Joint conditions in post-Medieval England:

A comparative assessment of modern risk factors and historic lifestyles

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Abstract

Pathological conditions of the joints are commonplace and incurable, affecting millions of living individuals and yet, surprisingly, there is a great deal that is still unknown about them, including the role and interactions of a variety of risk factors which underpin their development. Much can be learned about joint conditions in the past by marrying clinical and palaeopathological research and by examining patterns of prevalence for joint conditions amongst the living and the trends of the past, the risk factors can be better understood. This thesis aims to utilize palaeopathological evidence of specific joint conditions from past populations in an effort to critically evaluate and analyze the potential risk factors as researched in the clinical literature.

This body of research assessed the joint conditions osteoarthritis, ankylosing spondylitis, sacroiliitis and degenerative disc disease in a sample of skeletal remains from sites across England dating to the 18th-19th centuries. Mature individuals, both male and female, (18+ years) were included. This allowed for the determination and insight into how the lifestyles of each site category affect the development of the joint conditions.

A series of palaeopathological assessments were undertaken to generate a novel dataset that provided skeletal proxies for clinically identified risk factors of the joint diseases to determine whether any relationships/associations existed between the risk factors and joint conditions. Osteological assessments were conducted to create demographic profiles using the pertinent variables (age at death and biological sex), pathologies and the risk factors. These risk factors consisted of body mass (via skeletal height/weight estimation) and activity (via non-imaging cross-sectional geography and entheseal changes), which consisted of five variables, four of which were produced using a method of non-imaging cross-sectional geometry, with the fifth being the scoring of pertinent entheses.

The prevalence rates of the joint conditions fell within the upper ranges of similar sites of post-medieval England and followed sex and age trends also seen in clinical research. These trends showed that the rates increased with age, however statistical testing did not display significance. Body mass and activity did not correspond with joint conditions in the archaeological sample in the same fashion reported in the clinical trials, resulting in a discussion that raised questions about the (1) accuracy and efficacy of currently available osteological methods used to create proxies for these variables from skeletal data, (2) the extent

to which clinical and osteological methods of detecting joint condition offer comparable data and (3) the level which would cause changes to joint function cause a joint condition. However, the body mass of the samples used within this thesis may simply have been too low to have caused sufficient impairment/degradation to the joint, explaining the lack of correlation/association found compared to clinical studies. The variables used as proxies for activity levels did display a significance association with the joint conditions when tested individually. The final binomial logistical regressions found that only a small number of these activity variables were significant factors in the prediction of each joint condition, when all the variables were used in the test. Ankylosing spondylitis was not found to be present in any of the samples used and sacroiliitis was present in only a small percentage of samples and so were unable to be further tested.

Further tests on a larger sample size to test the validity of the results found within this thesis, such as the body mass and activity findings, will need to be conducted. This will help to check the validity of the current data as well as to expand it further so new assumptions/conclusions can be made. The joint conditions conformed to the clinical trends concerning age and sex but differed concerning BMI and activity, offering insights into further avenues to explore. For the spondyloarthropathies, a greater sample size would help to accurately study ankylosing spondylitis and sacroiliitis by increasing the level at which inferential analyses can be made. This research, while concluded in its present form, provides a list of future directions to continue to explore the questions and limitations that have arisen throughout.

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First and foremost, I would like to thank my family, which has continued to support me in many ways throughout my life. They encouraged me to follow my passions and continue with my studies, even though I am sure they would have been far happier knowing I was safe and secure in a job and able to provide for myself, rather than ever continuing to be a mature student.

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For my Aunt Peggy, who encouraged me to never stop learning

Ethical Considerations

The author of this thesis acknowledges that the skeletal remains are those of once living human individuals and consideration for their care and handling was thoroughly considered before the research began. Throughout this research, the remains were treated with care and respect, following all personal and professional ethical guidelines of the researcher and the curating institutions (McGowan GS and LaRoche CJ, 1996; APABE, 2017; BABAO, 2019). In no way did this researcher use the skeletal remains for any purpose other than the scientific research stated in the following aims and objectives.

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Key Abbreviations

%AA – Percent absolute asymmetry
%DA – Percent directional asymmetry
A – Absent
AP – Anteroposterior
APM – Anteroposterior diameter at midshaft
APS – Anteroposterior diameter at subtrochanteric
AS – Ankylosing spondylitis
BME – Body mass estimation
BMI – Body mass index
BS – Biological sex
CA – Cortical area
CSG – Cross-sectional geometry
CT- Computed Tomography
DDD – Degenerative disc disease
DJD – Degenerative joint disease
EC – Entheseal change(s)
Fem – Femur/femoral
FHD – femoral head diameter
FM – Femoral midshaft
FMA – Femoral midshaft area
FMJ – femoral midshaft polar second moment of area
FML – Femoral maximum length
FMR – Femoral midshaft robusticity
FMS – Femoral midshaft shape
FS – Femoral subtrochanteric
FSA – Femoral subtrochanteric area
FSJ – Femoral subtrochanteric polar second moment of area
FSR – Femoral subtrochanteric robusticity
FSS – Femoral subtrochanteric shape
HFD – High fat diet
HOA – Hip osteoarthritis
IL – Interleukin
J – polar SMA
KOA – Knee osteoarthritis
MA – Medullary area
ML – Mediolateral
MLM – Mediolateral diameter at subtrochanteric
MLS – Mediolateral diameter at subtrochanteric
MRI – Magnetic resonance imaging
OA – Osteoarthritis
OR – Odds ratio
P – Present
RR – Risk ratio
SI – Sacroiliitis
SMA – Second moment of area
SnSpA – Seronegative spondyloarthropathy
SOA – Spinal osteoarthritis
ST – Stature
TA – total area
Tib – Tibia/tibial
UD – Undeterminable

Chapter 1 Introduction

The research within this thesis will explore the relationships between the skeletal manifestations of joint conditions and a series of risk factors implicated in the development of these diseases within clinical and palaeopathological literature. While the original data generated by this research concerns archaeological human remains, and therefore placed the project within the palaeopathological field of study, a significant contribution is made by clinical research. Clinical studies play a vital role by providing longitudinal data which offers insights into the development of the joint conditions over time, which can in turn help palaeopathologists understand the stages of development on the surface of the bone, as well as providing information as to the effects the conditions could have had on living individuals. The joint conditions that will be covered within this thesis include osteoarthritis, ankylosing spondylitis, sacroiliitis and degenerative disc disease and the joint locations of the lumber vertebrae and sacroiliac joint (lower back), acetabulum (hip), and tibiofemoral/patellofemoral (knee) joints. The risk factors under investigation include both archaeological and clinical: site type (urban v rural), age at death, biological sex, activity, and body mass. The skeletal samples come from English populations dating to the late post-medieval period, circa 1700-1850 CE and only the adult individuals that can have their age at death reasonably and reliably estimated to be over the age of 18 years were used for this research. By studying the different site categories, as well as site locations, it should become clear which, if any, risk factors impacted the prevalence of the joint conditions for each joint.

1.1 Thesis Structure

This thesis begins with an introduction to the aims and objectives, which leads into the literature review, materials, research methods and results, finishing with the discussions on how the results pertain to the aims and objectives. Each of the following sections will explain the layout while offering detailed, but brief, descriptions for the material, thus this section will work as a guide for what can be expected throughout the dissertation.

1.1.1 Chapter 2 Aims and Objectives

The aims and objectives of this research will be explained within this chapter. The aims and objectives are the focal point of the thesis and allow the reader to understand the direction taken with latter assumptions and assessments. The research questions, which will be answered in the discussion chapter, can be found after the description of the aims and objectives.

1.1.2 Chapter 3 Joint Conditions

This chapter will introduce the reader to the pathological conditions that can affect the joints, to include their clinical definitions and aetiologies, their key osteological markers and diagnosing criteria and prevalences in the past and present. The risk factors implicated in the formation of the joint conditions are compared with the palaeopathological variants to offer a balanced viewpoint on each of the conditions. This chapter provides a critical justification of why it is important to study joint conditions in the past to help better understand their effects on the living, as well as our ancestors.

1.1.3 Chapter 4 Sample Selection

This chapter has a dual purpose: (1) to explain the criteria for the selection of viable samples and (2 to offer insights into the histories of the populations to enable the reader to gain an insight into the people's lives. The criteria for selection will go into detail what was included, as well as reasons for possible exclusion from the data pool. The locations of the sites, curating institutions, and number of samples for each population are displayed in a table within this chapter. The histories of the sites are brief, emphasizing the pertinent information for the purpose of this research from the areas as well as focusing on the specific site/feature where the samples were recovered.

1.1.4 Chapter 5 Methodology

The methodologies used will be explained within this chapter, as well as why a method was used and why one may not have been used, in a fashion similar to a literature review. Each

stage of the research process will be discussed, to include what methods will be used at each stage and what data will be created from those methods and why the data is necessary. The chapter has been divided between the physical, laboratory-based palaeopathological assessments and the more analytical, computer-based statistical assessments and tests. Equations used to produce numerical scale values have been listed and the validity for the methods explained.

1.1.5 Chapter 6 Results

The results of all the analyses completed will be found within this chapter. The structure will also follow a logical layout, starting with demographic profiles describing the populations by site category, biological sex, and age at death. The prevalences of the joint conditions follow and are further divided by the categories listed above and followed by the specific variables of the risk factors. Once basic prevalences, counts and ranges of the above-mentioned data is explained, inferential statistical data are presented, starting with bivariate assessments exploring the relationships between the joint conditions and risk factor variables, as well as between the risk factors themselves.

1.1.6 Chapter 7 Discussion

The discussion section will attempt to answer the three research questions listed in Chapter 2. Each of the sections will examine the results of the data as it pertains to each research question, as well as make comparisons with data from similar published research. The first section will relate to prevalence rates according to site, biological sex, and age at death, the second will address the relationships with body mass and activity and the third and final section will discuss the spondyloarthropathies. The implications of the research and potential remedies for any limitations that arose as a result will be described within this section.

1.1.7 Chapter 8 Conclusion

This chapter is relatively straight forward as it will be the conclusion to the body of the thesis and the last text-based chapter. The research will be summarized offering any final insights and thoughts on the implications. As potential issues and remedies were discussed in the discussions, proposed future research, which can attempt to explore the ways to better unravel these limitations to allow for future research to be able to create more accurate and reliable diagnoses. As a note, this research is not planned by the author but are suggested avenues to explore.

This thesis is the culmination of research regarding joint condition and its risk factors, combining palaeopathological and clinical research to reconcile the varying schools of thought to provide a better understanding of the diseases and how they affect individuals of the past. The idea was meant to step away from standard prevalence-based research and to focus on the lives and lifestyles of past English populations. To better understand these diseases, research must be opened to include various disciplines.

Chapter 2 Aims and Objectives

The aim of this research project was to utilize palaeopathological evidence of joint conditions from human skeletal remains from archaeological sites to explore and critically evaluate the potential influence of risk factors implicated in clinical evidence in the development of osteoarthritis (OA), ankylosing spondylitis (AS), sacroiliitis (SI) and degenerative disc disease (DDD) in the past.

2.1 Objectives

I. To critically review current clinical evidence concerning osteoarthritis, ankylosing spondylitis, sacroiliitis and degenerative disc disease to establish the key risk factors implicated in their formation among modern populations

II. To critically review and identify appropriate palaeopathological methods to generate a new, detailed dataset concerning these joint conditions in a sample of human skeletal remains from the post-medieval period

III. To collate, and generate where necessary, data concerning age, sex, and site type (rural or urban) for this skeletal sample. To make comparisons of joint condition between these groups to explore the influence of lifestyle and demographic factors on joint condition prevalence

IV. To identify an appropriate range of skeletal proxies for the key risk factors implicated in the formation of joint conditions by the clinical literature (body mass and activity levels), generate new skeletal evidence for these proxies and explore the influence of these factors on joint condition prevalence

V. To undertake a multivariate assessment which explores the extent to which multiple risk factors acting in concert might better explain the prevalence of joint condition in an archaeological population than individual risk factors acting in isolation

VI. To compare the data generated in this thesis with extant studies of joint condition prevalence in the clinical literature and archaeological studies of the last millennium in general, and the post medieval period specifically, to help explain and understand any patterns observed

VII. To critically reflect on the value of comparing clinical and osteological data to the advancement of the understanding of joint condition in the past and the extent to which we can effectively explore the influence of clinically identified risk factors on joint condition prevalences obtained from human skeletal remains.

2.2 Research Questions

1. To what extent does the prevalence of osteoarthritis, ankylosing spondylitis, sacroiliitis and degenerative disc disease vary with age and sex, or between urban and rural populations?

2. What are the relationships between the skeletal variants of the risk factors implicated in clinical studies of joint condition – increased body mass and activity levels – and osteoarthritis, ankylosing spondylitis, sacroiliitis and degenerative disc disease? To what extent do the clinically identified risk factors explain prevalence of joint condition in skeletal populations?

3. Can seronegative spondyloarthropathies be recorded in skeletal remains in a manner that enables population-level study of their prevalence and assessment of the role of clinically identified risk factors? What problems are implicated in this assessment, and how might they be resolved?

4. How might archaeological and clinical approaches to the study of osteoarthritis, ankylosing spondylitis, sacroiliitis and degenerative disc disease be integrated to the benefit of our understanding of joint conditions and the present in human skeletal remains, and therefore our understanding of these diseases in the past in general?

This thesis was an attempt to bring clinical knowledge to the palaeopathological field to help better understand how peoples of the past would have been affected by joint condition. By using the clinical risk factors in a palaeopathological setting, the prevalence rates will gain further context as the individuality of the development of the conditions is brought to the fore. The joint conditions are often discussed in the population setting, as this thesis will do, but when the lifestyles are discussed with a focus on the causes of the conditions, effects on the skeleton and the effects on the individual and population. This should, in theory, help to better understand these people and allow their stories to continue to live on through this research. The following Chapters will demonstrate the value of studying joint condition and the need for researchers to understand both the palaeopathological and clinical literature surrounding the subject.

Chapter 3 Joint Conditions

Joint condition is an overarching term used to classify any disease or injury that affects the joint space and corresponding skeletal material (Roberts and Manchester, 2010). The key osteological characteristics of joint conditions are the formation of bony protrusions and lesions on the joint (see Figure 3.1) (Rogers and Waldron, 1995; Roberts and Manchester, 2010; Burt *et al.,* 2013). There are numerous conditions that fall under this category that include, but are not limited to, osteoarthritis, rheumatoid arthritis, septic arthritis, and spondyloarthropathies (Burt *et al.,* 2013). Each condition has its own unique aetiology, definition, and method for diagnosis (Roberts and Manchester, 2010), but the conditions share similar markers. The conditions can also be referred to as interrelated, as one may cause or influence another's development. This research will focus on osteoarthritis of the lumbar vertebrae, hip, and knee as well as degenerative disc disease, ankylosing spondylitis and sacroiliitis. These joint conditions were selected as they all affect the load bearing joints of the lower appendicular skeleton, with the addition of the related vertebral elements and can often be found together.

Figure 3.1 Femoral head with gross eburnation, macro and microporosity and possible subchondral cyst. Photo is courtesy of Andy Brown and the Image Speaks project.

3.1 Bio-Mechanical Function of Joints

The joints of the human body are locations where one or more independent skeletal elements meet and connected via soft tissue cartilage. There are three types of joints within the human body: synarthrosis (immovable), amphiarthrosis (semi-moveable) and diarthrodial (moveable). These joints allow for the stability and motion of the musculoskeletal system. For the purpose of this research, the lumbar vertebral facets, hip, knee (diarthrodial) and joints between the bodies of the lumbar and sacral vertebrae (amphiarthrosis) were examined.

3.1.1 Diarthrodial Joints

Diarthroidial joints, or synovial joints, allow for specific types of movement (see Figure 3.2). In mammals, the glenohumoral (shoulder) and acetabulofemoral (hip) joints are ball-and-socket joints that facilitate locomotion by enabling a wide range of movement about a single center point (Tözeren, 2000). The ball-and-socket joints are multiaxial, which allow for the skeletal features to move in multiple directions and increases the function and type of activity that can be completed (Kapandji, 1970; Tözeren, 2000). In contrast to the hip joint, in humans the locomotion feature of the shoulder has been altered over the course of the evolution of bipedalism and now has a greater range of motion, but less stability, as an open ball-and-socket joint (Kapandji, 1970; White and Folkens, 2005). The acetabulofemoral joint is the most proximal joint of the lower limb and has three axes of movement, the transverse, anteroposterior and the vertical axes, which then offers three degrees of freedom (Kapandji, 1970). The articular facets of the lumbar vertebrae differ from the diarthrodial joints of the hip and knee, and instead allow for the vertebral movements described in the following section on amphiarthrodial joints, while keeping the vertebrae properly aligned (Tözeren, 2000).

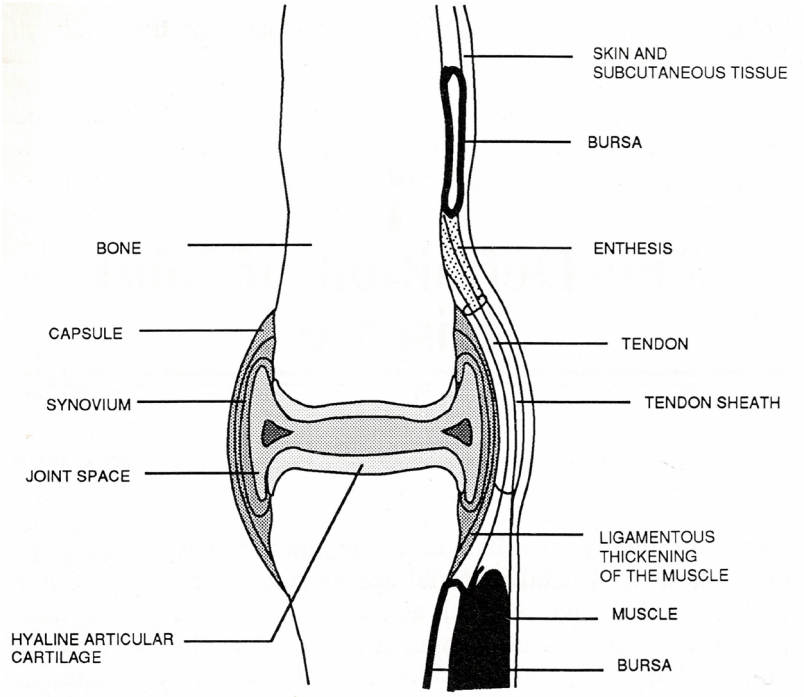

Figure 3.2 Illustration showing a diarthrodial joint and the component parts (Rogers and Waldron, 1995).

The tibiofemoral and patellofemoral joints, which combine to create the knee, are uniaxial hinge joints that act as a lever for flexion and extension for an approximate 180° range of motion (Tözeren, 2000, Roberts and Manchester, 2010). The ranges of motion create a unique set of abilities and movement types for each joint. Different activity types will require the joint and musculo-skeletal system to respond in a typical behavioral pattern. For instance, known patterns of movements would include adduction, abduction, flexion, extension, pronation, supination, and rotation (Adrian and Cooper, 1995; Tözeren, 2000). While each joint has its own limitations, the body working in harmony allows for the varied and complex motions that humans experience daily. A simple example of this is sitting to type a document, the body of the author is using the hips and spine to sit upright, the legs to stabilize the position of the chair and assist in angling the torso, the shoulder to move the arms forward and back, the elbow to move the hands to different key levels and the wrists to move the hands so that each finger can hit a key. As complex organisms, these motions are done without thought as to why and how, the motion is accomplished as a means to complete a task (Adrian and Cooper, 1995; Tözeren, 2000; Winter, 2009).

Within each joint is a non-renewal stabilizer called a synovial capsule, or joint capsule, and while the synovial capsule is non-renewable, the fluid inside able to regenerate via the introduction of omega-3 fatty acids (Lorenz and Richter, 2006; Henrotin *et al.,* 2012). The

synovial joint is not innervated and has no vascular supply and rely on nutrients from the enveloping cartilage. Surrounding the joint capsule are the ligaments and tendons that act as stabilizers to maintain the structural integrity of the joint and, barring specific pathological conditions, these can be repaired, but the fibrous synovial capsule cannot be repaired without the external assistance of a physician, and in many cases, surgical replacement as an option (Abrahams *et al*., 2013).

3.1.2 Amphiarthrodial Joints

The amphiarthrodial joints are joints that stabilize two or more skeletal elements but allow a minimal degree of mobility. The spinal column is the most common location to find such joints as the vertebral column acts as a central stabilizing column for the torso as well as the rest of the human body. The vertebral column has its own distinctive qualities of having both stability and plasticity (Kapandji, 1974). The vertebral column acts as a stabilizing structure, like an architectural column, as well as a protective structure for the neuroaxis, demonstrating the need to be flexible and strong. Each section of the vertebral column has a role, with the upper three sections of the vertebral column (cervical, thoracic, and lumbar) allowing for flexion, extension, and rotation of their individual regions (Tözeren, 2000). The cervical vertebral allow for moving the neck and head, the thoracic vertebrae the torso, the lumbar acting as a general support structure for the entire system, and the sacrum acting as both general support and connection with the lower appendicular skeleton. These elements together form an S-like structure.

The intervertebral disc is a fibrocartilaginous structure made up of a nucleus pulposus (center) and the annulus fibrosis (shell) and cartilaginous endplate (connector) (see Figure 3.3) (Karajan, 2012; Newell *et al.,* 2017). The nucleus pulposus takes up roughly 50% of the total volume of the disc (Karajan, 2012). The annulus fibrosus is the outer shell of the vertebral disc and is made up of 15 – 25 concentric layers called lamellae (Newell *et al.,* 2017). These lamellae, a fibrous structure that alternates alignment at each layer, are approximately 0.05-0.5 mm in thickness and the thickness decreases from the nucleus pulposus to the outer most layer (Karajan, 2012; Newell *et al.,* 2017). The superior and inferior portion of the disc, called the cartilaginous endplate, forms a foundation and roof to support and connect the disc to the

vertebrae. The intervertebral disc is primary formed of an extracellular matrix and made up of collagen and water (Karajan, 2012).

