Patterns of ingestive behavior and anterior tooth use differences in sympatric anthropoid primates. *American Journal of Physical Anthropology*, *95*(2), 197-219.

Ungar, P. S. (2015). Mammalian dental function and wear: a review. *Biosurface and Biotribology*, *1*(1), 25-41.

Ungar, P. S., & Grine, F. E. (1991). Incisor size and wear in *Australopithecus africanus* and *Paranthropus robustus*. *Journal of Human Evolution*, *20*(4), 313-340.

Ungar, P. S., Grine, F. E., Teaford, M. F., & Pérez-Pérez, A. (2001). A review of interproximal wear grooves on fossil hominin teeth with new evidence from Olduvai Gorge. *Archives of Oral Biology*, *46*(4), 285-292.

Ungar, P. S., & M'kirera, F. (2003). A solution to the worn tooth conundrum in primate functional anatomy. *Proceedings of the National Academy of Sciences*, *100*(7), 3874-3877.

Ungar, P. S., & Williamson, M. (2000). Exploring the effects of tooth wear on functional morphology: a preliminary study using dental topographic analysis. *Palaeontologia Electronica*, *3*(1), 1-18.

Van Der Merwe, N. J., Thackeray, J. F., Lee-Thorp, J. A., & Luyt, J. (2003). The carbon isotope ecology and diet of *Australopithecus africanus* at Sterkfontein, South Africa. *Journal of Human Evolution*, *44*(5), 581-597.

Van Valkenburgh, B. (1988). Incidence of tooth breakage among large, predatory mammals. *The American Naturalist*, *131*(2), 291-302.

Van Valkenburgh, B. (2009). Costs of carnivory: tooth fracture in Pleistocene and Recent carnivorans. *Biological Journal of the Linnean Society*, *96*(1), 68-81.

Vandermeersch, B., Arensburg, B., Tillier, A. M., Rak, Y., Weiner, S., Spiers, M., & Aspillaga, E. (1994). Middle Palaeolithic dental bacteria from Kebara, Israel. *Comptes rendus de l'Académie des sciences. Série 2. Sciences de la terre et des planètes*, *319*(6), 727-731.

Varrela, T. M. (1991). Prevalence and distribution of dental caries in a late medieval population in Finland. *Archives of Oral Biology*, *36*(8), 553-559.

Veiberg, V., Loe, L. E., Mysterud, A., Solberg, E. J., Langvatn, R., & Stenseth, N. C. (2007). The ecology and evolution of tooth wear in red deer and moose. *Oikos*, *116*(11), 1805-1818.

Vodanović, M., Hrvoje, B., Mario, Š., & Željko, D. (2005). The frequency and distribution of caries in the mediaeval population of Bijelo Brdo in Croatia (10th–11th century). *Archives of Oral Biology*, *50*(7), 669-680.

Vodanović, M., Peroš, K., Zukanović, A., Knežević, M., Novak, M., Šlaus, M., & Brkić, H. (2012). Periodontal diseases at the transition from the late antique to the early mediaeval period in Croatia. *Archives of Oral Biology*, *57*(10), 1362-1376.

Wadsworth, G. R. (1992). Physiological, pathological, and dietary influences on the hemoglobin level. In: Stuart-Macadam, P., & Kent, S. (Eds.), *Diet, demography and disease: changing perspectives on anemia*, 63-104. New York: Aldine de Gruyter.

Walker, A. (1981). Dietary hypotheses and human evolution. *Philosophical Transactions of the Royal Society of London B: Biological Sciences*, *292*(1057), 57-64.

Walker, M. J., Zapata, J., Lombardi, A. V., & Trinkaus, E. (2011). New evidence of dental pathology in 40,000-year-old Neandertals. *Journal of Dental Research*, *90*(4), 428-432.

Walker, P. L. (1986). Sex differences in the diet and dental health of prehistoric and modern hunter–gatherers. *Proceedings of the VI European Meeting of the Paleopathology Association*, 249–260. Madrid: Universidad Complutense de Madrid.

Wallace, J. A. (1974). Approximal grooving of teeth. *American Journal of Physical Anthropology*, *40*(3), 385-390.

Wallace, J. A. (1975). Dietary adaptations of *Australopithecus* and early *Homo*. In: Tuttle, R., (Ed), P *Paleoanthropology, morphology and paleoecology*, 203-223. The Hague: Mouton.

Wang, X., Zhao, Y., Yang, Y., & Qin, M. (2015). Novel ENAM and LAMB3 mutations in Chinese families with hypoplastic amelogenesis imperfecta. *PloS one*, *10*(3), e0116514.