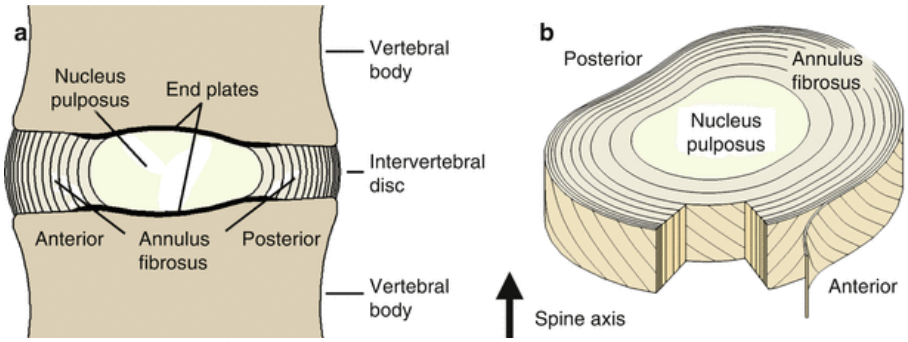

Figure 3.3 The anatomy of the vertebral joint space between the vertebral bodies (Cortes and Elliot, 2014).

The lumbar vertebra and sacrum are the lower most elements of the vertebral column, both sections each consisting of five vertebrae. The lumbar vertebrae are the largest of the vertebra and allow only the slightest flexion, extension, and rotation. The sacral vertebral are unique in that they fuse to form a single structure, the sacrum, connected to the pelvic girdle and form a distinct shape depending on the biological sex of the individual. The lumbar and sacral vertebrae absorb and distribute the stress of body mass down into the lower appendicular skeleton (see Figure 3.4).

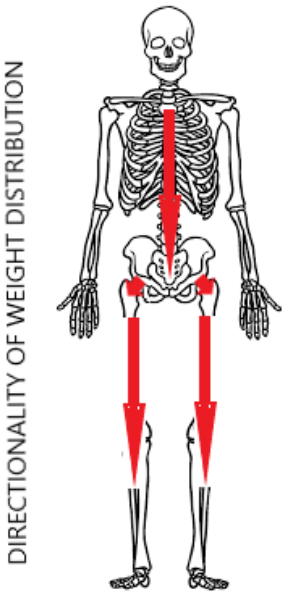

Figure 3.4 Illustration of the direction of weight distribution through the human body. Note that the vertebral column takes all of the downward force until distributed along the dual branches of the lower appendicular skeleton.

3.2 Appendicular Joint Conditions

Osteoarthritis (OA), a degenerative joint condition, is a general term for a condition that affects diarthrodial joints, with different aetiologies, factors, and rates. Osteoarthritis is characterized by a decrease in the joint space and breakdown of corresponding skeletal structures (see Figure 3.5) (Rogers and Waldron, 1995; Roberts and Manchester, 2010). With OA, the joint space typically becomes inflamed, causing fibrillation of the articular cartilage as the joint tissues split, soften and fragment (Williams and Spector, 2006). OA is a common pathological condition, with approximately 15% of the modern population manifesting symptoms (Johnson and Hunter, 2014) and are found at increased rates over the age of 55 years (Rogers and Waldron, 1995; Roberts and Manchester, 2010) in Western Europe and North America. In individuals over the age of 60, an estimated 10% of males and 18% of females are affected by the condition, causing a socioeconomic burden costing 1-2.5% of gross domestic products (Glyn-Jones *et al.,* 2015). However, despite the high prevalence of OA observed in modern groups, much is still unknown about the condition.

Figure 3.5 Osteoarthritis hip joint with the femoral head inserted into the corresponding acetabulum. Note the development of lipping along the rim of the acetabulum and the base of the femoral head. Photo is courtesy of Andy Brown and the Image Speaks project.

The articular cartilage is not innervated and has no vascular supply, relying on nutrients for the chondrocytes that have been diffused through the synovial fluid, and the cartilage has limited repair abilities compared to the synovial capsule (Lorenz and Richter, 2006). The

cellular mechanism believed to be the cause of cartilage degradation is "an excessive production of matrix metalloproteinases and aggrecanases by the chondrocytes and the hypertrophic differentiation of chondrocytes leading to the calcification of the cartilage matrix" (Henrotin *et al.,* 2012: S847). This would cause the joint cartilage to, over time and after much alteration, be unable to withstand the normal mechanical stress and load for that joint. The changes would cause the matrix of the joint to become stiff and affect the brittleness as the turnover for type II collagen and cartilage proteoglycan becomes reduced with age (Sacitharan and Vincent, 2016).

There is no consensus as to the proper naming of the condition within palaeopathological context – some researchers use osteoarthrosis in place of osteoarthritis. The suffixes -itis and -osis have different meanings, the former being associated with inflammation and the latter with non-inflammatory degeneration (Burt *et al.,* 2013). Inflammation is a major characteristic of clinical osteoarthritis affecting the synovial capsule and surrounding soft tissue; however, inflammation does not manifest in osteological material (Waldron, 2009); therefore, in cases of osteological examination, the name can be misleading as it denotes an inflammatory response when one is not present. Some researchers argue for the use of the term osteoarthrosis because of the apparent non-inflammatory nature of the osteological condition, but it is not possible to definitively determine inflammatory response on the osteological material (Burt *et al.*, 2013). For the purposes of simplification and consistency for later comparisons with osteological and clinical research, the term osteoarthritis, as it is still the common palaeopathological term, will be used throughout this body of research.

There are two main classifications of osteoarthritis: primary and secondary (Fergusson, 1987). These terms denote the apparent cause of the condition – whether the condition has developed within the joint or was affected by an external condition. The differences are not always direct or straightforward, as the body is a biomechanical structure with inter-related systems. Primary osteoarthritis is a condition that affects the joint without influence from a pathology located elsewhere within the body (Buckwalter and Martin, 2006). Secondary osteoarthritis is generally the result of trauma or ailment at a different location to the joint that will still affect the joint (Fergusson, 1987; Buckwalter and Martin, 2006; Roberts and Manchester, 2010). For instance, a broken or infected leg may cause an individual to have an altered gait, which then causes increased stress to the joint. The injury may be to the femur, tibia, or fibula, but the altered gait may also affect the acetabulofemoral, patellofemoral or

tibiofemoral joint. The change to the joint structure, as a result of injury, will not happen in the short term and would result from chronic or sustained injuries over time.

Osteoarthritis has different prevalence rates for the different joints and can also vary by biological sex, age, body mass or activity level (Roberts and Manchester, 2010; Burt *et al.,* 2013). As such, there are range of risk factors that are implicated in the formation of osteoarthritis. Females have an increased likelihood of developing the condition than men, with earlier ages of onset due to the effects of menopause (Oliveria *et al.,* 1999). The risk factors do not uniformly affect each joint, with some causing increased stress to the joints to a lesser/greater degree. For example, obesity and the effect of carrying increased mass on the skeletal frame will affect the knee joint more than the shoulder (Oliveria *et al.,* 1999, Ding *et al.,* 2005), however, obesity may also affect the joint of the wrist, although researchers do not yet understand the relationship (Felson and Chaisson, 1997; Grotle *et al.,* 2008). Due to the increase of obese individuals in modern times, the prevalence of tibiofemoral osteoarthritis has risen, and osteoarthritis at this location is thought to be a modern condition (Rogers and Dieppe, 1994).

Many theories abound about the causes and factors influencing the progression of OA. The condition was once thought to be an unavoidable consequence of aging (Burt *et al.,* 2013), but research has shown that onset and progression are more complex, with the lifestyle of the individuals being an important consideration (Arden and Cooper, 2006). The risk factors for osteoarthritis include, but are not limited to, age, biological sex, activity levels, genetics and body mass. The risk factors, explained in further detail below, can increase an individual's likelihood of developing the condition to a greater extent.

3.2.1 Clinical Approaches for Identification and Classification of OA

The approaches for diagnosis of OA in clinical contexts are different to those of osteologists, with two types of diagnoses depending on the material available: symptomatic and radiographic. Clinicians do not have direct access to the bone, without surgical intervention, and a symptomatic diagnosis instead focuses on severity of known osteoarthritic indicators, or symptoms, from external examinations and survey questions. Symptoms, such as pain, swelling and stiffness, can be assessed through direct access to a living patient through testimonial or clinical examination (Roemer *et al.,* 2006; Hooper and Moskowitz, 2007).

Symptomatic diagnoses deal with subjective testing regimes, such as the pain scales and stiffness, that cannot offer reproducible results and must be used only within the bounds of context and patient history (Roemer *et al.,* 2006). Symptomatic diagnoses tend to have a lower prevalence than radiographic due to the subjective nature of examinations and the reaction of an individual to a symptom, whereas radiographic diagnoses rely on objective tests that are more sensitive (Johnson and Hunter, 2014).

As the name implies, radiographic diagnoses rely on imaging techniques such as plain film radiography and MRIs. Imaging equipment is the only way for a clinician to view the skeletal material and soft tissue surrounding a joint of a living individual without invasive surgery. Imaging methods offer objective testing that is reproducible and verifiable (Roemer *et al.* 2006). Magnetic Resonance Imaging (MRI) will show the soft tissue and joint capsule as well as the bone and plain film radiography will show the bone without soft tissue (Braun and Gold, 2012; Burt *et al*., 2013). An MRI allows for clinicians to assess the body for subcutaneous lesions and inflammation or damage that could otherwise go unnoticed from an external examination of a living patient, while traditional radiographs allow for the assessment of joint space narrowing, cyst formations and subchondral sclerosis (Fergusson, 1987). A computed topography scan (CT) has increased sensitivity, which can help to illustrate changes occurring within the joint space to a higher level than radiography. CT scans also have less intra-observer variability, which can make them more reliable (Rubin, 1996). However, a CT image is not necessary if plain film radiography was effectively able to be used for diagnosis.

The Kellgren and Lawrence system, colloquially known as the K-L scale, can be used to help clinicians classify the severity and progression of the condition via radiographs (Arden and Cooper, 2006). The K-L scale consists of categories with established gradients for the development of osteological markers. The categories are strict, not allowing for flexibility and causing an objective method to require subjective analysis (see Figure 3.6) (Felson *et al.,* 2013). This method used a scale of grading that follows (Kellgren and Lawrence, 1957):

Grade 0 – None - No radiographic features of OA present.

Grade 1 – Doubtful - Doubtful joint space narrowing and osteophytic lipping.

Grade 2 – Minimal - Definite osteophytes and possible joint space narrowing on anteroposterior load-bearing radiograph.

Grade 3 – Moderate - Multiple osteophytes, definite joint space narrowing, sclerosis, possible bony deformity.

Grade 4 – Severe - large osteophytes, marked joint space narrowing, severe sclerosis and definite bony deformity.

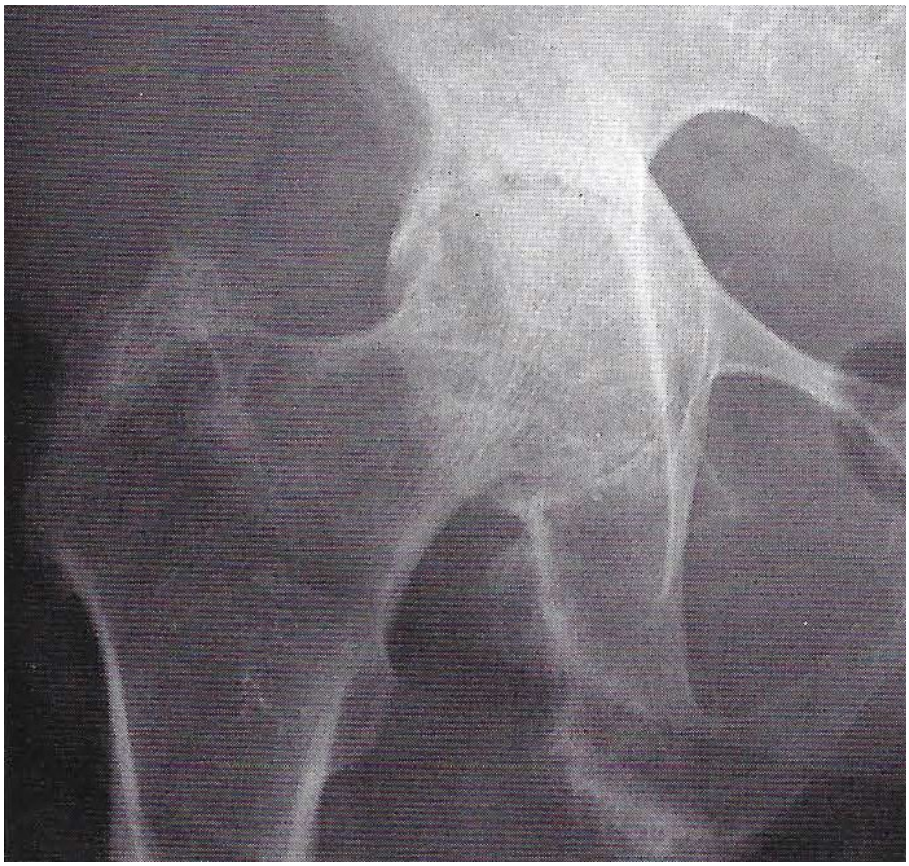

Figure 3.6 Radiograph of a hip joint with a narrowing of the joint space (Burgener 2006: 131).

However, in recent years, this method has come under criticism due to the limited scope at which it can be used (Felson *et al*., 2013). The strict, combined scoring does not allow for divided scoring, where the differing traits may fall into a range of categories, rather than be able to be lumped into just one. The need to update and refine the method for more accurate assessments to break apart the grades into the component features has been put forth with the suggestion of more flexible criteria (Arden and Cooper, 2006). Rather than group all the features into one grade, the markers, such as osteophytic formations or joint space narrowing, would be graded individually, allowing for more flexible and accurate diagnoses.

The Kellgren-Lawrence scale is the benchmark scale for assessing osteoarthritis via imaging, but it is not the only method. In 2011, Roemer *et al.* published a method for the scoring hip osteoarthritis using an MRI, but the hip joint creates inherent problems when viewing using imaging due to the shape and structural format. The method, known as the Hip Osteoarthritis MRI Scoring System, or HOAMS, scores the markers individually, but using criteria similar to the Kellgren-Lawrence scale.

Imaging techniques are not equal, and each offers its own unique benefits and drawbacks. Traditional radiographs are excellent for viewing the skeletal material but produce levels of harmful radiation and does not offer clear views of the surrounding soft tissues. Radiographs are cheaper and faster than CT or MRI scans and do not need a specialized room for completion, but only show a limited 2D image (Rubin, 1996). CT scanning allows for cross-sectional images to be viewed, which can help to differentiate loose osseous pieces from the skeletal frame, however, like traditional radiography, produces levels of harmful radiation and has difficulty contrasting the soft tissue (Roemer *et al.,* 2006). An MRI can take cross-sectional images with layering that allows for the assessment of the soft tissue. CT and MRI scans are significantly more expensive and time consuming, but the results can be viewed much more swiftly allowing for subsequent tests and imaging to be devised throughout.

The differences between symptomatic and radiographic methods of diagnosis creates a disjunct between the methods that requires careful navigation in order to use clinical data for comparison with osteological data. An individual may have radiographic osteoarthritis but not develop any outward indicators for a symptomatic diagnosis, while another individual may not have a radiographic diagnosis, but have developed the outward indicators for a symptomatic diagnosis (Nevitt, 2006; Parsons *et al.,* 2018). This is a conundrum that clinicians face due to the fickle nature of the human body and lack of understanding as to the cause of the condition.

3.2.2 Osteological Approaches to Identification and Classification of OA

Diagnosing OA osteologically initially appears to be a straightforward process, as the skeletal elements are visible to the researchers for direct examination. OA has many of the same osteological markers of other conditions, such as porosity and osteophytes, however, there is one marker that is considered to be pathognomic of the condition: eburnation (Ortner, 2003; Waldron, 2009; Burt *et al.,* 2013). As the markers for osteoarthritis are common

pathological markers, also observed in other disease processes, a differential diagnosis ruling out other pathological conditions is warranted. OA that has progressed over time can cause severe degradation of the joint and resemble septic arthritis. Anaemic conditions can cause porous regions across the skeletal frame, as the body shifts from metabolic to catabolic reaction, but porosity is also one of the markers for osteoarthritis (Roberts and Manchester, 2005). Therefore, to determine potential differential diagnoses for OA, it is important for the researcher to not focus on the joint alone, but to also examine the surrounding skeletal elements to garner as much information about the condition, as well as the individual.

Juliet Rogers and Tony Waldron (1995) published a set of diagnostic criteria for OA, which focuses on the presence of eburnation to conclude a probable diagnosis. In the absence of eburnation, three of the markers of osteoarthritis must then be present for a probable diagnosis to be made (see Figure 3.7) (Rogers and Waldron, 1995). This method becomes a trade-off between ease and speed of diagnoses and the more comprehensive knowledge and potential understanding of the condition.

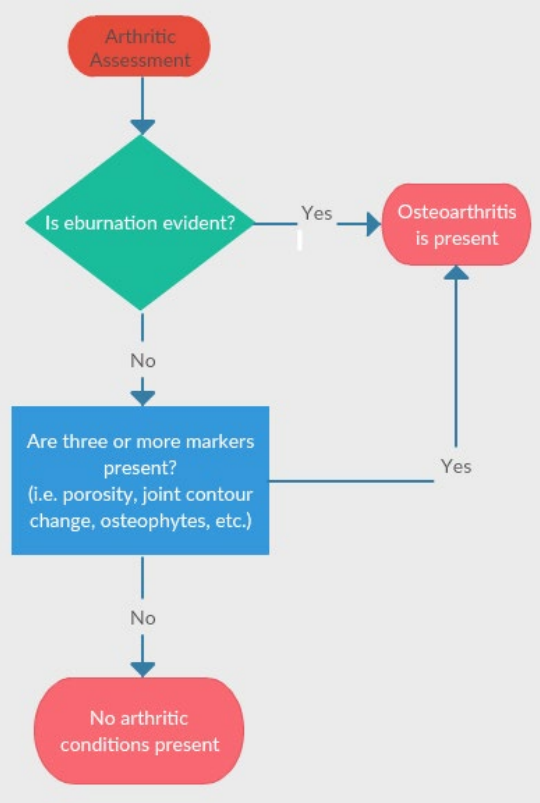

Figure 3.7 A flowchart of the key decisions for diagnosing osteoarthritis using Rogers and Waldron (1995).

Therefore, to diagnose OA, the researcher must accomplish multiple steps. The first step is to examine the joint and assess which markers are present. The second step is to assess the remaining elements of the skeleton in an attempt to provide a differential diagnosis. If the condition may be present, the researcher must then, by examining the evidence, determine the level of likelihood that OA is present by either saying the condition is probable or possible. A probable diagnosis, as the name suggests, is when the body of evidence shows that OA is most likely present and the main cause of the skeletal markers. A possible diagnosis indicates that evidence for the presence of the condition, but there is not enough evidence, or potentially differential diagnoses are present, to accurately infer a probable diagnosis (Ventades *et al.,* 2018). A possible diagnosis may be the result of damaged or incomplete remains, where there is evidence of OA being present, but there is information missing that would be crucial to the diagnosing process. If the evidence suggests the absence of OA, then no diagnosis is made.

3.3 Vertebral Joint Conditions

Vertebral joint conditions describe any number of conditions which can affect the different joints of the vertebrae and the axial skeleton. This research will focus on three such spinal conditions: ankylosing spondylitis, sacroiliitis, and degenerative disc disease. AS and DDD are primary pathological conditions with similar developmental features but different aetiologies, while sacroiliitis is a secondary condition (Hughes, 1992; Waldron, 2009; Kadwani and Mahmud, 2014).

Seronegative spondyloarthropathies (SnSpA) is a general term for inflammatory joint conditions that primarily affect the axial skeleton. Unlike some other arthritic conditions, these cannot be tested via blood for rheumatoid factor and anti-cyclic citrullinated peptide antibodies (Burt *et al.,* 2013; Kadwani and Mahmud, 2014). These related conditions have common clinical and radiological features and are associated with the HLA-B27 antigen (Hughes, 1992; Waldron, 2009; Burt *et al*., 2013; Kadwani and Mahmud, 2014). In cases of diagnosable ankylosing spondylitis between 60-70% of individuals have the HLA-B27 antigen (Weisman, 2011), with an estimated 90% of ankylosing spondylitis cases in the UK, an estimated 4-13% of the population has the HLA-B27 antigen and that only 1.3% of Europeans with the antigen will have ankylosing spondylitis (Sheehan, 2004; Stolwijk *et al.*, 2012). Characterized by spinal ankylosis and enthesitis (Waldron 2009), the conditions include, but are not limited to,

reactive arthritis, ankylosing spondylitis, sacroiliitis, and psoriatic arthritis (Waldron, 2009; Kadwani and Mahmud, 2014).

While SnSpA primarily affect the axial skeleton, they can also affect other areas of the body through soft tissue pathologies (Samsel *et al.,* 2014), including multiple enthesopathies of insertion points. Ankylosing spondylitis and sacroiliitis are spondyloarthropathies that can affect the joints of the appendicular skeleton, such as the hip and knee (Weisman, 2011). The hip is one such location where osteoarthritis can often be found when AS is also present, however, it may not be clear whether this is a concurrent or secondary development (Rogers and Waldron, 1995; Weisman, 2011). Locations external to the joints can develop enthesopathies or scleroses with additional bone formations and locations for these include the deltoid tuberosity, distal phalanx, sternal rib ends and the pubic symphysis.

Ankylosing spondylitis (AS) is the most common seronegative spondyloarthropathy (Stolwijk *et al.,* 2012) and is a chronic rheumatic condition (Weisman, 2011). AS is a condition that primarily affects the axial skeleton, which can also impact non-axial joints including the shoulder, elbow, hip, knee, and feet causing osteoarthritic symptoms. The key feature of AS is bilateral fusion, or ankylosis, of the vertebral bodies, in a sequential manner without skipping. This fusion is a result of ossified ligaments, or syndesmophytes, and typically commences within the sacroiliac joint, where the sacrum meets the auricular surface of the pelvis and progresses through the lower lumber spine and upwards (Rogers and Waldron, 1995). The condition will then begin to ossify the annulus fibrous of the inter-vertebral discs, eventually forming an attachment to the anterior surface of the vertebral body (Burt *et al.,* 2013, Kadwani and Mahmud, 2014). In the thoracic vertebrae, the fusion may affect the articulating ribs, causing fusion of the costovertebral joint and ossification of the costovertebral ligament. The ankylosis will resemble a shaft of bamboo, which has led to the colloquial term: bamboo spine. The condition is believed to be more common in males than in females, with the typical age of onset occurring between the ages of 17 and 30 (Khan, 2002).

The condition can be heredity (Hart, 1980; Khan, 2002). In 82% of individuals with ankylosing spondylitis, a fusion of the sacroiliac joint will occur within six years. The condition was first described in text by an Irish physician, Bernard Connor, while discussing a curious set of skeletal remains found in a French cemetery sometime during the middle to latter half of the 17th century, but the condition has been discovered in remains dating as far back in history as the pharaohs of ancient Egypt (Feldtkeller *et al.,* 2003; Waldron, 2009).

Ankylosing spondylitis will cause lower back stiffness and pain, particularly upon first waking, with an individual either being unable or having great difficulty with spinal forward, rotation, and lateral flexion, meaning the person cannot sit up or twist their torso (Hart, 1980). The pain may be amplified during periods of inactivity, such as during a sleep period (Weisman, 2011). The resulting fusion of the vertebrae and ribs may cause trouble with chest expansion, making it difficult for an individual to breathe deeply (Khan, 2009).

Fusion, which occurs within the sacroiliac joint, is a condition commonly referred to as sacroiliitis (Kettering *et al.,* 1996). Sacroiliitis, when linked to ankylosing will develop bilaterally, however, when ankylosing spondylitis is not present, it can occur unilaterally. This condition will have similar symptoms and patient complaints to ankylosing spondylitis such as chronic lower back pain, as well as additional chronic pain in the buttocks (Agarwal, 1980)

Degenerative disc disease is a vertebral joint condition which affects the central portion, or nucleus pulposus, of the intervertebral disc and corresponding intervertebral surface (see Figure 3.8) (Roemer *et al.,* 2006). This condition is characterized by pitting on the intervertebral surface, along with osteophytes forming along the edge of the marginal surface, resulting in a lipped appearance. The degenerative nature of this condition is believed to be caused by the damage or wear to the intervertebral disc and bony protrusions into the intervertebral foramen are not uncommon (Khan, 2002). These protrusions can damage the nerve connections and arterial passages, causing further degeneration (Kettering *et al.,* 1996). As the condition progresses and the osteophytes continue to form, fusion may occur, but unlike ankylosing spondylitis, bilateral and continuous fusion are not requirements. The damage to the nerves and growth of the osteophytes can be the cause of the pain and stiffness that an individual may feel in their lower back.

Figure 3.8 Lumbar vertebrae with a spondyloarthropathy, notably degenerative disc disease, and a combination of osteoarthritis and enthesophytes. These phytic formations have evidence for broken fusion and pseudo-joints forming. Photo is courtesy of Andy Brown and the Image Speaks project.

3.3.1 Clinical Approaches for Identification and Classification of Vertebral Joint Conditions

Much like osteoarthritis, identification of seronegative spondyloarthropathies is preliminarily based on the reporting of physical symptoms by a living patient. Lower back pain, morning stiffness of the back and neck pain are common complaints for patients with seronegative spondyloarthropathies (Kadwani and Mahmud, 2014). Due to the nature of the SnSpA, a blood test cannot be used to test directly for the conditions, however, the antigen HLA-B27 can be tested for (Kettering *et al.,* 1996; Sheehan, 2004). Individuals with the antigen are more likely to have conditions such as ankylosing spondylitis, but presence of HLA-B27 is not a diagnostic test for SnSpA. (Sheehan, 2004).

Without an accurate blood test, and as patient symptoms and complaints are generic to many other aetiologies, imaging techniques are the only reliable option to determine the potential presence of the conditions (Percy and Lentle, 1980). The various techniques can determine if there is inflammation within the joint capsule, or the relative location and progression of potential ossification or fusion of ligaments or soft tissue (Stolwijk *et al.,* 2012). The type of phytic formation can potentially be determined by the shape, location, and appearance to determine if it is an osteophyte, enthesophyte or syndesmosphyte, which can then be used to determine or exclude the different spondyloarthropathies (see Section 3.4.5).

Modern clinical diagnosis of ankylosing spondylitis relies on a combination of clinical and radiographic analyses. There are two traditional methodologies for diagnosis called the Rome criteria and the New York criteria (Moll, 1980) and are used by groups in Europe such as the European Spondyloarthropathy Study Group (ESSG). The diagnoses are similar and have been modified and updated through the years, but the Rome criteria favors clinical examinations, and the New York criteria relies more heavily on radiographical assessments (Akgul and Ozgocmen, 2011). The diagnoses can be split between definite and probable, similar to osteological assessments (Weisman, 2011). The modern criteria for diagnosis as used by ESSG, an amalgamation of both the Rome and New York criteria, includes chronic lower back pain and stiffness for longer than three months that improves with exercise, limitation of lumbar mobility in the sagittal and frontal ranges, limited chest expansion in comparison with similar age and sex groups (Khan, 2002; Akgul and Ozgocmen, 2011; Weisman, 2011). The radiographic components use a scoring method to determine the level of fusion between the sacrum and iliac surface. For a definite diagnosis, the radiographic component and one clinical component must be present. For a probable diagnosis of AS, three clinical components or the radiographic components must be present.

As sacroiliitis can be used to diagnose ankylosing spondylitis, the condition needs to be diagnosed as well. Similar to ankylosing spondylitis, an individual will exhibit symptoms of chronic lower lumbar back pain and stiffness which may or may not include the buttock area (Slobodin *et al.,* 2016). However, as the sacroiliac joint is a relatively stationary feature with little movement, and the joint is comprised of deep and angular surfaces, this can cause the resulting inflammation to be difficult to detect in a clinical diagnoses (Khan, 2002) Sacroiliitis can, however, be apparent on a radiograph. In early stages, it is characterized by a widening of the joint space and blurry peripheral features (Moll 1980). Bilateral bony sclerosis will soon

develop alongside the joint and in latter stages it will exhibit a reduction of the joint space, which may be accompanied by fusion (Moll, 1980; Khan, 2002; Weisman, 2011).

Degenerative disc disease, as with AS, can produce chronic lower back pain and stiffness. The condition is found more often in individuals with increased age and may cause a decrease in height as the intervertebral disc space compressed. DDD is most accurately diagnosed via radiographic imaging, such as the MRI (Oktay *et al.,* 2014). The clinician will evaluate the image for a reduction within the intervertebral space consisting of the intervertebral disc. Schmorl's nodes are a regular feature of DDD, along with annular tears and osteophytes (Kanna *et al.,* 2013) (see Section 2.4.7). Planar shape, intensity of change, context and texture are radiographic features that are assessed and scored for degradation or irregularity and new computer programs are able to accurately diagnose DDD in the lumbar vertebrae using MRI scans of the sagittal plane (Oktay *et al.,* 2014).

3.3.2 Osteological Approaches for Identification and Classification of Vertebral Joint Conditions

Philip Sager (1969) proposed a method for scoring spondyloarthropathies (SpA) in the cervical vertebrae. Sager created detailed stages for each marker of arthritis and spondyloarthropathies on each individual element on the vertebra. The method allows for researchers to review the recorded stage of the condition and know what markers, and their progression, are present. These methodologies are extremely useful when a researcher is not able to review the physical skeletal materials or photographs, but still have the ability to provide an accurate impression of what is happening to the bone.

The method combines a complex and detailed written scoring guide with pictures of each marker, at each stage, allowing for reproducible and more objective results. The method is akin to the K-L scale, but instead of set stages for the entirety of the condition, each stage represents a single marker. The method helps to display the extent of degradation of the element, as well as provide results that are unique to each individual. For example, unlike the K-L scale, a person can have different stages for the different markers.

A basic approach to diagnose ankylosing spondylitis relies upon recording the presence of fused, or ankylosed, syndesmophytes located bilaterally on vertebral bodies (Rogers and

Waldron, 1995). This fusion will most commonly begin development in the lower vertebrae and may include sacroiliitis or even fusion of that joint (Waldron, 2012). Fusion between the vertebrae and the presence of sacroiliitis is characteristic of a relatively advanced stage of the condition, making it difficult to diagnose in less advanced stages, prior to such fusion.

Degenerative disc disease uses many of the same osteological markers for osteoarthritis, but the clinical symptoms can be similar to AS. Pitting and/or porosity on the surface of the intervertebral plate and marginal osteophytes are the only markers required for a diagnosis (Waldron, 2009). However, there can be many more changes to the intervertebral plate, such as plaques of new bone, compression, or Schmorl's nodes that if present, could denote the possible presence of DDD (Roberts and Manchester, 2005). The intervertebral surfaces will undergo degenerative changes with age; therefore, a researcher must be aware of the difference between a vertebra with age related degeneration and a vertebra with pathological degenerative change.

3.4 Osteological Markers for Osteoarthritis and Seronegative Spondyloarthropathies

The osteological markers for osteoarthritis and seronegative spondyloarthropathies are also associated with a multitude of other pathological conditions. Singularly, they have limited diagnostic value, but in combination they can be used to differentiate and diagnose the joint conditions. The markers have different forms of manifestation conditional on the joint or skeletal element they affect. This requires the researcher to be familiar with the appearance of a healthy bone and that of an arthritic bone, as well as that of similar pathological conditions that could provide for differentials.

3.4.1 Eburnation

Eburnation is a result of long-term bone on bone friction (Sager 1969; Rogers and Waldron, 1995), caused by a reduction to the joint space (see Figure 3.9) (Burt *et al.,* 2013). This feature is uniquely pathognomic of OA and therefore the only marker which can give a diagnosis of OA alone (Rogers and Waldon, 1995). Eburnation manifests as an area of the

surface of the bone has become smooth and reflective. It may be accompanied by ridging along the surface where the corresponding bone planes have ground across each other.

Eburnation has three primary characteristics. The surface area of the bone that encompasses the eburnation will become sclerotic, or thickened, and will appear on a radiograph as sclerosis (see Section 3.4.2) (Roberts and Manchester, 2005). The surface will have developed a smooth texture compared to the normal porous or rough texture (Rogers and Waldron, 1995). Finally, the resulting structure is a highly reflective area of smoothed bone that resembles polished ivory. Taphonomic polishing, caused by the handling of skeletal elements and over time, can be confused with eburnation, but does not have the same ivory-like surface as eburnation.

Figure 3.9 Light source on a femoral head highlighting the glossy sheen of developed eburnation. Note that the area of eburnation appears to be raised from the normal joint surface which can be indicative of subchondral sclerosis.

3.4.2 Subchondral Sclerosis

Subchondral sclerosis is a reactive process resulting in the thickening of the subchondral bone under the joint surface (Sager, 1969; Rogers and Waldron, 1995). Sclerosis often occurs adjacent to eburnation on the surface of the bone (White and Folkens, 2005). The thickening of the subchondral bone layer is linked to the reduction of articular cartilage and

ossification at the endochondral bone layer across the entire joint (Cox *et al.,* 2013) and at the local level, severe degeneration of the cartilage may cause additional sclerosis (Cox *et al.,* 2012). This increase to the endochondral thickness can see an increase to the trabecular volume and can be linked to loading (Cox *et al.,* 2013; Crema *et al.,* 2014). One development that should be mentioned is that a bone with osteoarthritis will exhibit an increase to vascular or marrow pathways, with this increase being linked to the increase in further creation of subchondral bone enhancing sclerosis (Wong *et al.,* 2009; Pan *et al.,* 2012). Subchondral sclerosis is typically not visible through standard examination and so without either radiographic evidence or the creation of a cross section of the joint via bisection it is difficult to assess. On a radiograph, the area of sclerosis appears as white indicating a dense area of bone.

3.4.3 Porosity and Pitting

Porosity of the joint surface is characterized by multiple small holes which penetrate the subchondral bone to connect with cavities within the trabecular bone (see Figure 3.10). Porosity is commonly classified by the size of holes into two categories: micro porosity, less than 2mm in diameter, and macro porosity, greater than 2mm in diameter (Sager, 1969; Rogers and Waldron, 1995). Porosity can be caused by either the resorption of the bone or by the synovial fluid within the joint capsule intruding into the bone (Roemer *et al.,* 2006; Gunn, 2018). Porosity is similar to and can be confused with deep pitting, which is thought to have comparable origins, but perforates rather than penetrates the bone (Roberts and Manchester, 2005). The differences between porosity and pitting can be helped to differentiate with a light source and magnification tool.

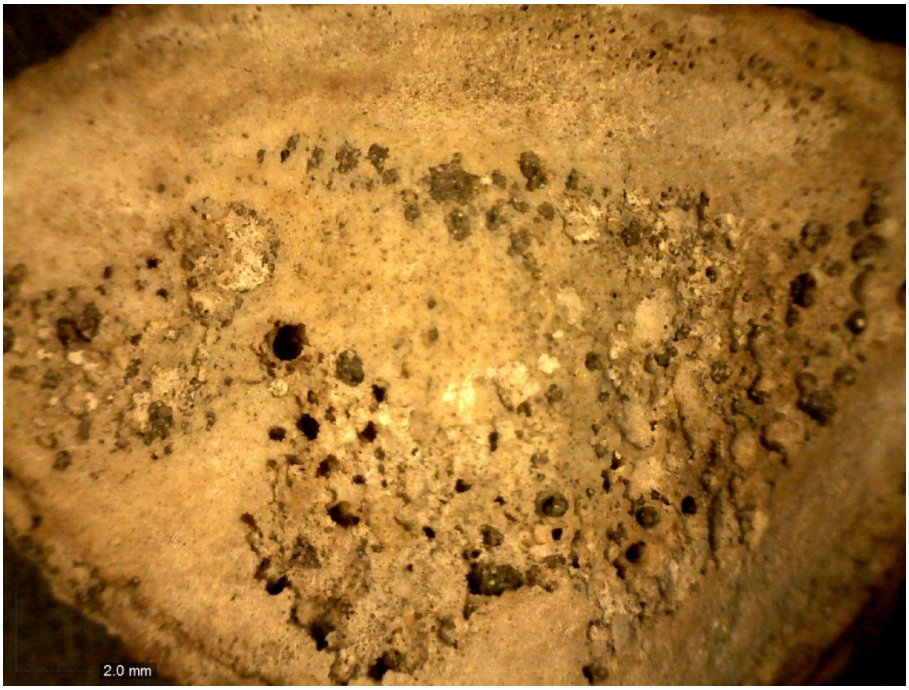

Figure 3.10 The perforating holes leading into the cortical bone of the patella are an example of porosity. This sample is a mix of micro- and macroporosity as well as taphonomic damage. This photo also helps to illustrate the need to be able to differentiate pathological porosity from taphonomic damages. For another example of porosity See Figure 3.7.

3.3.4 Subchondral Cysts

Subchondral cysts, also known as synovial cysts, are a sac-like structure that forms within the subchondral layer of bone (see Figure 3.11) (Sager, 1969; Rogers and Waldron, 1995). These cysts can also be associated with rheumatoid arthritis, calcium pyrophosphate deposition disease and osteonecrosis, though the relationships and aetiologies concerning the formation of the cysts are not known (Chiba *et al.,* 2014). Two leading theories as to the cause of the marker in relation to osteoarthritis are (1) traumatic injury (such as a fall with vertical landing) to the joint resulting in the resorption of the subchondral bone and (2) synovial fluid intruding through the periosteum into the subchondral bone during the collapse of the joint space (Roemer *et al*., 2006). Regardless of the theorized cause, the result is a cystic cavity in the subchondral bone that, as with sclerosis, is only visible on a radiograph unless damaged or a cross section is completed.

Figure 3.11 This large perforating hole may be linked to a subchondral cyst. An external void, such as this one, is the only way to be able to potentially determine the presence of a cyst without radiographs or destructive analyses. Note the surrounding porosity and the raised area of bone with eburnation.

3.4.5 Phytic Formations

Osteophytic formations are bony spurs which develop on the surface of the bone around synovial joints and can be divided into two categories: marginal and surface (van der Kraan and van der Berg, 2007). Osteoarthritis are formations of new bone that develop along the rim of a joint (see Figure 3.12). Surface osteophytes develop on the surface of the bone and can resemble a pimple in their early formation. Osteophytic formations can often be confused with enthesophytes or syndesmophytes, and to differentiate between the type of phytic formations, knowing the shape and the origin point is required (see Figure 3.12).

Osteophytes are thought to be a joint stabilizing mechanism and may lead to fusion of the joint, which will be discussed in the following section (Al-Rawahi *et al.,* 2011). Marginal osteophytic formations follow the membrane of the synovial capsule and could be indicative of a breakdown of the capsule (van der Kraan and van der Berg, 2007). Recent studies have explored and found relationships between osteophytes and mechanically induced stress (Venne *et al.,* 2020).

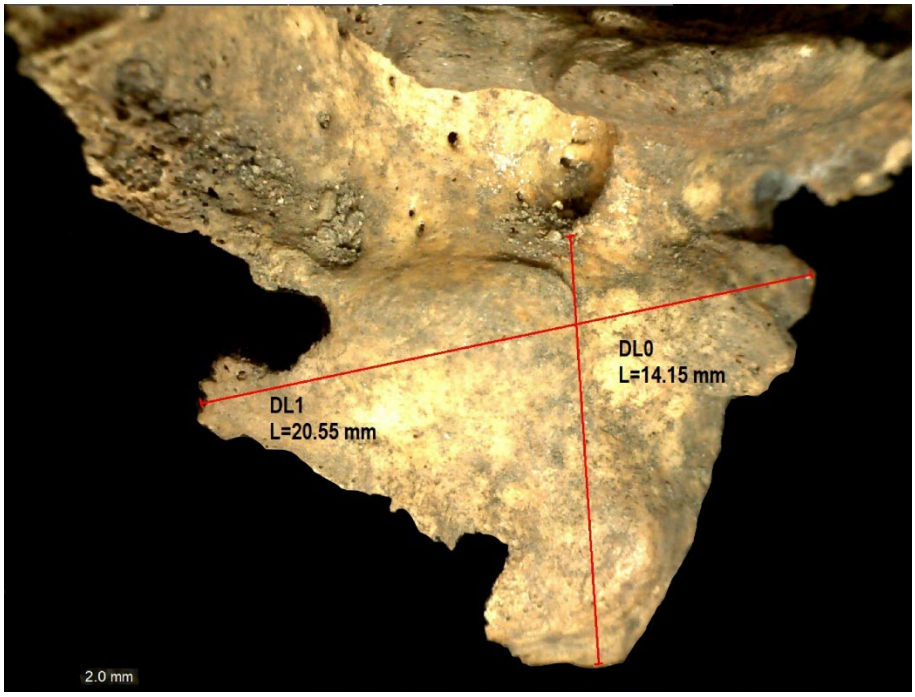

Figure 3.12 This claw-like osteophyte is a large representation of the types of phytic formations typically seen on a vertebral body.

Enthesophytes, a form of enthesopathies or entheseal changes, resemble osteophytes, in that they can form long, thin bridges of bone, but form at the points of origin or insertion for tendons and ligaments (Foster *et al.,* 2014; Villotte *et al.,* 2016) and result from the ossification of the tendons connecting muscle to bone, or ligaments connecting bone to bone (Waldron, 2009). Enthesophytes are therefore distinguished from osteophytes both by their location, appearance and aetiology. Osteophytes will have a curved or claw like feature, forming first along the horizontal axis and then curving vertically, while enthesophytes follow the path of the soft tissue, which develop in a singular directionality (see Figure 3.13) (Zumwalt, 2005; Villotte *et al.,* 2016). Two categories of entheses exist: fibrocartilaginous and fibrous (Foster *et al.,* 2014; Jurmain *et*

al. 2016). These terms refer to the anatomical structure of the attachment to the bone in which fibrous entheses attach directly at the bone, while fibrocartilaginous entheses have four structural zones (Benjamin *et al.,* 2006). Fibrous entheses attach to the bone through dense fibrous connective tissue and are commonly found on the metaphyses and diaphyses of long bones (e.g., adductor magnus and deltoid attachments), while the fibrocartilaginous entheses mediate the transition from soft to hard tissue via fibrocartilage and are commonly found at the epiphyses and apophyses (e.g., Achilles' tendon and rotator cuff) (Apostolakos *et al.,* 2014).

Regardless of the attachment medium, the structure is heterogeneous with different mechanical properties for the entheses and the bone (Alves-Cardosa and Assis, 2021).

While the exact aetiology of enthesophytes are largely unknown. Enthesophytes have been associated with increasing age and are largely considered a natural result of the aging process (Wilczak, 1998; Villotte and Knüsel, 2013; Nikita *et al.,* 2019). In a young individual, micro-stressors of repetitive and excessive mechanized stress can cause the development of enthesopathies and, in older individuals, enthesopathies can form due to decreased vascularization of the tendon (Jurmain *et al.,* 2012). Therefore, in an older individual, it may be difficult to determine the cause of enthesopathies without further examination of the skeletal structure and mechanisms. Entheseal changes have also found to be positively correlated and associated with increased body mass (Weiss 2004;2007; Weiss *et al.,* 2012).

Due to the developmental nature of entheses, at this time, it is impossible to accurately deduce specific types of activity, however, in combination with other methodologies, it may still be possible to infer activity levels (Jurmain *et al.,* 2012; Mazza, 2019). For instance, a recent study has shown that living in different types of terrain can impact the frequency of entheseal change in the population, and that higher mechanical loading during childhood could, in fact, help to protect against entheseal changes (Acosta *et al.,* 2017). This is extremely useful information to have to determine potential causes for osteoarthritis in a skeletal population and will be discussed further in the following chapter.

Figure 3.13 Entheseal growth developing anteriorly from the tibial tuberosity. This bony growth follows the path of the patellar ligament.