Ward, C. V., Leakey, M. G., & Walker, A. (2001). Morphology of *Australopithecus anamensis* from Kanapoi and Allia Bay, Kenya. *Journal of Human Evolution*, *41*(4), 255-368.

Washburn, S. L. (1959). Speculations on the interrelations of the history of tools and biological evolution. *Human Biology*, *31*(1), 21-31.

Watt, M. E., Lunt, D. A., & Gilmour, W. H. (1997). Caries prevalence in the permanent dentition of a mediaeval population from the south-west of Scotland. *Archives of Oral Biology*, *42*(9), 601-620.

Weerheijm, K. L. (2003). Molar incisor hypomineralisation (MIH). *European Journal of Paediatric Dentistry*, *4*, 115-120.

Wennberg, A., Mjör, I. A., & Hensten-Pettersen, A. (1983). Biological evaluation of dental restorative materials—a comparison of different test methods. *Journal of Biomedical Materials Research*, *17*(1), 23-36.

White, T. D. (1978). Early hominid enamel hypoplasia. *American Journal of Physical Anthropology*, *49*(1), 79-83.

Whiten, A., Byrne, R. W., Barton, R. A., Waterman, P. G., Henzi, S. P., Hawkes, K., ... & Dunbar, R. I. M. (1991). Dietary and foraging strategies of baboons. *Philosophical Transactions of the Royal Society of London B: Biological Sciences*, 334(1270), 187-197.

Whittaker, D. K., & Richards, D. (1978). Scanning electron microscopy of the neonatal line in human enamel. *Archives of Oral Biology*, *23*(1), 45-50.

Whittaker, D. K., Molleson, T., & Nuttall, T. (1998). Calculus deposits and bone loss on the teeth of Romano-British and eighteenth-century Londoners. *Archives of Oral Biology*, *43*(12), 941-948.

Williams, D. R., & Woodhead, C. M. (1986). Attrition. a contemporary dental viewpoint. In: Cruwys. E., & Foley, R. A., (Eds.), *Tooth and Anthropology*, 109-121. Oxford: British Archaeological Reports.

Williams, S. A., García-Martínez, D., Bastir, M., Meyer, M. R., Nalla, S., Hawks, J., ... & Berger, L. R. (2017). The vertebrae and ribs of *Homo naledi*. *Journal of Human Evolution*, *104*, 136-154.

Williams, S. A., Ostrofsky, K. R., Frater, N., Churchill, S. E., Schmid, P., & Berger, L. R. (2013). The vertebral column of *Australopithecus sediba*. *Science*, *340*(6129), 1232996.

Witkop Jr, C. J. (1976). Heritable defects of enamel. In: Prescott, G., & Stewart, R., (Eds), *Oral facial genetics*, 151-226. St Louis: CV Mosby Company.

Witkop, C. J. (1988). Amelogenesis imperfecta, dentinogenesis imperfecta and dentin dysplasia revisited: problems in classification. *Journal of Oral Pathology & Medicine*, *17*(9-10), 547-553.

Witthüser, K. T., & Holland, M. (2008). Hydrogeology of the cradle of humankind world heritage site, South Africa. In *the 12th international conference of international association for computer methods and advances in geomechanics*. Goa, India.

Wolpoff, M. H. (1973). Posterior tooth size, body size, and diet in South African gracile australopithecines. *American Journal of Physical Anthropology*, *39*(3), 375-393.

Wolpoff, M. H. (1975). Some aspects of human mandibular evolution. Determinants of Mandibular Form and Growth. *Craniofacial Growth Series Monograph*, *4*, 1-64.

Wood, B. A. (1976). The nature and basis of sexual dimorphism in the primate skeleton. *Journal of Zoology*, *180*(1), 15-34.

Wood, B. A., & Abbott, S. A. (1983). Analysis of the dental morphology of Plio-pleistocene hominids. I. Mandibular molars: crown area measurements and morphological traits. *Journal of Anatomy*, 136(1), 197.

Wood, B. A., Li, Y., & Willoughby, C. (1991). Intraspecific variation and sexual dimorphism in cranial and dental variables among higher primates and their bearing on the hominid fossil record. *Journal of Anatomy*, *174*, 185.

Wood, B., & Richmond, B. G. (2000). Human evolution: taxonomy and paleobiology. *Journal of Anatomy*, *197*(1), 19-60.

Wood, B., & Schroer, K. (2012). Reconstructing the diet of an extinct hominin taxon: the role of extant primate models. *International Journal of Primatology*, *33*(3), 716-742.