Syndesmophytes are a type of enthesophyte and are the result of ossification of the annulus fibrosis, the tough outer layer of the intervertebral disc (see Section 3.1.2/ Figure 3.14). Syndesmophytes are indicative of ankylosing spondylitis, which one study found in 75% of patients, and have a unique development and appearance than other osteophytes or enthesophytes (Baraliakos *et al.,* 2007). One such unique development is that these syndesmophytes may form bridges of ossified soft tissues between two vertebral bodies (van Tubergen *et al.,* 2012) The appearance resulting of the fusion of the vertebral bodies resembles that of a shaft of bamboo, leading to the term 'bamboo spine' (Rogers and Waldron 1995; van Tubergen *et al.* 2012).

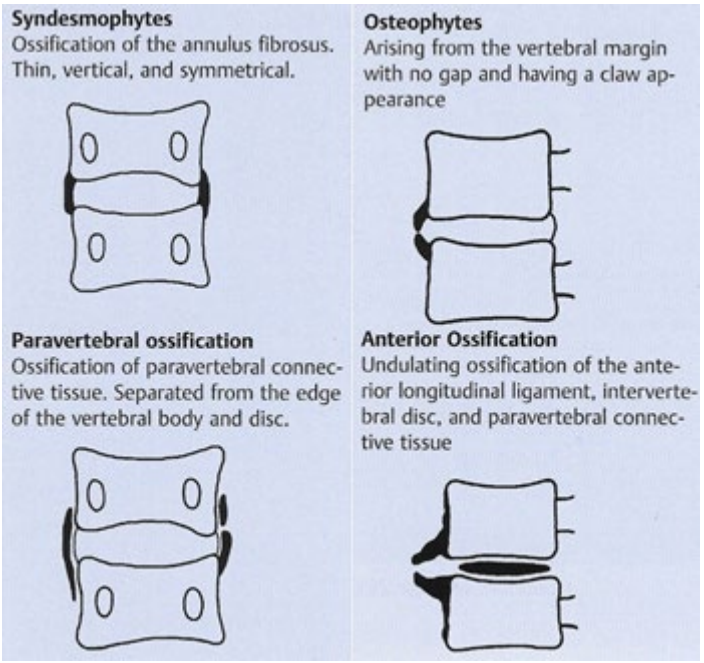

Figure 3.14 The different types of phytic formations as seen on the vertebrae and how to differentiate them. Note that enthesophytes would fall into the paravertebral and anterior ossification categories (Kormano and Pudas 2006: 259).

3.4.6 Fusion and Ankylosis

The anatomical term for the fusion of two or more skeletal elements via osteophytes (see Figure 3.15) or ossification of soft tissue is ankylosis. Ankylosis arising from joint conditions most commonly occurs between the vertebrae, such as in the seronegative spondyloarthropathy ankylosing spondylitis (Sager, 1969; Rogers and Waldron, 1995; Burt *et al.,* 2013). A potential cause of fusion in joint conditions, which arises from the ossification of muscle, tendons, and ligaments, could be part of the body's attempt at stabilizing a weakened joint structure (Roberts and Manchester, 2010). As well as stabilization, fusion can also be a symptom of traumatic injury or pathology, such as tuberculosis, ossifying the soft tissue and so is not a specific trait of vertebral joint conditions (Burt *et al.,* 2013). Fusion is not always the gross or complete joining of elements and can occur at a single location of a joint. Fusion can occur on any joint, but is more typical, and often more advanced, in less mobile joints.

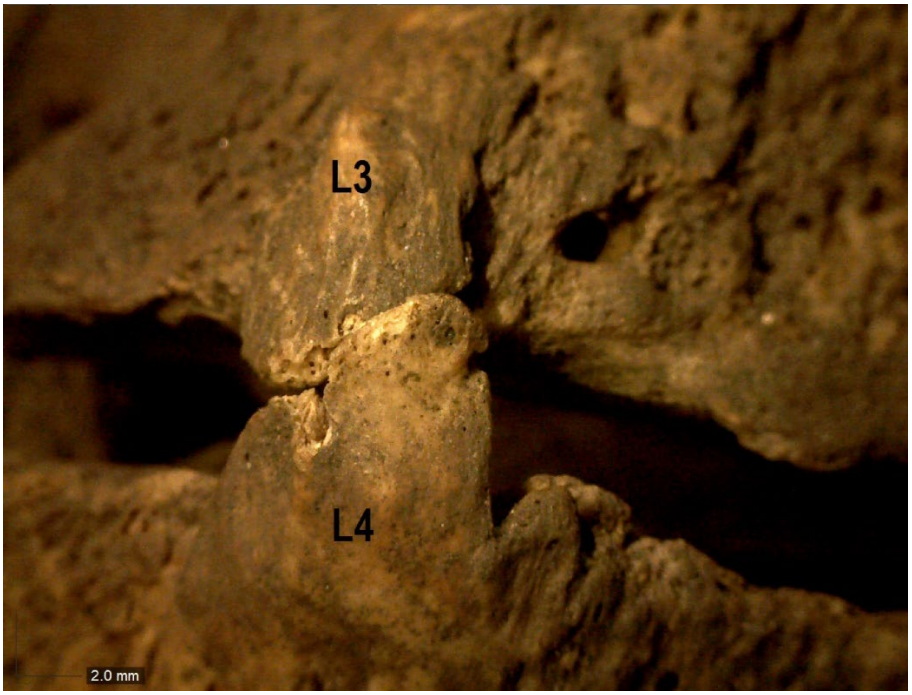

Figure 3.15 These enthesopathies show the early stages of fusion between two vertebral bodies. While the two elements are separate, there is evidence that these two enthesophytes were fused but have broken post-mortem.

3.4.7 Schmorl's Nodes

Schmorl's nodes are depressions on the intervertebral surface of the vertebral bodies (Burt *et al.,* 2013) and are a common occurrence on the thoracolumbar elements (see Figure 3.16 and Figure 3.17) (Kyere *et al.,* 2012). These nodes are the only marker listed within this section that is solely a characteristic of DDD and not OA or AS. The size of the nodes may vary but the shape is roughly oval, often resembling a kidney shape, and generally located along the central line of the surface dividing the left and right portions of the vertebral body (Sager, 1969; Rogers and Waldron, 1995).

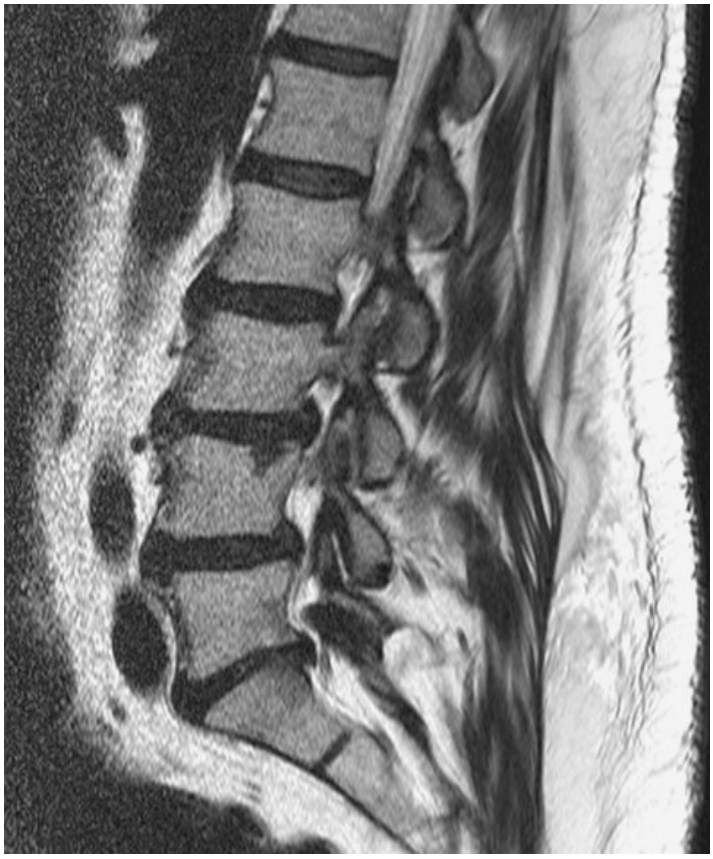

Figure 3.16 Schmorl's node on a lumbar vertebra as appearing on the radiograph of a living individual (Kyere *et al.*, 2012).

There is no consensus as to the pathogenesis of Schmorl's nodes and currently two theories exist as to the origins of such nodes. The first theory is that due childhood developmental issues, the nodes develop early in life and the negative space in the bone may lead to herniation. Due to the higher levels of stress to the thoracolumbar vertebrae, it is argued that this location is more prone to early trauma (Hilton *et al.* 1976). The second theory is that the nodes are believed to be caused by a rupture or herniation of the nucleus pulposus penetrating the intervertebral surface (Vernon-Roberts *et al.,* 2007; Kyere *et al.,* 2012). While these theories vary, the core argument remains similar in that herniation of the intervertebral disc is the cause and may in fact not have a single root cause, but a single effect.

Schmorl's nodes are found to be more frequent in males than females and may result in pain (Kyere *et al.,* 2012). In a study conducted by Moustarhfir *et al*., (2016) Schmorl's nodes were found to be more common in Caucasian individuals than Africans and individuals working in occupations for more than ten years that revolved about manual labour, than individuals working in such an occupation for less than ten years or sedentary workers. That

study also found that athletes participating in the activity for more than five hours per week had higher rates than those working less than five hours per week (Moustarhfir *et al.,* 2016).

Figure 3.17 The right indentation on the intervertebral surface is a Schmorl's node. Note the rough oval, almost kidney like shape and the smoothed edges turning into the indentation.

3.4.8 Reactive Bone Formations

Reactive bone formations, or new bone formations, are a generalized term to describe osteoblastic activity on the joint surface (see Figure 3.18) (Sager, 1969; Rogers and Waldron, 1995; Ortner, 2003). The formations can vary in size and shape, from small pimple-like formations no larger than 2mm to larger plateau-like formations larger than 2mm. Due to the degredation of the joint, the supposed cause is a stabilizing effect, although there is no definitive answer for why the reactive bone formations occur on the joint surface.

Figure 3.18 The bony growths, to the left of the fovea capitis, are larger than the typical growths and demonstrate the extent to which they can form. Note the irregular shape and overlapping folds along the surface of the femoral head.

3.5 Summation of Joint Conditions

The markers used within for this research are generic osteological markers that can be found in a host of pathologies, and while singly they provide no diagnostic value, a group of markers can help to diagnose the joint conditions. The diagnostic criteria must not include an examination of these markers looking for joint condition, but an objective analysis as differential diagnoses are essential for accurate and complete examination. Pathologies can have similar aetiologies and/or similar appearance on the bone and so it is important for the researcher to be able to accurately distinguish the pathologies and justify diagnoses with clear evidence.

The clinical and osteological methods for identifying and diagnosing are similar, yet vastly different. The clinician deals with subjective values of pain and severity of symptoms and objective radiographic images, while the osteologists can directly observe the skeletal material in an objective manner. However, neither method can truly capture the entire picture as they both lack key features. The clinicians lack direct observation of the skeletal material, while the osteologist lacks access to the soft tissue and the effects the condition has on the

living patient. Therefore, it is important for researchers to understand the principles and have a familiarity with the literature and studies of the differing fields.

Chapter 4 Risk Factors

There are numerous risk factors that have been associated with the development of osteoarthritis and vertebral joint conditions by both clinicians and palaeopathologists (Oliveria *et al.,* 1999; Lohmander and Felson, 2004). The risk factors for arthritic conditions are often co-dependent, acting in concert, but also include a level of independent causality. Age has traditionally been considered the chief risk factor in OA and is one of the few variables that could be considered an independent risk factor (Goldring and Goldring, 2007; Glyn-Jones, 2015), with biological sex being another. Dependent risk factors can include activity levels, diet, high body mass index, and genetic disposition. In SnSpA, the latter factor, genetic predisposition, seems to play a much more central role (Khan, 2009). As with the markers, the dependent risk factors do not have much significance when considered in a vacuum, but when the risk factors are combined, they can create the perfect storm for the development of arthritic conditions. The influence of different risk factors is not uniform across different joints and will vary, for example, depending on the function of the joint in question (Felson and Chaisson, 1997). Of the following risk factors discussed, only age and biological sex show relationships with ankylosing spondylitis.

4.1 Age

Age is the most discussed risk factor for arthritic conditions (Nevitt 2006; Burt *et al.,* 2013). Age has been implicated as the key risk factor in OA due to strong correlations reported between its progression and advancing age (Goldring and Goldring, 2007; Glyn-Jones *et al.,* 2015). Individuals have an increased likelihood for arthritic clinical symptoms and osteological markers to develop over the age of 55 (Rogers and Waldron, 1995; Roberts and Manchester, 2010). As advancing age is accompanied by the gradual breakdown of the biomechanical functions of the body's joints, it seems feasible that age should be considered the chief risk factor in joint condition progression.

However, more research over the last 20 years has questioned the direct correlation between age and the progression of osteoarthritis (Anderson and Loeser, 2010). In the modern world, individuals are living to older ages and yet some show no outward symptoms or signs of the condition, even into their ninth or tenth decade (Williams and Spector, 2006). That some

individuals should progress into later life completely free of osteoarthritis suggests that there must be a differential risk associated with the disease that is not purely age dependent. Other factors must contribute to the onset and progression of degenerative joint conditions.

While it has been shown that older individuals have a higher likelihood for developing joint condition, this relationship may arise from the accumulation of the other risk factors throughout a long life. One example that demonstrates this issue is the difference in rates of OA between males and females. Clinical studies have demonstrated that women are more likely to develop OA earlier in life than their male counterparts, and this has been explained as a result of hormonal changes, especially those associated with the menopause (Burt *et al.,* 2013; Mahajan and Patni, 2018; Prieto-Alhambra *et al.,* 2014). Therefore, in this case, it is not age that is primarily responsible for the relationship, but rather hormonal changes that have an age-dependent component.

Similar arguments can also be made for other risk factors as well, as their effects are cumulative and therefore do have an age-related nature (Holliday *et al.,* 2011). An appreciation of the coincidence of age and other risk factors would explain why there are individuals that live to older ages that show no symptoms of the conditions. An appreciation of the complex aetiology of osteoarthritis thus requires a detailed consideration of several risk factors other than age (Anderson and Loeser, 2010). For example, an individual becoming obese at a younger age, has a higher likelihood for the development and faster progression of the condition than individuals of the same age who are not obese (Holliday *et al.,* 2011). The earlier the risk factors develop, the higher the chance of becoming symptomatic, however, a lifestyle change resulting in healthier living, such as the reduction of mass by 5kg, has an increased chance of allowing the body symptoms of the conditions dissipate or become minimalized (Felson *et al.,* 1992; Johnson and Hunter, 2014).

4.2 Biological Sex

Biological sex has been found to affect the onset of the joint conditions (Roman-Blas *et al.,* 2009; Cross *et al.,* 2014). Ankylosing spondylitis is more frequent in males than females (3:1), however, the reason for this variance is largely unknown (Weissman, 2011). Men have higher rates of OA <45 years of age, thought to be due to injury (secondary OA), and women having higher rates >55 years of age and at more joint locations (Williams and Spector, 2006).

The factors behind the differing onset are multidimensional and thought to be caused by differing pathways relating to the basic differences of sex, such as hormones and reproduction (Williams and Spector, 2006; Liu *et al.,* 2009, Roman-Blas *et al.,* 2009). These factors affect each joint in contrasting ways and to varying degrees and contain much debate as to the relationships, with new studies being conducted to try and explain statistical discrepancies found.

Hormonal factors are thought to be a leading cause for the onset of primary joint degradation in women at younger ages (<55), however it is still unclear the direct role that hormones may play (Liu *et al.,* 2009; Roman-Blas *et al.,* 2009). The Million Women Study found that women that underwent a younger age of menarche, or onset of menstruation, (<11) experienced higher rates of hip and knee osteoarthritis to a degree needing replacement (Liu *et al.,* 2009). Sexual hormone deficiencies cause an imbalance within the skeletal system, which can lead to a weakening of the joint space and the surrounding bone, which may cause osteoporosis, and ultimately leading to a degradation of the joint, with joint space reduction, and potential osteoarthritis (Almeida *et al.,* 2017). One such hormonal deficiency that occurs in all women, usually between the ages of 45 – 55, is menopause and post-menopause, although reports are conflicted as to the relationship and effect on the joint (Liu *et al.,* 2009; Sacitharan and Vincent, 2016; Almeida *et al.,* 2017). Due to the relationship menopause and similar hormonal deficiencies have with osteoporosis, but the lack of a solid relationship between such deficiencies and OA, it may be reasonable to conclude that OA is then a secondary effect related instead to osteoporosis, not the hormones.

One of the most singular differences between the sexes is the reproduction process. Women have the ability to conceive and bear a child, which causes large amounts of strain to the body, as well as fluctuating hormone levels. This rapid weight gain and with accompanying stress and then return to normalcy, potentially multiple times, can cause a degradation to the hip and knee joints faster than males (Jørgensen *et al.,* 2011). The Million Women Study collected data on 1.3 million women, average age of 56, through the National Health Service of England and Scotland. The study found that the incidence of hip replacement due to osteoarthritis was increased by 2% per birth and 8% per birth for the knee (Liu *et al.,* 2009). A Dutch study found that the overall risk for OA to the spine and knee was higher for men with at least one child than the similar cohort of women (1.22/1.10), however, after adjusting for age and sociodemographic factors, women had a slightly higher risk (1.07/1.04) (Jørgensen *et*

al., 2011). The conflicting information demonstrates the complexity of the issue and relationships with other factors.

4.3 Activity

The impact of activity on the progression of joint condition is twofold, as there is reason to believe that both activity and the injuries of active individuals may act as a catalyst for the condition (Lequesne *et al.,* 1997; Carter *et al.,* 2015). The meniscus of the joint relies on a combination of anabolic and catabolic cells, known as fibrochondrocytes, which maintain the biomechanical integrity of the structure (McNulty and Guilak, 2015). The menisci are affected by genetics, which can mean that certain individuals simply have stronger menisci that can withstand more external forces or weaker menisci that can be prone to fail with less external strain, and there is a mid-load/intensity range within which the activity level is at its best to both maintain joint and muscle strengths (Fergusson, 1987), which would indicate that inactivity can be just as damaging to the joint as excess activity levels. To fall below that range will shift metabolism to catabolism (Griffin and Guilak, 2005) and to exceed the range will add stress to the joint, but both will increase the chance for degeneration of the joint. A balance of activity is required in the mid-load/intensity range to maintain joint integrity and decrease the chance for joint degeneration and onset of osteoarthritis. Inactivity when combined with obesity and osteoarthritis appear to have a cyclical logic: increased weight leads to progression of osteoarthritis which limits mobility and leads to further weight gain, as well as having higher chances for bilateral development over unilateral (Felson and Chaisson, 1997). However, by their very nature, joints will wear down and a common-sense reasoning develops around the idea that the more a joint is exploited, the faster the break down. It would appear to be inevitable, as the age risk factor seems to support the idea, yet not everyone will develop arthritis.

One issue, that appears counter-intuitive, is that exercise and regular physical activity may be beneficial for the health of the human body but can be harmful to the joint capsule. This can be seen in athletes that have developed arthritic symptoms at younger ages, than an individual of the same age that has a lower level of activity (Vannini *et al.,* 2016). A study of athletes with anterior cruciate ligament (ACL) injuries showed high prevalence rates for the symptoms of arthritic conditions developing at younger ages (Lohmander *et al.,* 1999; Vannini

et al., 2016), which could then indicate that the activity is not the cause, but the stress injuries brought about by the activity. Another explanation could be seen in a study conducted by Calce and colleagues (2018), regarding the relationship between cross-sectional skeletal properties and the prevalence of osteoarthritis. The authors of the study found a negative correlation between femoral robusticity and OA, which would suggest some form of protective property as torsional strength increases.

There are three essential ranges of activity: low, medium, high. Within the medium range, the joints are operating at peak efficiency, maintaining homeostasis within the entire joint structure. Within the low range, the body switches from metabolic to catabolic processes and can begin to cannibalize the materials utilized within the joint (Griffin and Guilak, 2005). When catabolic processes begin to occur, the structural integrity of the joint space and surrounding skeletal structures become compromised. Examples of this happening are found in amputees, paraplegics, and astronauts, where the decreased load and intensity of use results in decrease of muscle and skeletal mass. When the body is pushed to within the higher load ranges, there is a higher likelihood of injury to the joint or corresponding limbs. The increased loads will intensify the stress to the joint system, thereby increasing the wear.

In the palaeopathological world, it was believed that entheseal change was related to activity and larger entheses equated to an increase of activity (Palmer *et al.,* 2016; Alves-Cordosa and Assis, 2021). This has been found to be only partially correct, as entheseal changes are strongly related to age, yet broad activity differences are possible to be assessed for (pushing/pulling, individuals performing heavy physical tasks, sedentary), but not specific types (hammering, punching, kicking, etc.) (Milella *et al.*, 2015). These entheseal changes can be linked to OA and the degenerative processes of the femur ($r = .425$), however, after testing different locations, such as the humerus, no such relationship was found ($r = .098$) (He and de Almeida-Prado, 2021). A similar study using remains from an early post-medieval assemblage from Poland found entheseal changes to be the strongest predictor for the development of OA ($p = .041$) (Myzka *et al.,* 2020). This study also found that entheseal changes correlated with certain joints (wrist of hip) but not others (shoulder, elbow, knee, ankle). Studies like these could indicate that while activity plays a part in the degenerative process, the entheseal changes form independently or concurrently from the OA.

A potential gendered/sex-based division of labour has been theorized in past populations and a study of post-medieval Dutch populations found differing levels of entheseal

changes and OA (Palmer *et al.,* 2016). This study showed that entheseal changes had developed at different attachment sites for males and females, with different activity levels associated with those muscle groups (males = lifting/heavy loads; females = pushing/pulling). Palmer and colleagues (2016) also found that that EC was not significant in relation to OA. Similar findings were reported by Weiss and colleagues (2012), in which the researchers gathered data from a pre-Columbian northern California population and found the development of entheseal changes varied by sex, as well as body size. While the authors found reverse trends in the pattern of entheseal development to older studies concerning potential established activity for each sex, the differences between the sexes would still illustrate the potential for a gendered/sex-based division of labour.