Wood, B., & Strait, D. (2004). Patterns of resource use in early *Homo* and *Paranthropus*. *Journal of Human Evolution*, *46*(2), 119-162.

Woolgar, C. M., Serjeantson, D., & Waldron, T. (2006). *Food in medieval England: diet and nutrition*. Oxford: Oxford University Press.

Wrangham, R. W., & Conklin-Brittain, N. L. (2003). The biological significance of cooking in human evolution. *Comparative Biochemistry and Physiology, Part A*, *136*, 35-46.

Wrangham, R., Cheney, D., Seyfarth, R., & Sarmiento, E. (2009). Shallow-water habitats as sources of fallback foods for hominins. *American Journal of Physical Anthropology*, *140*(4), 630-642.

Wright, B. W., Ulibarri, L., O'Brien, J., Sadler, B., Prodhan, R., Covert, H. H., & Nadler, T. (2008). It's tough out there: variation in the toughness of ingested leaves and feeding behavior among four Colobinae in Vietnam. *International Journal of Primatology*, *29*(6), 1455-1466.

Wright, J. T. (1985). Analysis of a kindred with amelogenesis imperfecta. *Journal of Oral Pathology & Medicine*, *14*(5), 366-374.

Wright, J. T., Fine, J. D., & Johnson, L. (1993). Hereditary epidermolysis bullosa: oral manifestations and dental management. *Pediatric Dentistry*, *15*, 242-242.

Xing, S., Guatelli-Steinberg, D., O'Hara, M., Li, J., Wei, P., Liu, W., & Wu, X. (2015). Micro-CT imaging and analysis of enamel defects on the early late pleistocene xujiayao juvenile. *International Journal of Osteoarchaeology*, 26(6), 935–946.

Xu, H. H. K., Smith, D. T., Jahanmir, S., Romberg, E., Kelly, J. R., Thompson, V. P., & Rekow, E. D. (1998). Indentation damage and mechanical properties of human enamel and dentin. *Journal of Dental Research*, *77*(3), 472-480.

Yamamoto, M. (1988). Enamel hypoplasia of the permanent teeth in Japanese from the Jomon to the modern periods. *Journal of the Anthropological Society of Nippon*, *96*(4), 417-433.

Zanolli, C., Dean, M. C., Assefa, Y., Bayle, P., Braga, J., Condemi, S., ... & Macchiarelli, R. (2016). Structural organization and tooth development in a *Homo erectus* juvenile mandible from the Early Pleistocene site of Garba IV at Melka Kunture, Ethiopian highlands. *American Journal of Physical Anthropology,* 162(3), 533–549.

Zero, D. T. (1996). Etiology of dental erosion–extrinsic factors. *European Journal of Oral Sciences*, *104*(2), 162-177.

Zihlman, A. L., Bolter, D. R., & Boesch, C. (2007). Skeletal and dental growth and development in chimpanzees of the Taï National Park, Côte D'Ivoire. *Journal of Zoology*, *273*(1), 63-73.

Zilberman, U., Smith, P., Piperno, M., & Condemi, S. (2004). Evidence of amelogenesis imperfecta in an early African *Homo erectus*. *Journal of Human Evolution*, *46*(6), 647-653.

Zipfel, B., DeSilva, J. M., Kidd, R. S., Carlson, K. J., Churchill, S. E., & Berger, L. R. (2011). The foot and ankle of *Australopithecus sediba*. *Science*, *333*(6048), 1417-1420.

Ziscovici, C., Lucas, P. W., Constantino, P. J., Bromage, T. G., & Van Casteren, A. (2014). Sea otter dental enamel is highly resistant to chipping due to its microstructure. *Biology Letters*, *10*(10), 20140484.

Appendix A

Appendix A. Permanent teeth studied.