4.4 Body Mass

The biomechanical structures that make up the musculoskeletal frame are only capable of supporting a given mass, and any increase above the maximum tolerance will stress the load bearing joints. Obesity places an increased stress on the mechanical loading of the joint as well as having the potential for causing neuroendocrine-metabolic stimulus, which can cause the body's internal systems to fall out of homeostasis (Brahmabhatt *et al.,* 1998). The body mass index (BMI) was designed by clinicians to determine a healthy weight for an individual and is measured as kg/m². Women have been to have a higher association between obesity and osteoarthritis, mainly at the knee, than men (Williams, 2006). However, a study by Holmberg and colleagues (2005) found that men displayed significance with knee OA and normal a normal BMI (<25 kg/m²) around the age of 30. The Holmberg and colleagues (2005) study may suggest that it is not simply the weight, but the location of the addition weight, such as it being more common for men around the midsection and for women around the thighs and bottom, affecting the condition.

Using odds ratios shows that the chance of developing knee OA increases as BMI increases. A study of normal weight individuals showed that OR increased by a factor of 4 when comparing males <23 kg/m² and then a group 23-25 kg/m² (Holmberg *et al.*, 2005). In obese individuals the OR increases by a factor of 13.6 with a 36 kg/m² or higher BMI compared to the 0.1 for individuals that fall within the normal weight BMI spectrum (Coggon *et al.,* 2001). Individuals that fall into the morbidly obese spectrum present higher rates of joint

pathologies with an abundance of clinical data focused on OA in the knee (Shiozaki *et al.,* 1999; Syed and Davis 2000; DeVita and Hortobágyi, 2003). According to a study of BMI compared with knee OA, 18.3% of individuals between 20.0 – 24.9 kg/m² category, 47% of individuals between 25.0-29.9 kg/m² and 33.7% over 30.0 kg/m² showed evidence of knee OA (Coggon *et al.,* 2001). Obese individuals are 33% more likely to require a knee replacement due to increased stress and deterioration (Mooney *et al.,* 2011), with 24% of surgeries in England performed on obese individuals (Coggon *et al.,* 2001). Women who are obese have shown higher rates of developing OA in the knee than obese males (Felson and Chaisson, 1997, Coggon *et al.,* 2001). Obesity and increased mass may cause morphological changes to the skeleton, increasing skeletal mass as the body seeks to compensate with the increase stress (Brahmabhatt *et al.,* 1998, Auerbach and Ruff, 2004). The chance of development of knee OA in obese individual increases to 13.6 with a 36 kg/m² or higher BMI compared to the 0.1 for individuals that fall within the normal weight BMI spectrum (Coggon *et al.,* 2001).

The scale for BMI is 0 – 18.4 kg/m² underweight, 18.5 – 24.9 kg/m² normal weight, 25 – 39.9 kg/m² overweight, and 30 kg/m²+ obese, with different levels of obesity. An individual weighing 80kg with a height of 1.82m would have a BMI of 24.2 kg/m²: normal. An individual weighing 110kg with a height of 1.82m would have a BMI of 33.2 kg/m²: obese (WHO, 2000). Increased weight does not equal obesity creating an inherent flaw with the BMI chart, as individuals with increased muscle mass will have increased weight and therefore score higher. For instance, in the example above, the individual with the obese BMI score could be a body builder with less than 8% body fat, not an individual that would be considered grossly overweight or obese. However, for the purposes of examining osteoarthritis, individuals with a BMI in the overweight to obese ranges should still, in theory, have increased wear over time to load bearing joints (Tözeren, 2000).

Obesity is itself the result of a suite of additional risk factors which include diet, activity levels and genetics. Increased consumption of food or eating foods rich with fats or sugars can cause an increase to body mass in the form of fat. A high caloric diet with a lack of activity can cause an individual to gain mass and become obese as the body is unable to burn off the excess calories (Brahmabhatt *et al.,* 1998). These factors can contribute to increase body mass and obesity as single factors or as a combination of factors.

4.5 Diet

Dietary factors can be linked to body mass; however, the types of food can be as important as the quantity (Williams and Spector, 2006). High fat diets (HFD) and vitamin intake have been linked to the onset and progression of the joint conditions; however, the relationship may not be completely clear whether the affect promotes primary or secondary conditions (Mooney *et al.,* 2011; Chaganti *et al.,* 2014; Sansone *et al.,* 2019). By managing or changing the diet, to include weight loss or anti-inflammatory properties, an individual can manage the effects and progression of OA (Baures and Ivorres, 2019).

High fat diets, which can also be linked to the cause of obesity, have been linked to the onset of OA, especially in combination of a previous injury (Mooney *et al.,* 2011). Research using meta-data from laboratory mice studies found that a HFD will often exacerbate the early stages of OA and induce the onset of the conditions (Sansone *et al.,* 2019). A HFD is linked to the pro-inflammatory gene IL-1β, which is further linked to the onset of OA (Mooney *et al.,* 2011), and the laboratory mice studies showed that the HFD can induce an increase of the gene (Sansone *et al.,* 2019). Therefore, a HFD not only increases the risk of OA by causing obesity and the subsequent stressors of increased mass, but also impacts the joints at the microscopic level via these interleukin (IL) proteins.

The Vitamins C, D and E, which are linked to the development of new bone essential for a healthy joint, have been found associated to the onset and progression of OA when found to be imbalanced within the body (Williams and Spector, 2006). Vitamins C and D are also linked to the anemic disorders, scurvy (Vitamin C) and rickets (Vitamin D), when deficient, which can cause a change to the gait of the individual, causing external stress and wear on the joint, which may be linked (Roberts and Manchester, 2005). Vitamin C and E are antioxidants, once thought to help prevent osteoarthritic conditions, however, high levels have been associated with the onset and progression of OA, especially within the knee (Chaganti *et al.,* 2014). Being deficient or overly saturated with these vitamins can be harmful to the health of the joint, and as with activity, the middle ground just may be essential to a healthy joint (Williams and Spector; 2006; Chaganti *et al.,* 2014).

While diet may be linked to OA, for the purpose of this study, it was not able to be tested for, as that would require destructive methodologies, namely isotopic analysis. Isotopic analysis causes the destruction of the source material, which, over time, decreases the amount

of skeletal material that is available for study and can only give carbon and nitrogen values for of the food sources. These types of analyses are important but require highly focused research questions with many institutions not allowing such practices to preserve the materials.

4.6 Summary

These risk factors, with the exception of diet, have been chosen as they can be assessed on the skeleton without destructive methodologies being utilized. The risk factors are complex, and while considered singly, are often found in conjunction with one of the other risk factors (e.g. lack of activity and increased body mass; sexual dimorphism concerning body size). Unfortunately, as the osteoarchaeological samples do not come with this information, best estimations via established, accurate and repeatable methodologies will need to be created as proxies for the living variants. These techniques will be discussed in Chapter 6.

The joint conditions being assessed within this body of work (osteoarthritis, ankylosing spondylitis, sacroiliitis, and degenerative disc disease) are relatively common and complex conditions. The aetiologies of the pathologies share similarities, making assessment of the markers and skeletal features quick and simple, but it is the differences between these pathologies that can illuminate information on how these individuals lived. While the joint conditions can be assessed with ease, the risk factors require more skillful methods for assessment and analysis. The relationships between the risk factors and the joint conditions display a more complicated relationship and this body of research intends to review and evaluate these relationships, as well as examine the prevalence rates for the populations in comparison to both modern and past data.

Chapter 5 Materials

5.1 Sample Criteria

Skeletal sample selection was based on two factors: age at death and presence of necessary skeletal elements. The first factor required the skeletal material to be that that of a biologically mature individual, assessed to be over the age of c. 18 years at the time of death. This is because research has shown that age is a major factor of the conditions being researched, and juveniles would not provide any data that would be usable due to the rarity of the conditions being found so early in life (Waldron and Rogers, 1995). The second factor was the presence of the skeletal elements needed for assessments: lumbar vertebrae, pelves, femora, patellae, and tibiae (see Figure 5.1 and Figure 5.2). The joints of these bones have been chosen due to their relationship with locomotion and load bearing.

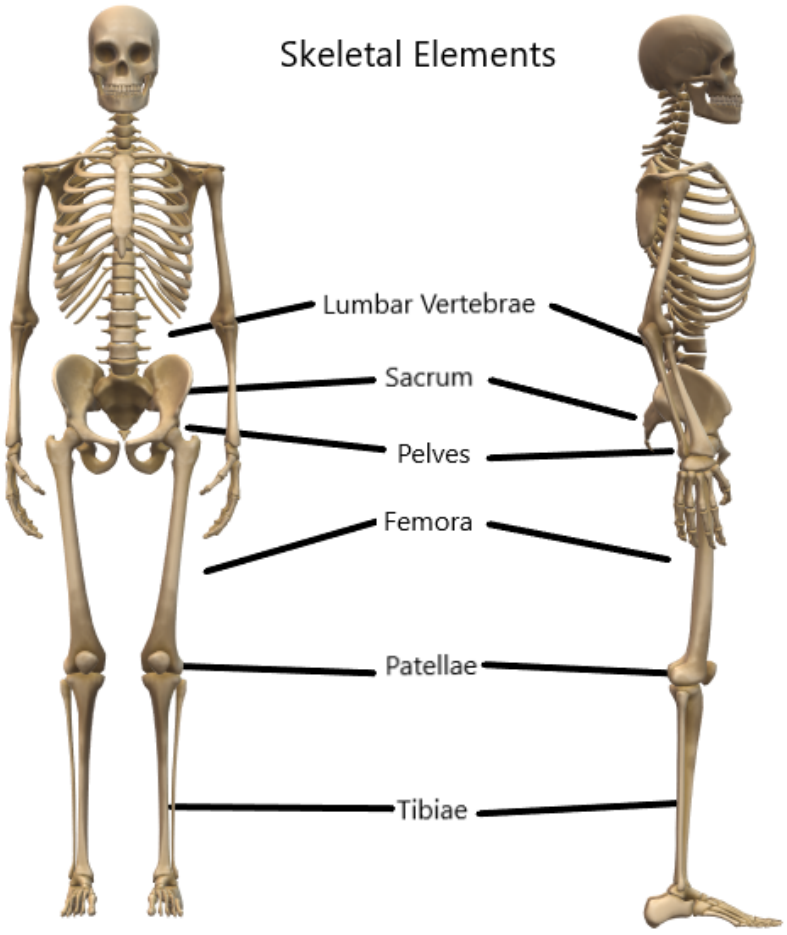

Figure 5.1 The skeletal elements assessed during this thesis for joint condition.

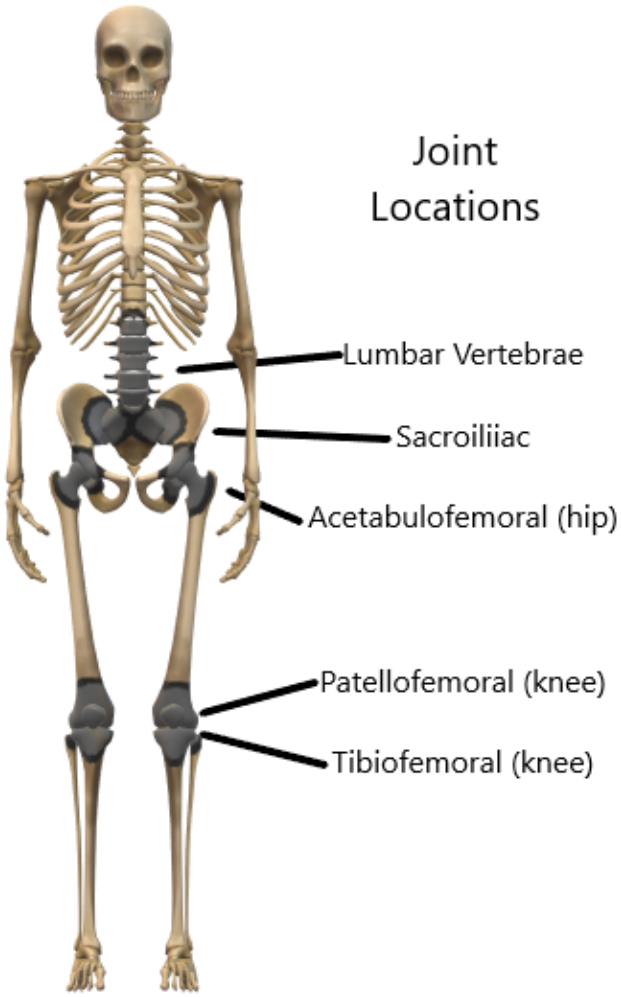

Figure 5.2 The joint locations that were assessed during this thesis for joint condition.

5.2 Sample Collections

A total of 187 individuals, selected from skeletal collections representing English post-medieval populations (c. 18- 19th centuries CE), were able to be used for this research (see Table 5.1 and Figure 5.3). The post-medieval period was chosen to research joint conditions due to the massive adjustments in the society as technology and daily life change substantially throughout this era. Five of the samples are from urban populations and the sixth, Barton-upon-Humber, was a small rural village. The maintenance of a distinction between urban and rural populations should help to distinguish between the different habitual activity and lifestyle patterns of the populations. Moreover, selecting collections from the same country and from a similar time periods helps to increase the chance for similar habits, technology, and cultural practices across the collections. These similarities will create a more pseudo-homogenous population type with decreased chance of an external factor affecting the data or results,

allowing for the internal risk factors to be assessed more accurately. The sites have been numbered for data analysis with sites 1-5 being urban and site 6 being rural.

Due to poor preservation of some skeletal remains, not all skeletons were complete or well preserved for in-depth assessments. Skeletons may have been initially utilized and been part of the skeletal assessments to bolster sample size, but when doing the further prevalence and statistical assessments, the increased samples did not also provide an increase to the data. Therefore, those samples were not included within the results sections of this research.

Table 5.1 The skeletal collections included in this thesis with location, sample size and curatorial information.

SITE #	SITE NAME	LOCATION	SAMPLE SIZE	CURATING INSTITUTION
1	CHURCH OF ST HILDA, CORONATION STREET	SOUTH SHIELDS, NEWCASTLE UPON TYNE	46	UNIVERSITY OF SHEFFIELD
2	CARVER STREET METHODIST CHURCH	SHEFFIELD	4	UNIVERSITY OF SHEFFIELD
3	ST PETER'S COLLEGIATE CHURCH	WOLVERHAMPTON	20	UNIVERSITY OF BRADFORD
4	QUAKER BURIAL GROUND	KINGSTON UPON THAMES	24	BOURNEMOUTH UNIVERSITY
5	ST AUGUSTINE THE LESS	BRISTOL	2	BOURNEMOUTH UNIVERSITY
6	ST PETER'S CHURCH	BARTON UPON HUMBER	91	ENGLISH HERITAGE

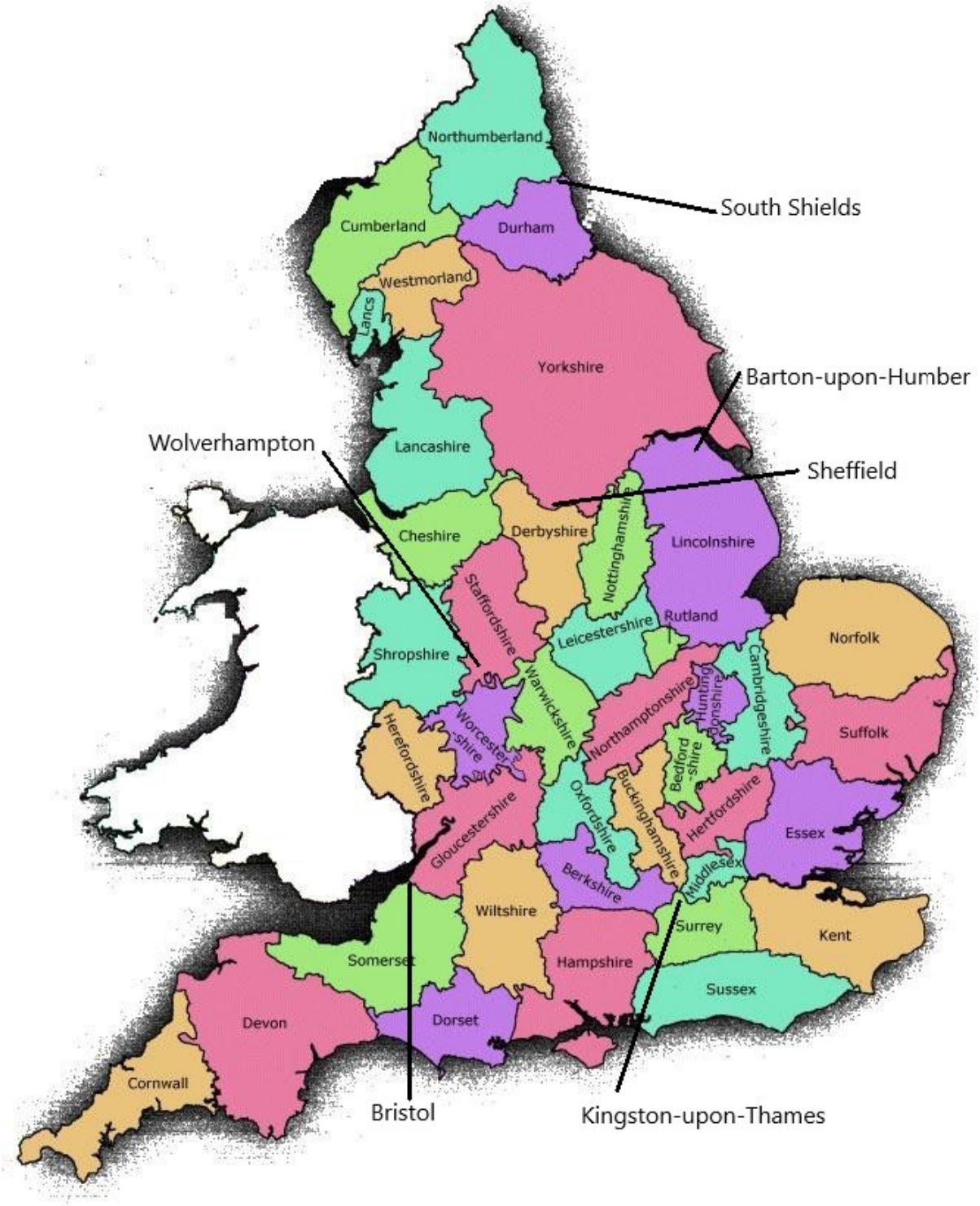

Figure 5.3 The site locations with the historic counties of England.

5.3. Post-Medieval England

The post-medieval period in England covers the span of history from the 16th to the late 19th or early 20th centuries CE, ending with the start of the early modern period. This period of history is broken into two sub periods with the early post-medieval period covering roughly the 16th and early 17th centuries CE and the late post-medieval period covering the late 18th century CE to the end of the post-medieval period (Jenkins, 2018). This period is significant as a result of two major events: the industrial and agricultural revolutions.

The industrial revolution was a period of unprecedented economic and technological expansion, giving rise to the steam engine, railroads, and the factory, enabling mass production and availability of goods to locals as well as to international markets (Hey, 2009; Wrigley, 2016). People of lower socioeconomic status were no longer as confined to the geographical areas of their birth and could access greater opportunities to leave to find work or settle down for family life (Barker and Hughes, 2020). During the agricultural revolution, new technologies and techniques helped turn simple plot farming into an industry unto itself (Bujak, 2007). Larger fields were able to be utilized, producing goods for not just the family and locals, but that could be sold wholesale to external markets (Allen, 1999). The increase to food production would mean greater stores in times of famine or hardship and allow for a greater variety of food stuffs to people that may not have had access before.

These significant events that characterized the post-medieval period in England occasioned widespread change in many people's social and working lives, impacting both urban and rural communities. In the following section, some of the major events of the Industrial Revolution are introduced in order to offer a historical context for the skeletal assemblages studied in this thesis. In light of the opportunity for this study to examine a combination of rural and urban individuals, this discussion focuses on revolutions in both industry and agriculture.

5.3.1 Industrial Revolution

The steam engine was one of the most famous technological marvels to come out of the industrial revolution in 1712 with the Newcomen atmospheric engine (Allen, 2009). This engine was revolutionary but inefficient in fuel and power output and over the next 150 years

engineers sought to improve on the design. The steam engine used a coal furnace to boil water which moved internal components creating power that could be harnessed (Nuvolari *et al.,* 2011). The steam engine was important not just for the railroad but for altering the effectiveness of waterborne vessels and powering industrial machinery, such as water pumps, looms and assisting water wheels. Seafaring vessels of the time relied on wind and sail, which worked effectively, but was not always reliable if the winds were to die for any length of time. Not only could the ships now travel more reliably on the open ocean, but they could also travel faster, allowing for more goods and people to be transported over a shorter period of time.

River travel was growing, as the steam engine allowed for ships to move more smoothly upriver, against the current. Canals were dug and extended to allow for the transportation of goods from one location to another without clogging the rivers with traffic, such as the burgeoning steel industry in Sheffield (Hey, 2009; Wrigley, 2016). The canals had a consistent depth and width, making easing navigation through these section with less fear of bottoming the ship on a sandbank or rocky outcropping. These canals became the highways of the era and the cargo ships were the lorries.

The steam engine was also used on land and gave us an invention that is very much in use today: the railroad. The early iterations of the railroad were slow and clumsy, needing constant maintenance, but allowed for the hauling of cargo that would otherwise have needed to be carried by multiple horse and donkey drawn carts (Wrigley, 2016). A technological race to create a better engine quickly ensued and quickly newer variants were coming into service that could haul more tonnage at faster speeds and to further locations (Nuvolari *et al.,* 2011). However, the drawback of the railroad was some of the features that make England so beautiful: the numerous rolling hills and valleys. The early trains had difficulty travelling uphill while towing cargo, which meant that tunnels would need to be dug and bridges built to accommodate this issue (Gregory and Hanneberg, 2010).