A. africanus	*P. robustus*	*H. naledi*	early *Homo*	*A. sediba*
TM 1527	STW 566	UW 1662	SE 255	MH 1
TM 1531	SK 83a,b	UW 010	SE 1508	MH 2
TM 1532	SK 876	UW 1463	SE 1937	
STS 556	SK 843 + 846a	UW 591	STW 42	
TM 1528	SKX 242	UW 1277	STW 19	
STS 32	SKX 240	UW 1269	STW 53	
STW 498	SK 34	UW 1261	STW 80	
STW 188	SK 12	UW 037	STW 151 (7)	
STW 34	SK 79	UW 516	STW 151 (3)	
STW 407	SK 61	UW 001	STW 151 (1)	
STW 54	SK 856	UW 1006	STW 151	
STW 492	SK 6	UW 999	STW 151 (6)	
STW 56	SK 14246	UW 1004	SK 27	
STW 405	SK 23	UW 706	SK 2635	
STW 317	SK 826a	UW 932	SK 15	
STW 451	SK 25	UW 38	SK 80	
STW 564	SK 841b	UW 1012	SK 45	
STW 57	SK 881	UW 709	SK 43	
STW 109	SK 1587	UW 816	SKX 2356	
STW 505	SK 64	UW 808	STW 75	
STW 140	SK 3913	UW 455		
STW 14	SK 65a	UW 445		
STW 24	SK 67	UW 1015		
STW 502	SK 65	UW 1063		
STW 191	SK 877	UW 789		
STW 277	SK 85	UW 809		
STW 120	SK 93	UW 887		
STW 327	SK 1591	UW 889		
STW 146	SK 16	UW 1076		
STW 5	SK 839	UW 998		
STW 209	SK 14132	UW 1005		
STW 384	SK 17	UW 377/1014		
STW 13	SK 101	UW 1002		
STW 560b	SK 102	UW 1689		
STW 213	SK 37	UW 1135		
STW 233	SK 20	UW 501		
STW 241	SK 830	UW 358		
STW 222	SK 857	UW 1610		
STW 270	SK 1594	UW 182		
STW 230	SK 1595	UW 908		

A. africanus	*P. robustus*	*H. naledi*
STW 232	SK 838a	UW 417
STW 231	SK 24	UW 344
STW 241	SK 35	UW 005
STW 59	SK 831	UW 654
STW 202	SK 31	UW 602
STW 183a	SK 39	UW 293
STW 23	SK 44	UW 1403
STW 287	SK 71	UW 1362
STW 189	SK 2	UW 796
STW 212	SK 69	UW 528
STW 419	SK 70	UW 527
STW 413	SK 822	UW 359
STW 414	SK 825	UW 653
STW 333	SK 833	UW 1510
STW 415	SK 826a	UW 589
STW 332	SK 870	UW 006
STW 430	SK 14000	UW 593
STW 242	SK 838b	UW 583
STW 412	SK 88	UW 708
STW 215	SK 72	UW 505
STW 224	SK 24613	UW 285
STW 128	SK 851	UW 582
STW 447	SK 14030	UW 886
STW 450	SK 89	UW 1131
STW 126	SK 840	UW 1133
STW 183b	SK 855	UW 1132
STW 569b	SK 14133	UW 1126
STW 169	SK 10	UW 145
STW 127	SK 1648	UW 507
STW 133	SK 49	UW 1398
STW 424	SK 1586	UW 1396
STW 429	SK 57	UW 1401
STW 427	SK 62	UW 1402
STW 446	SK 1588	UW 1558
STW 425	SK 47	UW 1684
STW 132	SK 46	UW 1556
STW 420	SK 14003	UW 1560
STW 421	SK 858	UW 1561
STW 120	SK 844	UW 1471
STW 487	SK 55	UW 850
STW 107	SK 55b	UW 1304
STW 138	SK 1589	UW 1142
STW 192	SK 11	UW 594
STW 509	SK 74	UW 867
STW 513	SK 48	UW 525

A. africanus	*P. robustus*	*H. naledi*
STW 487b	SK 1517c	UW 1676
STW 116	SK 852	UW 1522
STW 118	SK 849	UW 418
STW 117	SK 29	UW 284
STW 119	SK 885	UW 905
STW 195	SK 1596	UW 297
STW 206	SK 33	UW 786
STW 33	SK 3	UW 729
STW 204	SK 40	UW 337
STW 518	SK 3975	UW 73
STW 519	SK 880	UW 39
STW 560c	SK 1	UW 333
STW 110	SK 30	UW 1107
STW 493	SK 86	UW 184
STW 111	SK 1593	UW 144
STW 495	SK 7	UW 1588
STW 404	SK 19	UW 686
STW 252k	SK 3977	UW 335
STW 252h	SK 41	UW 339
STW 280	SK 871	UW 506
STW 252i	SK 3976	UW 383
STW 536	SK 14001	UW 334
STW 369	SK 862	UW 347
STW 281	SK 42	UW 931
STW 252j	SK 849	UW 952
STW 252g	SK 73	UW 412
STW 252l	SK 827	UW 1565
STW 532	SK 872	UW 985
STW 246	SK 105	UW 1075
STW 58	SK 5	UW 601
STW 558	SK 9	UW 245
STW 534	SK 24661	UW 298
STW 531	SK 850	UW 20
STW 134	SK 75	UW 814
STW 72	SKX 50079	UW 1688
STW 286	SKX 3354	UW 1548
STW 123	SKX 3601	UW 544b
STW 61	SKX 3355	UW 1305
STW 234	SKX 3356	UW 1400
STW 3	SKX 1016	
STW 1	SKX 311	
STW 106	SK 81	
STW 220	SKX 4039	
STW 130	SKX 2003	