Rail lines spread throughout the country acting like veins, starting with travel from a coal mine to a factory and by 1876 connected the entire country (Gregory and Henneberg, 2010), bringing an influx of workers and goods to areas that were lacking and uniting areas of England with Great Britain that had may have had little to no contact in previous centuries (Joyce, 2007). Now trade goods, such as coal, could be mined/created in one part of the country and be available in other parts of the countries within the week, far quicker than at any other point in history thus far (Wrigley, 1967). Goods no longer had to be produced locally and goods

from Europe and further foreign markets could be found more readily available, when once these goods were considered a luxury (Hey, 2009). Port towns grew up to accommodate this influx of external trade, such as Bristol and ports on the Humber nearby to Barton-upon-Humber, which then imported the goods to the rest of the country via these new rail systems.

As travel by railway became more popular and routine, individuals could use it to travel for leisure as well as work. By the 1820s, the travel times between Manchester and London would fall from 16 hours for mail and 30 hours for passengers to 5 hours and 40 minutes and by 1910 would decrease further to 3 and a half hours (Gregory and Henneberg, 2010). This would lead to the expansion of cities as individuals travelled in from the countryside in search of work, and more towns began appearing around each new industrial center (Wrigley 2016). In the case of Sheffield, an 1851 census shows that 36.3% of individuals living in the city had immigrated from other parts other areas (Hey, 2009). Overcrowding in cities and poor sanitary conditions would have affected the health of the individuals living there, as evidenced by the higher prevalence of cribra orbitalia in a sample population from York and an increase in poor health in Wolverhampton (Joyce, 2007; Watts, 2013; Western and Bekvalac, 2020).

With the importance of the steam engine expanding, coal mines expanded and become essential to the management of these new transport systems. Urbanization began as small towns and cities began grow and develop industry relating to the railroad, creating a boon to the economy, as seen by the coal mining at South Shields (Joyce, 2007; Hey, 2009; Wrigley, 2016). This wealth of industry allowed England to grow and prosper and maintain its status as a power in the world (Crouzet, 1967). The birth of factories was both a boon and curse on the individuals living during this time. Factories allowed for the mass production of goods that had previously requires years of mastery as a tradesman to be able to create (Nuvolari *et al.,* 2011). The rich and poor alike could share in the goods that had previously belonged in the domains of the elite and wealthy. Textile and cutlery factories were two of the many types of factories that arose during this time, allowing for fabrics to be created more swiftly using powered looms and new styles of clothing to be available to all classes and allow for different forms of cutlery to become more common to the average individual (Hey, 2009; Nuvolari *et al.,* 2011). Factories producing cutlery from the local steel works helped turn Sheffield from a small semi-industrial town into a large, prosperous city that continued into the present.

However, the factories had a darker side that has given rise to many of the laws and procedures in use today. When the children reached adolescence, they could begin to assist

with jobs outside the household to help out the family, such as gathering wood for fires, working in the fields to help harvest or working in factories, which had long hours and miserable conditions (Heaton, 1967; Houlbrooke, 1984; Barker and Hughes, 2020). However, it has been noted that children would begin doing any work that could assist their parents while still within the household, such as weaving wool (Horrell and Humphries, 1995). Small children were often used for the operation of delicate, yet still dangerous, machinery due to their small stature and petite hands (Horrell and Humphries, 1995). While this allowed for an increase to the family's income, children were abused in the system as workhouses for the poor were developed as a prelude to modern day orphanages and children, as well as women, were utilized as little more than slave labour (Orsi, 2017). This may mean that child labour was not a new phenomenon but may have simply been a more visible one. These conditions were not the sole issue of children, and women and men alike were working in dark, dirty, cramped and often dangerous conditions. Any debilitating injury, such as the loss of a limb, meant the loss of income and job for the individual, and due to the medical conditions of the time, may also have meant a loss of life (Hey, 2009).

5.3.2 The Agricultural Revolution

The timing of the agricultural revolution is contentious, and it is unclear as to which revolution came first, if one helped precipitate the other, or if they simply developed concurrently (Allen, 1999). Agriculture was largely affected by the industrial revolution as new technology was developed and employed which allowed for greater yields using less material and land, making farming more efficient and profitable. Farmlands were able to expand as larger areas were able to be cultivated, producing more goods, up to 3.5 times that of the previous eras, that could be sold at markets outside of the immediate vicinity (Williamson, 1998).

At the start of this new agricultural revolution the techniques and processes were labour intensive requiring considerable help from hired hands (Mingay, 1991). While these methods improved crop output, the physical input required on the fields as also increased. Newer technologies were ignored, such as seed drills and new drawn horse hoes/plows were ignored or neglected. It was thought that the advantages of using such new tools were too small and only helped increase seed output and land arability while increasing labour input. It was not

until the late 18th century CE that advances in mechanical technologies, such as the steam driven thresher or mechanical reaper, began to be seen in the agricultural world. The newer mechanical technology not only increased outputs but also decreased the human labour involved in the process.

Land usage for agriculture had expanded, with newer and larger farms being created using the new processes being developed. Advancements in irrigation began to allow for water to be circulated among fields more efficiency, thereby increasing yield and productivity. The process of floating a field, a process which was not new, but becoming more common place, was believed to have become popular sometime in the late 16th century CE (Williamson, 1998). Floating was the process irrigation by-which a leat was used to feed water down a series of channels down the sides of a hill into a valley. Sluices could be used to allow for the build-up and release of water, or to direct the flow of water to a specific area. Marshlands and other natural features that were otherwise unsuitable to inhabit were being utilized, via new drainage methods, to expand farms, hunt, and fish, and for expansion of grazing enclosures (Barber and Pelling, 2019). This expansion of land into otherwise unusable or barely usable lands meant for greater areas of cultivation and expansion of rural or semi-urban towns with increased housing. In Barton-upon-Humber, these new practices allowed the town to flourish and with the additional factories along the Humber brought in fresh workers and goods.

Crop rotation was improved significantly during this period, starting in the east of England, and spreading to the rest of the country. Cereal grains were combined with newly introduced crops, such as radishes and artificial grasses (Mingay, 1991). This allowed for the artificial grasses to replace the nitrogen in the soil via atmospheric transference with the need to only leave the field fallow for a single year, rather than 2 as was the traditional rotation practice (Williamson, 1998).

The revolution in the practices and output of this time did not have the unstoppable force that was comparable to the industrial revolution and a period of stagnation occurred in the late 18th century CE (Allen, 1999). This stagnation did not last long, but food consumption per family dropped and a surge of production began around the start of the 19th century CE. While many agricultural revolutions have occurred over the course of human history, the first of which allowed humans to settle into villages, this revolution developed many of the techniques and technology that, while more refined, is still in used today.

5.4 The Sample Sites

The sites chosen for this research represent populations from a group of industrial centers spread throughout post-medieval England. England has had a long and continuous period of occupation, providing a rich and unique history that has been well documented and studied. This would make English populations a valuable resource to study to better understand how joint condition affects not just the populations at large, but also create a timeline for the conditions across multiple English historical periods when compared with similar studies. While the cemeteries may have been in continual use for centuries, this research focuses only on the latter burials that fall into the post-medieval period of English history.

5.4.1 Church of St Hilda, Coronation Street, South Shields, Newcastle-upon-Tyne

Named after St. Hilda of Whitby, an important figure who helped to convert England to Christianity in the 7th century CE, the Church of St Hilda is a parish located within the town of South Shields. The town was located roughly 5 miles east of Newcastle-upon-Tyne, on the southern shore of the River Tyne, and by the 19th century CE, the town had reclaimed its historic lands along the banks of the Mill Dam, which the early town had grown away from (Raynor *et al.,* 2011; Archaeological Services Durham University, 2006). During the 19th century CE, South Shields had grown into a city with industries focusing on coal mining and salt-panning (English Heritage, 2004; Archaeological Services Durham University, 2006).

After the medieval period, the landscape around the town changed, with build-up from the dumping of ships' ballast and industrial ash waste and by around 1816 CE Mill Dam was infilled. Concurrent with the change in landscape, the population of the town increased, however, St Hilda's remained the sole church for the town and was expanded to adapt in 1753 and 1786. The booming population left the church yard overcrowded with burials and the threat of disease from the unsanitary conditions forced the council to investigate alternative options for the burial of the dead (Raynor *et al.*, 2011).

The church of St Hilda is believed to be on the site of an earlier chapel, potentially dating to the 7th century CE, however the cemetery from which the skeletal remains studied in this thesis are derived from is post-medieval internments, which was expanded from the earlier medieval cemetery sometime after 1631 CE (Raynor *et al.,* 2011). By the early 19th century

CE, the church yard had become overcrowded with graves and work began on expanding the grounds (Archaeological Services Durham University, 2006). The graveyard was officially closed on 1 July 1855 CE, with only exceptional burials allowed until 1860 (Raynor *et al.*, 2011).

Excavation of the cemetery took place in 2006 and more than 188 burial plots were uncovered containing both articulated skeletons and charnel deposits (Raynor *et al.,* 2011). The population consisted of 114 adults, 3 adolescents and 87 sub-adults, with a slightly higher percentage of females to males. Overall, the adult skeletons were well preserved and are thought to be those of the laboring class.

5.4.2 Carver Street, Methodist Chapel, Sheffield

Sheffield is a city located in South Yorkshire near the confluence of the River Don and River Sheaf. It has seen varied occupation with physical evidence dating back as far as the Bronze Age (Hey, 2007), however the majority of archaeological evidence for occupation of the area dates to the medieval and post-medieval periods. The earliest documented accounts of industry in the area, from the Sheffield manorial accounts of the mid 15^{h} century CE, show evidence for the production of coal and charcoal, nevertheless there is archaeological evidence for charcoal burning much earlier, during the Roman period in nearby Templeborough. Following the Norman Conquest, a castle was built on a hill overlooking the town that, centuries later, the future Mary, Queen of the Scots, would spend a significant portion of her imprisonment (Moreland and Hadley, 2020).

The first Methodists arrived in Sheffield in 1738 CE. However, Methodists were viewed as radical troublemakers and were not welcomed in the area, and so the first church was destroyed by rioters during a sermon in 1743 CE (McIntyre and Willmott, 2003; Price 2008). The Methodist Chapel located at Carver Street was opened in 1805 with an attached cemetery open to burials for roughly half a century between 1806 – 1855 CE. W. Jenkins, the first minister, designed the chapel for simplicity over beauty and by 1851 CE the chapel had a congregation of 766, with roughly 1600 inhumations in the adjoining graveyard (McIntyre and Willmott, 2003; Mahoney-Swales *et al.,* 2011). The cemetery was no longer able to be utilized at the location within the city after the passage of the Burial Grounds Act of 1857, and burial ceased from this point.

The population of Sheffield increased from roughly 10,000 in the mid-18th century CE to roughly 45,000 at the turn of the 19th century CE due to an influx of new people to support growing and diversifying industries (Sheffield City Council, 2006). By 1820 CE, the canal was completed, and the cutlery and steel industries were developing and became key industries that would help build Sheffield in a richer city. Sheffield was a heavily industrialized city, and in the 17th century CE an estimated 1 in 5 men worked in one of the many cutlery trades and there was a smithy to over 2.2 households (Hey, 2007). To assist with the population boom, new schools and centers for learning were built, along with public hospitals and parks, such as the extant Botanical Gardens.

An excavation of the burial grounds in 1999 revealed 47 graves with 101 articulated skeletons and roughly 30 disarticulated remains (McIntyre and Willmott, 2003). The population of the cemetery is believed to be that of skilled laborer's, consistent with the population of Sheffield at the time. During examinations, half of the skeletons were unable to be reliably sexed with the remaining half being almost split between males and females. 37 of the skeletons were found to be immature, four of which were neonates, and the final 99 were determined to be adult. The remains from this collection were of mixed preservation with missing elements being common.

5.4.3 St Peter's Collegiate Church, Wolverhampton

Wolverhampton, formerly Wulfruna and Wulfruna's Heanton, is a city located in the County of West Midlands, formerly the County Staffordshire, founded in the late 10th century CE. During the mid-19th century CE, Wolverhampton was heavily influenced by the industrial revolution, prospering due to the local industries and new railway system in the area. These industries included coal mining, iron, steel, japanning, Bilston enamels, and ceramics, making the city of Wolverhampton a hub for industry within the Midlands (Joyce, 2007).

St Peter's Collegiate Church, located within the northern portion of the city of Wolverhampton, is an active church whose founding date is unknown but believed to be towards the end of the Anglo-Saxon period in the 11th century CE. The current structure of the church dates to the 15th century CE.

Between 2001 and 2002 CE an overflow cemetery was excavated by the Birmingham University Field Excavation Unit and 157 individuals dating to the mid-19th century CE were excavated (Adams and Colls, 2007). As the remaining individuals left for exhumation by the archaeological team were located within the church, it could be assumed then that these individuals were well to do within the city. The remains from this collection were typically well preserved, however, missing elements was not uncommon.

5.4.4 Quaker Burial Ground, Kingston upon Thames

Kingston-upon-Thames is a town currently located at the south-west extent of Greater London. Evidence for the earliest occupation of the area occurred during the Romano-British period, however, the earliest known development took place under the Anglo-Saxons. The Anglo-Saxons named the town Cyninges tun in 835 CE and turned it into the earliest known royal boroughs for the Anglo-Saxon kings. As a royal borough, the industry of the area was based around the needs of providing for the members of the royal families and the upper-class members of the court (Malden, 1911). The area was originally used for agriculture, as evidenced by the orchards used later by the Quakers for burial, as well as for stud farms to produce quality mounts for the nobles. As the town grew into a city around the 16th century CE, the city had multiple markets and fairs, as well as trading companies.

The Quaker Burial Ground is located on what was the fringes of the medieval town at the foot of the Kingston Hill, later incorporated into the town of Kingston-upon-Thames (Bashford and Sibun, 2007). A Society of Friends (Quakers) community has existed in the area from the mid-17th century CE with the first burial, that of Ann Stevens, recorded on 26 June 1664 CE. The Burial Ground was an orchard, in accordance with Quaker traditional of burials taking place away from the traditional Christian church yards. By the late 19th century CE, the majority of the grounds had been covered over by a Quaker chapel, porch, and glass houses with only a third of the land remaining open. While the Quakers recorded the events of the community, including births and deaths, it should be noted that the records are incomplete and partial, however, due to the records, these burials included named individuals.

The excavation at the site took place in 1996 and 364 individual burials were identified (Bashford and Sibun, 2007). However, despite the individual burials and the coffin materials, only 39 were established to be well preserved with many of the remains being fragmentary.

Individuals determined to be adult by osteological samples were the majority, with more females than males in the burials. The Quakers of this area are believed to have been in the middle to upper classes and represented many of the local merchants.

The Quaker Burial Ground, Kingston upon Thames offered a unique perspective for the research as there were cases of named individuals with known age at death and biological sex. When assessing for biological sex or age at death there is a degree of error and misinterpretation, especially concerning age at death, and so these individuals allowed for more accuracy. The names of the individuals were not used within this study. The remains from this collection were generally well preserved, however missing elements were not uncommon.

5.4.5 Church of St Augustine the Less, Bristol

Bristol is a city located in south-west England between Somerset and Gloucestershire and was established sometime in the 11th century CE (Boore, 1986). The city included a port with trade ties to western Europe and was notable as being a key port for the English participation in the slave trade (Dresser, 2009). The city had an abbey, now the cathedral and an adjacent church, both believed to be named after the Roman missionary St Augustine, the Apostle of the English, first archbishop of Canterbury, however as the order was Augustinian founded by St Augustine of Hippo, there is a question as to which St Augustine is the actual namesake (Higgins, 2012).

The adjacent church, known as St Augustine the Less, built along the River Frome, was thought to have existed before the construction of the abbey and may have been a parochial church known to have existed in that area by the late 13th century CE. Another theory is that the church was built as a temporary chapel for the religious order as the abbey was under construction (Boore, 1986). The church had fallen in disrepair by the 15th century CE, was rebuilt in 1480 CE and further extensive repairs had taken place in 18th century CE.

The earliest burials found on the site date to the 11th century CE. The site had a larger than normal number of burials during the first half of the 17th century CE, due to a local pestilence (Boore, 1985). During the mid to late 19th century CE, burials within the church yard were being exhumed and moved to make way for contemporary urban development, halting the use of the cemetery. St Augustine the Less was demolished in 1962 CE and excavated

1983-84 CE (Boore, 1986), uncovering a total of 136 stratified burials from within the church and surrounding yard. The post-medieval period saw the building of bricked vaults containing remains of coffins, 107 of the vaults were found within the church itself. This expenditure would not have been cheap, and therefore it would be assumed that the inhabitants would have been more affluent. Many of the burials within the church had been cut through or exhumed as later burials were deposited, which lead to the comingling of individuals.

5.4.6 St. Peter's Church, Barton-upon-Humber

St. Peter's Church is located within the town of Barton-upon-Humber, North Lincolnshire, located on the southern banks of the River Humber. There is evidence of human activity at the site pre-Roman times, however, there is no evidence for significant occupation of the site, such as developed later, prior to the Early Anglo-Saxon period (Waldron 2007). The town consisted of a small port with a ferry across the Humber and the town initially prospered (Watts, 2013). By the close of the medieval period, economic growth had stagnated, and the town was considered to be poor by contemporary standards with children, as young as 10 years of age, being sent off to work in the fields for extra income (Houlbrooke, 1986; Clapson, 2005). The economic growth of the village experienced a revival when in the 17th century CE factories were built along the river, shifting the workforce from the maritime/agriculture into the factories (Bryant, 2003). Isotopic analysis found the food sources to be varied from fish, meat, vegetables, and other marine resources (Beavan *et al.,* 2011).

St Peter's Church was established in the Anglo-Saxon period (Waldron, 2007) as a simple square tower. Over the century the church experienced continued expansion, adding new structural pieces to enlarge the space. This expansion lasted until the late 19th century CE when the current structure was finished and a newer and larger church, on an adjacent plot of land, became the dominant place of worship within the town. The final burial within the church was in 1844 and the final burial in the church yard, which had been expanded to the south in 1850, took place in 1855 (Rodwell and Atkins, 2011).

Excavations of the church and churchyard took place between 1978 and 1984. Roughly 2800 articulated burials were excavated from within the church and the churchyard (Waldron, 2007). A total of 484 burials were in the church, however, many of which were not originally intramural but had subsequently had the church extended over them. The contemporary

registrars of the church state that the individuals buried exemplified the more affluent members of society with the elite upper class and middle class being present. These burials were found within 5 phases representing the differing periods of time the church was in use with an additional 4 phases covering both adjacent periods (i.e., Phase A/B, B/C) between the time periods. 427 burials were found to be in Phase A covering 1700-1855, which was divided evenly between males and females. Age is not listed by period but roughly 1800 of the total assemblage was considered to be adult. The skeletal samples within this collection were, for the most part, well preserved with few elements missing for assessment.

5.5 Summary

The joint locations selected for this research represent joints that are load bearing from the lower spine and lower appendicular skeleton. The post-medieval period in England saw major upheaval as new technologies and practices changed the lives of the citizenry from the highest to the lowest stations. The changes should, therefore, be evident on the skeleton in the form of joint conditions and using the sites discussed above, provide a good cross section of the individuals from the time over differing occupation types and social strata.

Chapter 6 Methodology

The methods used within this body of research have two main components: (1) skeletal assessments and (2) statistical analysis. First, skeletal examinations were completed to generate an original dataset tailored to the aims of the present project. This required the skeletal samples to be present in either a laboratory setting, or as in the case of Barton-upon-Humber, in the storage location of the materials. The skeletal assessments included determining the biological sex and age at death of the samples, recording measurements for the assessments of height, mass, and cross-sectional geometry, assessment of entheses related to the joints, and palaeopathological assessments. The palaeopathological assessments included appraisal of the joints for the markers of joint disease and the entire skeleton for any potential differential diagnoses and to attempt to find any insult to the skeletal material that could cause secondary cases of the joint conditions. The statistical testing was completed using IBM SPSS Statistics software v.23-26. and included tests for association/correlation, measuring asymmetry, binomial logistic regression and determining odds ratios.

Initially, osteological methods were used to create a demographic profile of the sample populations to determine distribution of biological sex and age at death across the entire population, as well as between the individual sites and site categories. With these demographic profiles, prevalence of the joint conditions can be assessed to help determine any trends. The methods used to create the metric variables for height, skeletal mass, and cross-sectional geometry, discussed in detail further in this chapter, will help to assess how activity and body mass affect the prevalence of the joint conditions. The combination of these methods should create a detailed profile to help identify and further understand the complex relationships between skeletally assessable risk factors and joint conditions.

6.1 Skeletal Assessments

The skeletal assessments took place in a laboratory environment, except for Barton-upon-Humber which took place within the onsite bone storage in the church. Standard assessments of demographic variables were first completed, recording biological sex and age at death. Biological sex is important to conduct first, as later assessments are dependent on knowing the sex of the individual. A variety of measurements of the long bones from the lower

limb of the skeletal samples were recorded to calculate skeletal stature, body mass, body mass index and cross-sectional indices. These variables are split between the demographic information that is independent (age at death, biological sex and site category) and not potentially caused by something else and the dependent variables (BMI, EC, cross-sectional indices) that can be influenced and caused by the independent variables.

The final portion of the assessment entailed analyzing the skeletal material for markers and features that would allow for differential diagnosis of the joint conditions, as well as identify any other pathological conditions that may be present that could affect the onset of the joint conditions or cause secondary cases. These pathological conditions can include, but are not limited to, DISH, scurvy, rickets, and trauma. If these pathological conditions were present, the sample skeleton was deemed to be unreliable for use within this research and was then excluded.

6.1.1 Assessment of Biological Sex and Age at Death

Data concerning age at death and biological sex of the sample skeletons were necessary to characterize the demographic profile of the sample and for the exploration of age and sex-specific characteristics of joint condition manifestation. A few of the collections utilized had pre-recorded ages and biological sexes produced using up-to-date methods, and therefore did not require this analysis to be repeated. These collections included Wolverhampton, Kingston-upon-Thames, and Barton-upon-Humber. The collections that did not have accurate or up-to-date age at death or biological sex assessments were reassessed by the author using the various methods listed below, with different methods used depending on the presence of the skeletal elements necessary.

6.1.1.1 Biological Sex Assessment

Skeletal morphology is an accurate, reliable, and repeatable method for determining the biological sex of a skeleton (Ferembach *et al.,* 1980; Buikstra and Ubelaker, 1994; Walker, 2008; Klales *et al.,* 2012). Assessing biological sex of an individual based on pelvic methods has shown to be 90-98% accurate depending on the individual method, however skull-based assessments using either the cranium or the mandible very in accuracy (roughly 80% but as

high as 92%) and are highly dependent on the skill and experience of the researcher (White and Folkiens, 2005; Klales *et al.*, 2012). Methods based on morphological traits use different skeletal elements (e.g., cranium, mandible, and pelvis) and established morphological differences associated with each element, to provide an estimate of biological sex. These methods include: Ferembach and colleagues (1980), shape of the obturator foramen and width of lateral ischium; Buikstra and Ubelaker (1994), morphological traits of the skull and pelvis; Walker (2008), a variation of Buikstra and Ubelaker's (1994) method using the scores of the skull morphologies to calculate potential biological sex; and Klales and colleagues (2012): revision of Phenice (1969) pubic region assessments using scored markers to calculate a potential biological sex. Within this body of research, all these methods were utilized, and applied to every skeleton where the relevant skeletal elements or features were present and observable.

A single element used for the assessment of biological sex has a margin of error for accuracy and reliability, but with multiple methods used for the determination, that margin of error is reduced (Meindl *et al.,* 1985). Due to human variation, it is not always possible to determine a definitive biological sex, and as can be seen with living individuals, some skeletons are more gracile or robust, which can cause a genetic female to have more male traits in their skeleton, or vice versa. This leads to five potential biological sex categories: probable female, possible female, indeterminate, possible male, probable male. Assessed remains that fell within the indeterminate or were unable to be assigned a sex during this research were omitted from the sample, as these would not provide any useful information towards the research questions.

6.1.1.2 Age at Death Assessment

Age at death in adult skeletons is primarily estimated by the assessment of degenerative changes at key immobile joints throughout the skeleton, however in early adults, which have not completed skeletal maturation, stages of growth are assessed. To estimate age at death, assessments for biological sex must first be completed due to a difference in the degenerative variation that can occur between a male and female for elements such as the pubic symphysis (Brooks and Suchey, 1990). Age at death in skeletal remains from mature individuals can be assessed through examination of degeneration of the immobile joints of the pelvis, fusion stages of the cranial sutures, as well as dental eruption (Meindl and Lovejoy, 1985; Webb and

Suchey, 1985; AlQahtani *et al.,* 2010). Although the skeletons assessed in this project comprised individuals aged c. 18 years and over, a range of age assessment methods based on the last stages of skeletal maturation could still be used. As the skeleton does not fully mature until c. 25-30 years of age, in younger adults, fusion of epiphyseal elements to the diaphyseal elements can be applied (Webb and Suchey 1985; Scheuer and Black 2000; Scheuer *et al.,* 2008). Moreover, teeth will erupt in a predictable pattern, and by noting which teeth have erupted, the level of eruption and which teeth have not erupted, it would be possible to predict an age range for individuals that are largely under the age of 18 (Ubelaker, 1989; AlQahtani *et al.*, 2010). The final 3rd molars should begin to erupt and settle in place between the ages of 16-23 (AlQahtani *et al.,* 2010, and when used in conjunction with the other age at death methodologies, it can help pinpoint a specific early age range.

It is impossible with currently available methods of age assessment in adults, to accurately narrow down an assessed range to a specific age, and so age ranges are typically used (Brooks and Suchey, 1990; Buckberry and Chamberlain, 2002). In this study, ADBOU v2.1 software (Milner and Boldsen 2002) was utilized for the age at death assessment. This program combines data derived from the methods for assessing cranial sutures, the auricular surface and the pubic symphysis into a computer program that calculates age ranges, the lower and upper 95% confidence intervals and maximum likelihood age of each feature, as well as an age range based on all of the methods combined. The software uses Bayesian statistics and transitional analysis to create a more accurate age range those traditional methods of age assessment, by creating a probability model based on known individuals. When tested against individuals with known ages, a strong correlation index was found ($p = .9$) between the age estimates and the known chronological ages and exhibited an R^2 value of .82, displaying a high level of explanation to the variation of the data (Lopez-Cerquera and Casella, 2018).

Within this software is the ability to choose the potential biological sex, mortality model of the assessment (archaeological or forensic) and simple ancestry (white, black, or unknown). For each of the three methods – cranial suture closure, pubic symphyseal wear, and auricular surface wear – the user can score the feature using a defined scoring method that includes the varying stages of wear or degeneration (see below). The skeletal material may not fit a single score and, unlike in the traditional means of interpreting these methods, the software allows the input for a range of scores (i.e., instead of just a score 3, the user can put 3-4). When input of the data is finished, the software uses Bayesian analysis to provide the user with the potential

age ranges for each method, as well as an overreaching range with both corrected (using Bayesian equations) and uncorrected (based on raw scores) values using Bayesian statistics. The user can also choose the levels of probability for the ranges (99%, 95% or 90%). For this research, 95% probability was used. (see Figure 6.1 and Figure 6.2).

With most methods of age assessment, such as Suchey and Brooks' pubic symphysis method (1990) or Buckberry and Chamberlain's auricular surface methods (2002), damage to the assessed element may cause the methods to become invalid or unusable. Whereas these methods may not produce a valid age range if missing even a single score, which is a possibility when examining samples that are not well preserved, the ADBOU software will then rely on the Bayesian statistics and transition analysis to complete the analysis (Boldsen *et al.*, 2002; Milner and Boldsen, 2012). For instance, five scores are necessary to accurately score using traditional cranial suture methods, but with this software, it will take in as little as one suture score and compute an age range, though, as might be expected, fewer input variables decrease the accuracy of the final output. Importantly, any decrease in accuracy resulting from substitution is shown in the age ranges calculated, thus precision of the final age generated is explicit (Milner and Boldsen, 2012).

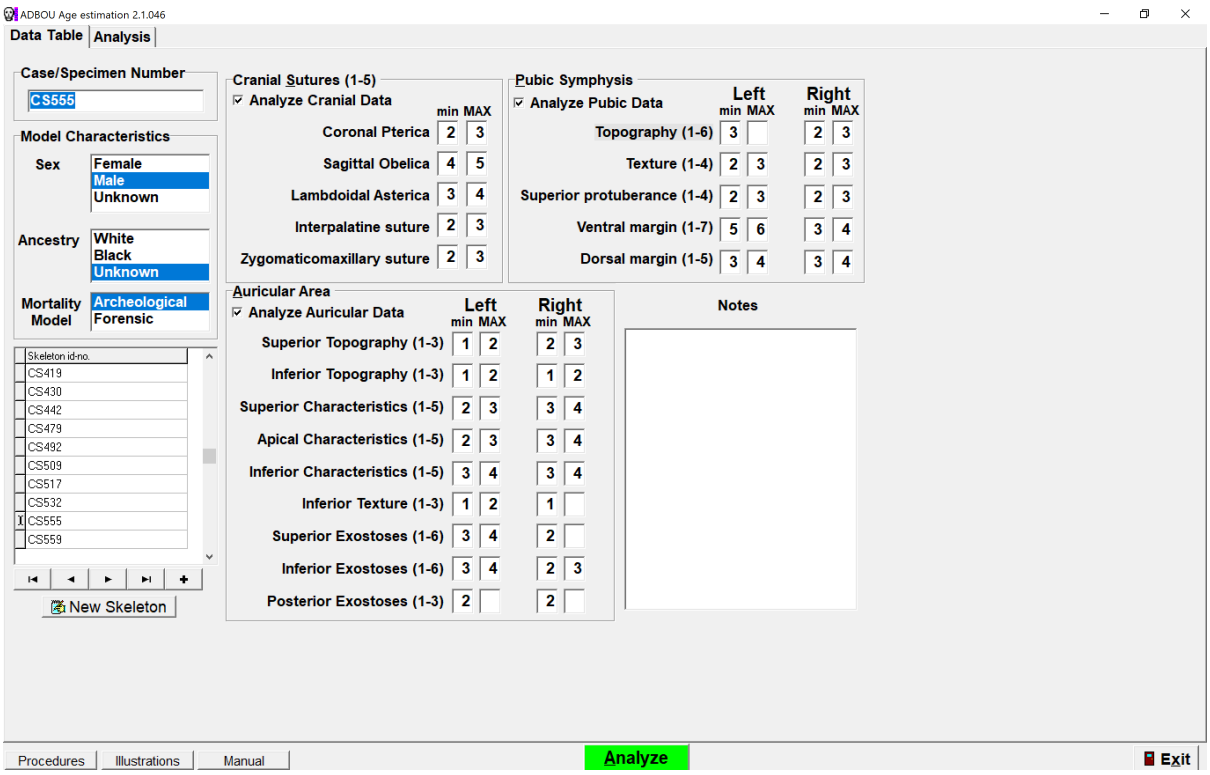

Figure 6.1 Example of scoring elements using the ADBOU software on South Shields skeleton CS555. Note the different input boxes that allow for a range of scores, as well as the ability to record notes for later use.

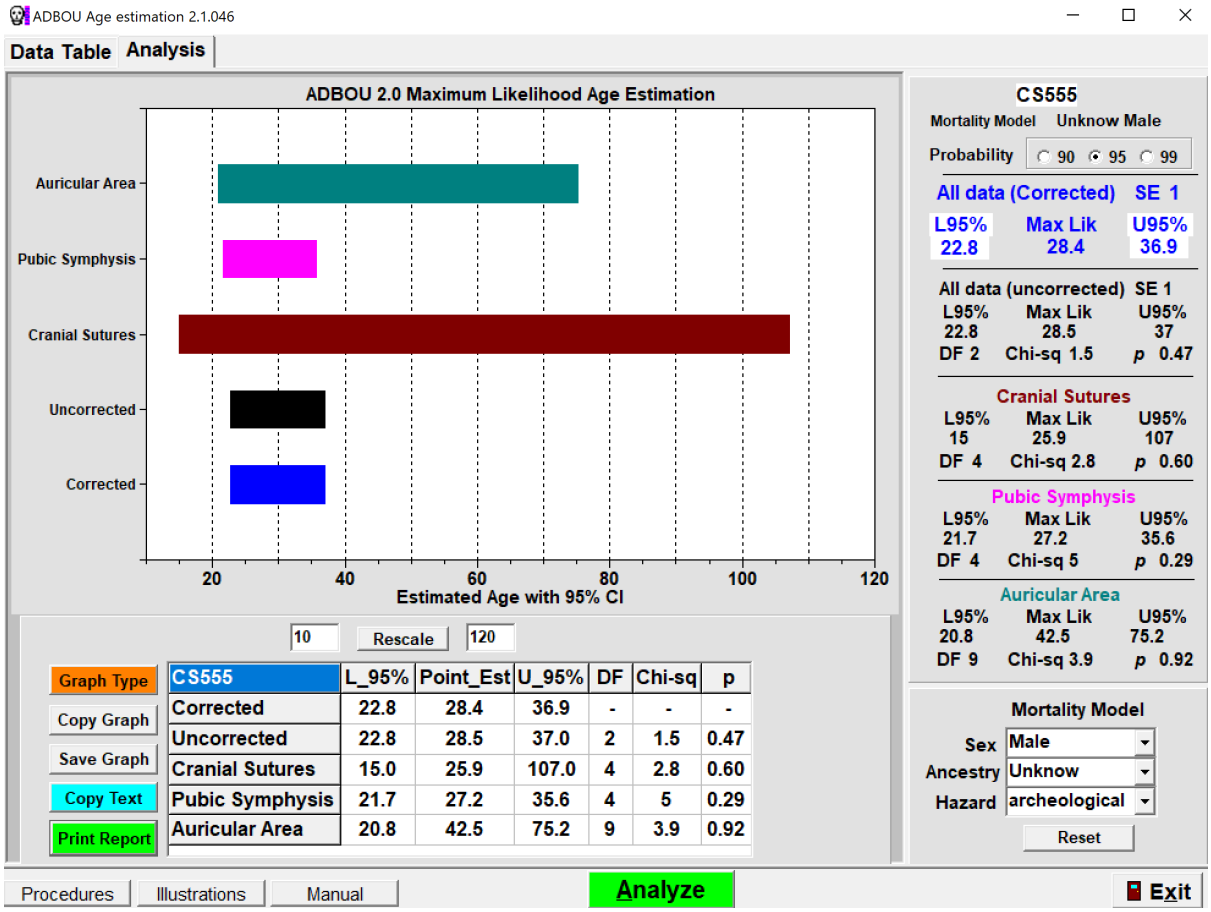

Figure 6.2 Example of age estimation using the ADBOU software on South Shields skeleton CS555. Note the list of figures along the right-hand side and the bottom giving age ranges and statistical values.

While the ADBOU software is a great tool for any palaeopathologist, it is but a single tool among many. In order to capitalize on the benefits of using the ADBOU software, data was gathered for age at death from the pubic symphysis, auricular surface, and cranial sutures. The Suchey-Brooks pubic symphysis method (1990) is used widely and has been found to be among the most accurate at predicting age at death in mature individuals (Djurić *et al.,* 2007). The pubic symphysis degrades throughout life, resulting in a suite of changes to the surface morphology, texture, porosity, and rim shape that correlate with advancing age. As the rate of degeneration varies by biological sex, the sex of the individual must be determined prior to the use of this method. A difficulty of this method is that there is a focus on a total overreaching description of the stages. Due to this, a researcher may not find that the pubic symphysis of a skeleton fits into one category, but the different elements (dorsal margin, ventral margin, superior protuberance, etc.) may fall into the different stages and thereby have a larger overreaching age range. To assist with this method and help compensate for the strict phases, plastic casts of known samples fitting into each stage, with the extremes of each phase and

sorted by biological sex, can be used to provide side-by-side comparisons with the skeletal sample being assessed.

Age at death assessment based on auricular surface morphological changes, (Lovejoy *et al.,* 1985*;* Buckberry and Chamberlain, 2002; Osborne *et al.,* 2004) score different markers (porosity, texture, transverse organization, and apical changes) on individual numerical scales, the values from which are totaled and allocated into a category that has an accompanying age range. The scored categories allow for flexibility that is not offered by other methods, by allowing researchers to score based on single characteristics rather than trying to fit into an overall category with many characteristics (Buckberry and Chamberlain, 2002). In contrast, the Lovejoy and colleagues (1985) method uses detailed descriptions of the overall surface of the joint, and so the test surface might fit multiple stage descriptions based upon the different features. The Buckberry and Chamberlain (2002) method is not as accurate as the Lovejoy and colleages (1985) method in individuals of younger ages, but more accurate with individuals of an older age and more inclusive of different ancestry profiles (Mulhern and Jones, 2005). For this reason, the Buckberry and Chamberlain method was the method used in this research. However, the auricular surface provides a less precise age range that the pubic symphysis method but does provide an accurate age range.

The cranial suture method (Meindl and Lovejoy, 1985) was not used, outside of the ADBOU software, due to the variability of suture closure that can create unreliable and inaccurate results (Boldsen *et al.,* 2002; Wolff, 2013). The Meindl and Lovejoy Method (1985) focuses on 7 ectocranial vault locations and 5 ectocranial lateral-anterior locations. At each location, the suture is scored on a scale of 0-3. This scale reflects the level openness (0) to obliteration (3) and the scores are added up and the resulting tally will provide an age range. The main downfall of this method is the high level of inter-variability between individuals affecting the closure of sutures, making the use of sutures unreliable for the estimation of age (Boldsen *et al.,* 2002).

Other methods for assessing adult age assessment are available but were not used in this study. In particular, methods based on tooth wear were not used, as teeth may be lost or unrecovered, potentially limiting any assessment. Additionally, methods that focus on tooth wear are not accurate for any period of history after the Medieval, as the rate of wear changes with new food types and dental care (Brothwell, 1981; Alayan *et al.*, 2018).

Once age at death had been assigned to each skeleton using ADBOU, the data were grouped for comparison with joint condition patterns. Three categories were used for age within this research. The ranges were as follows: early adult – 18-35 years, middle adult – 36-64 years, late adult – 65+ years. These ranges are reflective of key documented ages of onset of osteoarthritis, 65+ years, and ankylosing spondylitis, c. 35 years (Rogers and Waldron, 1995; Burt *et al.,* 2013) and so facilitate a targeted investigation of age as a risk factor in the onset and progression of these joint conditions in skeletal material. The median ages from the ADBOU assessments were used to assign each individual to these categories.

6.1.1.3 Extant Recorded Data

Age at death and biological sex data recorded previously in published and unpublished reports of the sample collections was used where possible, provided the initial assessment had been undertaken within the past 10 years and standard methods had been used. The reason for this was due to time constraints affecting data collection in certain labs; using the extant demographic data allowed for more time to assess joint condition and markers related to risk factors of joint condition in the level of detail necessary for the aims of the present study. Collections with pre-recorded biological sex and age at death data were St Peter's Collegiate Church, Wolverhampton; Quaker Burial Grounds, Kingston-upon-Thames; and St Peter's Church, Barton-upon-Humber, the data for each stored within the archives of the curating institutions.

In each collection for which extant records existed, a preliminary assessment for biological sex and age at death were completed to test and confirm the accuracy of the records. This step also ensured the comparability of the data to that generated by the author of the present study and the pre-recorded data from other sites. If the extant records were found to differ from the preliminary assessment, a full examination of all skeletons would have been undertaken to generate new data, as explained in the selections above. Where these discrepancies existed, the curating institution was provided with the conflicting information to be able to further check that the records were correct and up to date. This was a rare discrepancy and was discovered in only a handful of skeletal samples.

6.1.2 Osteometric Assessment

The osteometric assessments detailed below were completed using sliding calipers and an osteometric board. The measurements taken were on femur and tibia and included the femoral head diameter, femoral subtrochanteric/midshaft anteroposterior and mediolateral diameters, femoral maximum and bicondylar length, tibial nutrient foramen anteroposterior and mediolateral diameter, and tibial anatomical length. The raw osteometric data was entered into a series of equations for the calculation of body mass and cross-sectional indices. These equations were dependent on biological sex, males and females having different equations, and while the measurements could be taken prior to that assessment, the equations themselves could not be completed until after sex had been assigned.

6.1.2.1 Estimation of Body Mass (BME) and Body Mass Index (BMI)

Data concerning body mass were needed to explore the impact of body size on patterns of joint condition. As an osteoarchaeologist typically has access to only the skeletal remains of an individual, specific methods have had to be devised to estimate the body mass from the skeleton alone. These methods comprise regression-based equations involving skeletal features that have the strongest correlations with body size (Ruff *et al.,* 1991; McHenry, 1992; Grine *et al.,* 1995; Ruff *et al.,* 1997; Auerbach and Ruff, 2004). The two main methods of BME calculations for Western Europeans and Americans include one method based on stature combined with bi-iliac breadth measurements (Ruff *et al.,* 1997) and another method based on the measurement of the breadth of the femoral head (Auerbach and Ruff, 2004). While the stature and bi-iliac breadth method has been tested across broader selection of ancestry profiles than the femoral head method and able to be used on collections with a wider ancestral demographic profile (Ruff *et al.,* 1997), it requires an intact pelvic girdle to enable accurate measurement of the breadth as well as multiple assessors to hold the different skeletal elements together and take measurements. Due to these requirements, the bi-iliac breadth method was unable to be used. Therefore, focusing on measurement of the anteroposterior breadth of the femoral head was preferred and had been tested to find an error rate of 10-16% in living individuals (Ruff *et al.,* 1991). This single element preserves much better in many archaeological samples than a complete pelvic girdle, and with measurements able to be completed by a lone researcher. However, the femoral head has shown that it is has higher

correlation was seen to be higher with the living weights that were reported closer to the age of 18 than the latter ages. Methods for the estimation of body mass based on the femoral head have been tested for use on multiple populations, such as North Americans, Central African foragers, and Khoisan (McHenry 1992), Africans, European Americans, and Native Americans (Grine *et al.* 1995), and a population from Baltimore, Maryland in the USA (Ruff *et al.,* 1991), and further tested on remains from across European populations and time periods (Auerbach and Ruff, 2004).

The anteroposterior (AP) diameter of the femoral head (FH) was measured in millimeters and the following equations applied for body mass estimation:

$$\text{Male Body Mass} = (2.741 * \text{ Femoral head anteroposterior diameter} - 54.9) * .90$$

$$\text{Female Body Mass} = (2.426 * \text{ Femoral head anteroposterior diameter} - 35.1) * .90$$

Separate calculations are provided for males and females, which result in a more accurate estimation of body mass when applied to individuals of the specified sex (Auerbach and Ruff 2004).

Body mass index is a way to examine if an individual is of a healthy weight, which can determine if something is of a 'normal' weight or considered under/overweight (WHO, 2000) (see Literature Review Section 4.4). The combination of body mass estimation and stature can help to create an estimated body mass index value of an individual. A body mass index value does not account for the reasons why an individual might be overweight – this can be due to having increased muscle mass or increased body fat as increased mass is increased mass, no matter the form. Body mass index is primarily used to determine a healthy weight in living individuals, however, as the sample populations are not alive and therefore the soft tissue builds are unable to be assessed to determine if an overweight individual was fatty or muscular, the terms were altered to reflect this (see Table 6.1). These new categories signify whether the skeletal frame was under reduced or increased stress due to lack of or excess mass, rather than focusing on determining whether an individual was a healthy weight.

$$BMI\ (Traditional) = \frac{Weight}{Height^2} \qquad BMI\ (Skeletal) = \frac{BME}{Stature^2}$$

Table 6.1 Body mass index ranges with traditional and proposed skeletal categories.

Range	Category – Traditional	Category - Skeletal
0 – 18.4	Underweight	Under massed
18.5 – 24.9	Healthy/Normal weight	Normal massed
25 – 30	Overweight	Over massed
30.1 +	Obese	Extremely over massed

Calculation of skeletal BMI required a value for BME, calculated as described above, and a value for living stature. Osteometric assessment was used to ascertain a potential living stature from long bone lengths (Trotter, 1970). The method devised by Trotter and Gleser (1952) and refined by Trotter (1970) was used. This method includes equations developed on European samples, which should be consistent with the expected population of England circa 1700-1850 CE. The calculations provide an approximate estimation of stature, with a prescribed amount of error (see Table 6.2). The calculations combining the femur and tibia were preferred, as these are the most accurate, but the calculations using the singular femur or tibia were also utilized when one or the other was too fragmented to be measured accurately. The measurements for each anatomical (femur) or physiological (tibia) length, were taken in millimeters on an osteometric board, were converted to centimeters and inserted into the equations.