A. africanus	*P. robustus*
STW 148	SKX 1788
STW 71	SKX 5013
STW 422	SKX 4446
STW 60	SKX 3890
STW 112	SKX 6013
STW 7	SKX 313
STW 401	SKX 10649
STW 393	SKX 1313
STW 87	SKX 5004b
STW 566	SKX 5024
STW 364	SKX 10645
STW 574	SKX 50078
STW 410	SKX 1015
STW 306	SKX 19031
STW 551	SKX 271
STW 540	SKX 5007
STW 309b	SKX 1437
STW 365	SKX 6277
STW 319	SKX 312
STW 537	SKX 3559
STW 249	SKX 310
STW 539	SK 341
STW 538	SKX 5014
STW 309	SKX 7781
STW 560e	SKX 3300
STW 218	SKX 27524
STW 243	SKX 1017
STW 240	SKX 26967
STW 143	SKX 35416
STW 32	SKX 32162
STW 131	SKX 37663
STW 560d	SKX 308
STW 295	SKX 5002
STW 248	SKX 19892
STW 312	SKX 7992
STW 149	SKX 5023
STW 141	SKW 30
STW 561	SKW 32
STW 494	SKW 3068
STW 560a	SKW 4769
STW 73	SKW 5
STW 285	SKW 12
STW 290	SKW 3033
STW 284	SKW 33

A. africanus	*P. robustus*
STW 385	SKW 10
STW 386	TM 1517
STW 193	TM 1601e
STW 198	TM 1601d
STW 95	TM 1601b
STW 194	TM 1601c
STW 203	TM 1603
STW 252e	TM 1602
STW 252d	TM 1600
STW 252	TM 1536
STW 283	KB 5383
STW 530	KB 5223
STW 529	KB 5222
STW 322	
STW 321b	
STW 321a	
STW 351	
STW 21	
STW 476	
STW 475	
STW 288	
STW 324	
STW 323	
STW 237	
STW 353	
STW 282	
STW 284	
STW 278	
STW 555	
STW 541	
STW 542	
STW 20	
STW 248	
STW 543	
STW 296	
STW 469	
STW 416	
STW 147	
STW 145	
STW 308	
STW 269	
STS 61	
STS 71	
STS 12	

A. africanus
STS 36
STS 37 + 28
STS 17
STS 44/66
STS 8
STS 56
STS 55
STS 10
STS 59
STS 4
STS 6
STS 35
STS 42
STS 52
STS 18
STS 1
STS 1534
STS 47
STS 43
TM 1520
STS 41
TM 1523
STS 50
TM 1519
STS 23
TM 1512
TM 1511
STW 402
STW 9
STW 480
STW 50
STW 408
STW 45
STW 16
STW 35
MLD 44
MLD 18
MLD 4
MLD 29
MLD 42
MLD 43
MLD 28
MLD 23
MLD 19

MLD 24
MLD 9
MLD 40
MLD 11 and 30
MLD 2

Appendix B

Appendix B. Deciduous teeth studied.

A. africanus	*P. robustus*	*H. naledi*	early *Homo*
STW 59	SK 61	UW 544	SE 255
STW 62	SK 841	UW 655	STW 151
STW 488	SK 64	UW 728	STW 151 (6)
STW 305	SK 839	UW 287	SK 27
STW 104	SK 438	UW 1611	SKX 1756
STW 97	SK 3978	UW 824	
STS 18	SK 1595	UW 595	
STS 40	SK 838a	UW 384	
MLD 5	SK 90	UW 1571	
MLD 2	SK 91d	UW 1687	
	SK 62	UW 823	
	SK 55	UW 544c	
	SK 55b	UW 1331	
	SK 63	UW 1377	
	SK 852	UW 1376	
	SK 2147	UW 1400	
	SKX 16060	UW 1612	
	SKX 163	UW 1685	
	SKX 32832	UW 1686	
	SKX 27151		
	SKX 37321		
	SKX 50081		
	TM 1601f		
	TM1601		
	TM 1604		
	TM 1536		
	KB 5223		
	KB 5503		