Table 6.2 Calculations for stature by element and biological sex (Trotter 1970).

Element	Males	Females
Femur (F) (anatomical length)	= 61.41 + 2.38 (F) ± 3.27	= 54.10 + 2.47 (F) ± 3.72
Tibia (T) (physiological length)	= 78.62 + 2.52 (T) ± 3.37	= 61.53 + 2.90 (T) ± 3.66
Femur + Tibia (FT)	= 63.29 + 1.30 (FT) ± 2.99	= 53.20 + 1.39 (FT) ± 3.55

6.1.2.2 Measurements for the Estimation of Non-Imaging Cross-Sectional Geometry

Skeletal robusticity is a determination of the tensile strengths of the bone under different conditions such as torsion, bending, or fracturing. Robusticity can be assessed via cross-sectional geometry (O'Neill and Ruff, 2004; Hind *et al.,* 2011). Cross-sectional geometry (CSG) has traditionally relied upon destructive methods for sectioning the bone to allow for physical measurement of the cross-sectional structure and shape, however more modern methods apply non-invasive methods using radiographic imaging techniques, such as

computed topography and magnetic resonance imaging. This project was designed to be non-destructive as permission for destructive sampling was not able to be obtained for the collections at Bournemouth University and permissions strictly limited at the collection from Barton-upon-Humber. Access to radiographic or imaging equipment was limited, especially for collections that the author had to travel away from the University of Sheffield to access, and thus a radiographic imaging-based method was not suitable. Wescott and Zephro (2016) have designed a non-destructive method that can be used to gather cross-sectional geometric data by using external measurements on the femur, and this was deemed the most appropriate method to use in the present study.

Cross-sectional geometry can allow researchers to understand the levels of skeletal stress that the individual bones, in this case the femora, can withstand. Bio-mechanical loading has similar properties and characteristics of mechanical engineering load and the effects of torsion to a mechanical joint or brace (Adrian and Cooper, 1995). The Westcott and Zephro method (2016) was used in the present study to calculate the cross-sectional geometric properties from femora in the sample populations. Set points on the femora (femoral max length, head diameter and subtrochanteric/midshaft anteroposterior and mediolateral diameters) were measured to that could then be inserted into the associated equations to determine the values for the different cross-sectional variables. The measurements taken are not exclusive to this method and many are the same used for the meric indices of Bass (2005) or for Trotter's (1970) stature method (see Table 6.3).

The cross-sectional variables calculated from the measurements are shape, robusticity, polar second moment of area (SMA), and area. Shape describes the direction of the femoral head in relation to the diaphyses, the angle of which, when combined with the femur, can affect gait and angle of movement of the femora in relation to the tibiae. Robusticity is the measure of the strength of the bone withstanding torsional effects and polar SMA is a measure of the reflection to the torsional effects. The area calculated from these equations relates to the total size of a 2-dimensional plane within the shell of periosteal bone of the femur. For these measurements, area is an estimated measurement of the combined cortical and medullary cross section.

Table 6.3 The terminology, abbreviations, and equations for determining robusticity (Wescott and Zephro, 2016).

Measurement	**Abbreviation (ABB)**
Femoral maximum length	FML
Femoral head diameter	FHD
Anteroposterior diameter at Subtrochanteric	APS
Mediolateral diameter at Subtrochanteric	MLS
Anteroposterior diameter at Midshaft	APM
Mediolateral diameter at Midshaft	APM
Body Mass [Estimation]	BM

Property	**ABB**	**Equation**
Subtrochanteric shape	FSS	APS/MLS
Subtrochanteric robusticity	FSR	100 * √ (APS x MLS) / FHD
Subtrochanteric polar second moment of area	FSJ	100 * (-124812 + 2925 * APS + 3360 * MLS) / (BM * FML)
Subtrochanteric area	FSA	100 * (π * (APS / 2) * (MLS / 2) / BM)
Midshaft shape	FMS	APM / MLM
Midshaft robusticity	FMR	100 * √ (APM * MLM) / FHD
Midshaft polar second moment of area	FMJ	100 * (-102286 + 2721 * APM + 2697 *MLM) / (BM * FML)
Midshaft area	FMA	100 * (π * (APM / 2) * (MLM / 2) / BM)

6.1.3 Palaeopathological Assessment of Osteoarthritis and Spondyloarthropathies

This research focuses on the assessment of pathological changes to the following joints: acetabulofemoral (hip), sacroiliac, tibiofemoral/ patellofemoral (knee), and the zygapophysial (vertebral facets) and intervertebral joints. There are two main approaches comprising the methodology for diagnosing osteoarthritis in skeletal materials (Rogers and Waldron, 1995). The first approach is stricter and requires a single pathognomic marker, eburnation, to be present. With the pathognomic marker being present, a probable diagnosis is able to be given without the need for further analysis of the joint. Eburnation tends to develop when osteoarthritis is in its latter stages or the joint is well worn, which means that less developed osteoarthritis will not be recorded, creating a lower prevalence of the condition than may exist. Moreover, this approach ignores any nuance in the way in which the skeleton responds to joint

condition, something that is especially important when exploring the associations between skeletal manifestations of joint conditions and their clinical risk factors. The second approach is used when the pathognomic feature is not evident and relies on the presence of a minimum of three other osteological markers.

To assess for osteoarthritis, each element consisting of the joints of the lumbar intervertebral facets, hip and knee were assessed. At each joint location, the markers of OA, discussed in Section 3.4, were assessed for. These markers included eburnation, subchondral sclerosis, porosity/pitting, subchondral cysts, phytic formations and fusion, and were recorded as present or absent with the locations and size of each marker. The size of the markers was irrelevant for a diagnosis but was included for record keeping. Once all of the markers were assessed for, a diagnosis could be attempted. If eburnation was present, then the diagnosis was probable and if not present, then the presence of three of the subsequent markers would provide a probable diagnosis.

To diagnose the vertebral joint conditions, ankylosing spondylitis, sacroiliitis and degenerative disc disease, the methodology presented by Philip Sager (1969) in his thesis was used. The first element assessed would be the sacroiliac joint, as this section would have been examined previously for age at death. If fusion of the sacrum to the iliac surface was present, then sacroiliitis was probable. As DDD and AS may present with similar features, the next assessment was to check for bilateral fusion of the syndesmophytes along the posterolateral portion of the vertebral bodies without skip and starting with the L5 vertebrae. If such fusion was present, then AS was probable, however, if fusion was evident, but skipped and/or not bilateral, then DDD was possible (Rogers and Waldron, 1995). Without fusion, the surface of the vertebral plate and was assessed for morphological changes, such as depression, Schmorl's nodes or osteoporosis and if present, then DDD was probable. The exterior surface, particularly the rim of the vertebral plate was assessed for phytic activity and type of phytic lesion. As with osteoarthritis, these markers were first assessed for and recorded for location and extent with potential differential diagnoses taken into account.

The outcome of diagnosis was represented on three levels: 0- Absence, 1- Possible diagnosis, 2- Probable diagnosis. Absence indicates that there is no evidence of the condition on the skeleton. Possible diagnoses indicate that markers of the different joint conditions were present, but either did not meet diagnosing criteria, or other possible differential diagnoses were found during skeletal assessment. Probable diagnosis indicates that the diagnosing criteria

were met, and no other probable differential diagnoses were available or as plausible during skeletal assessment.

Subchondral sclerosis and cysts are generally not able to be assessed without radiographic imaging, though clear exceptions would be damage or cross sectioning (Rogers and Waldron 1995). Without using destructive methods, radiographs or digital imaging are generally used to view the inner mechanisms of the skeletal elements. However, due to a lack of access to a radiograph device, this was unable to be completed for this body of work, but in the case of radiographs already in existence (Wolverhampton), those were used to help define the makers for the researcher.

Not all markers are directly visible to the naked eye without proper light and viewing instrumentation. For this, the Dino-Lite Pro Digital Microscope and DinoCapture 2.0 software were used. This imagining device, along with different light sources, allowed for the viewing of more subtle features that are not directly visible and the software could take calibrated measurements on the image. For instance, subtle ridges and grooves forming on the joint surface may be felt with fingers rubbing the bone, while not seen with the naked eye (see Figure 6.3). While using this device, different light sources helped to highlight and contrast features, providing a more dynamic visual of the element. To maintain accuracy, the camera was calibrated to provide accurate measurements for each different skeletal element (e.g., femoral head, patella, intervertebral surface, etc.). This allowed for more accurate measurements to be used within the software, as well as allowing for the camera to be used at different heights and angles.

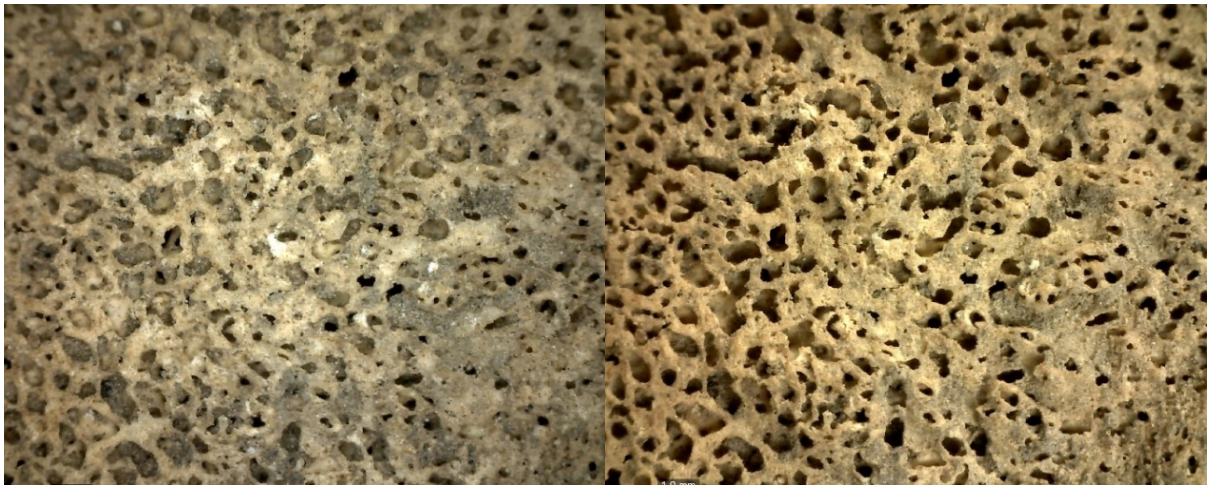

Figure 6.3 The vertebral surface of a lumbar vertebrae. By alternating the light source between direct and indirect the topography of bone becomes more evident. Note that on the left, the surface appears to be more pitted, while on the right it appears more porous. On the vertebral body, heavy porosity could be evidence of osteoporosis. Left: Direct. Right: Indirect.

6.1.4 Assessment of Entheses

Recording of entheses was used for a measurement of activity. Entheses are the attachment sites for tendons and ligaments on the bone. There are two types of entheses: fibrous and fibrocartilaginous. Osteoblastic activity at these sites occurs naturally and so soft tissues may ossify over time meaning development of entheseal changes should be more common in older individuals (Listi, 2016)**.** The surface morphology can vary with each entheseal change leaving much to be recorded, such as porosity, extent of cavitation, curvature, and other features, which are highly dependent on the location of the enthesis (Zumwalt, 2005; 2006; Weiss, 2015). The features, along with the differing proposed aetiologies behind the changes to the two types of enthesis, create a complicated array of markers for assessment (Villotte and Knüssel, 2013).

Methods for recording entheses are not entirely reliable, and interobserver error can make reproducibility low (Davies *et al.,* 2013). The Coimbra Method (Henderson *et al.*, 2013b; 2016; 2017) has sought to create a standardized recording that is both reliable and reproducible by researchers. However, while the Coimbra method is reliable, it is only effective when assessing for fibrocartilaginous entheses and has not been tested on the lower limb (Palmer *et al.,* 2019). As the enthesis assessed within this study are a mix of fibrous and fibrocartilaginous and locations include the lower appendicular skeleton and vertebrae, this method was not able to be used for this research.

The entheses assessed for this study were located on the lumbar and sacral vertebra, pelvis, femur, patella and proximal tibia (see Table 6.4). The changes were scored for each location as: 1- gracile, 2- moderate 3- robust (see Figure 6.4) and are based on the method devised by Mariotti and colleagues (2004; 2007) for use on the lower appendicular skeleton and pelvic girdle and the methodology of Philip Sager (1969) for the vertebral enthesophytes. Mariotti and colleagues (2004; 2007) developed a method for recording entheses both fibrocartilaginous and fibrous entheses (Palmer *et al.,* 2019). The method has a description for the recording of the entheses at each location however instead of scoring each marker, the score describes the entheses as a whole. The following is an example of the description for scoring an enthesis on the femur:

M. vastus medialis (superior part)

1. a – slight impression: the surface is practically smooth, even though an oblique line is perceptible to the touch; b – low development: the insertion is marked by a rugose, oblique line; c – medium development: the line of insertion forms a continuous or discontinuous ridge, not very raised.

2. high development: the line of insertion forms a raised and/or rugose crest.

3. very high development: very raised and/or rugose crest (Mariotti et al., 2007: 309).

The lumbar vertebrae had the enthesophytes measured with 1 - >1mm, 2 – 1-5mm, and 3 - <5mm and is based on the work by Sager (1969). As the individual entheses were scored on the skeletal, an average was created to allow for an overall assessment of the entheseal development to be made on the skeleton.

Table 6.4 Location of entheses assessed within this research.

BONE	ENTHESES	ATTACHMENT	LOCATION
PELVIS	ILIACUS	ORIGIN	ILIAC FOSSA
PELVIS	ALL ATTACHMENTS	ORIGIN	ILIAC CREST
FEMUR	GLUTEUS MEDIUS	INSERTION	GREATER TROCHANTER
FEMUR	GLUTEUS MINIMUS	INSERTION	GREATER TROCHANTER
FEMUR	PSOAS MAJOR	INSERTION	LESSER TROCHANTER
FEMUR	ALL ATTACHMENTS	INSERTION	LINEA ASPERA
PATELLA	QUADRICEPS TENDON	INSERTION	SUPERIOR ASPECT
TIBIA	SEMIMEMBRANOSUS	INSERTION	POSTERIOR PORTION OF MEDIAL CONDYLE
TIBIA	PATELLAR LIGAMENT	INSERTION	TIBIAL TUBEROSITY
TIBIA	SOLEUS	ORIGIN	SOLEAL LINE

Figure 6.4 The image demonstrates the degree of change used for recording the entheses. The left image of a femur (female, CS300) displays slight changes, the middle (male, CS208) displays moderate changes, and the right (male, CS268) displays robust changes.

6.1.5 Exclusion of Samples

As well as performing in-depth assessments for joint conditions, secondary conditions that may impact or affect the development of the joint conditions were, by necessity, needed to determine inclusion/exclusion of the individual sample skeletons. The conditions were assessed using Roberts and Manchester's (2005) *The Archaeology of Disease* and Tony Waldron's (2009) *Palaeopathology* as guides. Skeletons found to have any conditions that could cause secondary joint conditions were noted and removed from the raw sample. The reason for this is that it would bias the subsequent statistical testing, as the periphery conditions would be the cause of the secondary joint conditions, not the risk factors.

6.2 Statistical Testing

Statistical testing was essential to investigate the relationships between demographic variables, joint condition prevalence and the various variables representing potential risk factors. Biological sex, age at death, entheseal changes and joint conditions were able to utilize the scale or categorical groupings previously listed (see Table 6.5). Body mass index and the cross-sectional indices utilized the methods for assessment previously listed in the above section, which created scale data. Assessments of correlation, association, and regression testing were performed on these data.

Table 6.5 List of the type of variables with the type of data categorization used to represent the data for statistical analyses.

VARIABLE	DATA CATEGORIZATION		
	ORDINAL	NOMINAL	SCALE
SITE CATEGORY		X	
AGE AT DEATH	X		X
BIOLOGICAL SEX		X	
ENTHESEAL CHANGES	X		
JOINT CONDITIONS		X	
RAW SKELETAL MEASUREMENTS			X
SKELETAL HEIGHT			X
SKELETAL WEIGHT			X
SKELETAL BMI	X		X
CSG INDICES	X		X

6.2.1 Asymmetry

The left side of paired skeletal elements were recorded in this study, but to confirm this decision did not introduce bias into the dataset, asymmetry tests were conducted comparing right and left values. By its very definition, asymmetry denotes a disparity between two similar features (i.e., left and right-side long bones). When taking a measurement a few millimeters might not seem like a large discrepancy, but that discrepancy can be amplified when placed into an equation to determine a variable like stature or body mass. If asymmetry existed and was variable between individuals within the database, this could skew or bias the data in a way that would have problematic effects during subsequent analyses. Therefore, it is important to deduce if any asymmetry exists, and if so, its extent and directionality.

Percentage directional asymmetry (%DA) and percentage absolute asymmetry (%AA) were checked for each individual using the equations (Steele and Mays, 1995):

*%DA = (right – left values) / (average of left + right values) * 100*

*%AA = (maximum – minimum value) / (average of maximum + minimum values) * 100*

The variables that were checked included all basic measurements of the femora and tibiae as well as the calculated measurements for skeletal height, mass, BMI, and the cross-sectional indices. Once the asymmetry values were created, paired t-tests were used to assess whether significant difference existed between sides (Gonzalez-Chica *et al.,* 2015).

6.2.2 Correlation and Association Testing

The data is a mix of categorical and scale data, which meant that the variables with a scale data set would need to be placed into categories to allow for a uniform testing system for correlation and association tests. Association was tested using Chi-squared with the Bonferroni z-test. Levels of significance were tested primarily using Kendall's tau-b criteria because the data sets within this research were discordant and non-parametric, however, the phi and Pearson correlation coefficients were also used were applicable. To determine the relationship between the individual variables, as well as the relationships between the risk factor variables and the joint conditions, bivariate correlation testing was conducted (Field, 2013).

6.2.3 Risk Ratios

Risk ratios help to determine levels of risk for a given factor as the factor increases or decreases compared to the studied conditions (Holmberg and Andersen, 2020). Levels of risk can also be calculated within a single category, or, in the case of the Table 6.6, a single row. Biological sex is a binary variable and so an exception, as the categories do not increase or decrease. The variables, in categorical form, were completed using the following equation (see Table 6.6):

Prevalence Ratio (for Ratio 1) = Count of present diagnoses [A_2] / (count of absent diagnoses [A_1] + count of present diagnoses [A_2])

Risk ratio = (count of present diagnoses [A_2] / (count of present diagnoses [A_2] + count of absent diagnoses[A_1])) / (count of present diagnoses / (count of present diagnoses [B_2] + count of absent diagnoses [B_1]))

Table 6.6 Example of a risk ratio computed in table form.

	COUNT		PREVALENCE RATIO	
	A	P	A	P
1	A_1	A_2	= A_1 / (A_1 + A_2)	= A_2 / (A_1 + A_2)
2	B_1	B_2	= B_1 / (B_1 + B_2)	= B_2 / (B_1 + B_2)
	RISK RATIO			
2/1	= (B_2 / (B_1 + B_2)) / ((A_2 / (A_1 + A_2))			

For the purposes of this study, the resulting figure of the incidence rate was equivalent to the prevalence rate – the number of individuals with a joint condition as a proportion of the total number of individuals for whom the joint location was observable. The value of the risk ratio is the ability to determine to determine potential likelihood for risk of one category of the variable over another. For example, using the variables biological sex and osteoarthritis, an incidence rate of .369 for males with a present diagnosis means that 36.9% have the condition. A risk ratio of 1.21 when comparing males to females with a present diagnosis would imply that males experience the condition 1.21 times more often than females.

6.2.4 Regression Based Testing

Bivariate statistical testing will display the strength, direction, and significance of association or correlation between two variables. However, the clinical understanding of joint conditions suggests it is unlikely that a single risk factor will sufficiently explain its occurrence. Therefore, a means of exploring the relationship between all of the risk factor variables and the joint conditions is required. The risk factors are a determinate of the different joint conditions researched within this body of work and as such, their potential predictive value for a binary outcome, presence/absence of a joint condition, can be explored using binary logistic regression models (Sheather, 2009; Field, 2013). By using this form of regression, it was possible to determine which variables were significant contributors to the outcome of joint

condition presence/absence and to what extent they played a part in predicting the presence of the joint conditions.

The joint conditions (dependent variable) were measured against every risk factor variable (covariates) in a backwards, stepwise manner. By using backward steps, SPSS will start the tests with all risk factor variables included and each subsequent step will produce another model until the variables are significant, unless specified otherwise. A series of tables were produced with information that helps to understand predictability, variance, and relationship of the risk factor variables with the joint conditions. The Omnibus Tests of Model coefficients provides the chi-square values and significance for each step and a Model Summary will display the level of variation (-2 log likelihood) as well as the percentage explained in the variation (an R^2 value). The Hosmer-Lemeshow tests explains how well the model fits the data, if the p value > .05. A classification table will be produced indicating the percentage that the model is able to predict the outcome, in this case absence, probable diagnoses and overall predictability. The final table of import describes the risk factor variables in each step, providing a regression coefficient value (B and expected B with confidence level), a Wald statistic and p value. This testing helped to determine the contribution of the variables and predictive value when all the variables were included, as well as when the best fitting variable determined from the stepwise statistical procedures.

6.3 Summation of Methodologies

The methodologies used within this thesis allow for the development of an in-depth demographic profile of the skeletal samples that could be analyzed in detail at multiple layers. These layers could have variables added and removed to assess the efficacy of each variable and the role and strength they have in the development of joint conditions in the sample populations. The profiles offer insights into the lives of the individuals and the populations as a whole.

On the face of the analyses, graphs and tables may display patterns that appear to be significant but are not. That is the reason the statistical methodologies are vital. The basic statistical tests (asymmetry and correlation/association) can determine if there is a bias between the sides and show the strength of relationships between the variables representing the risk factors and the joint conditions. The regression testing provides a check, using objective testing

to determine the relationships/associations, strength, direction, and the level of variation found using multiple variables, which helps to display and explain the true complexity of the risk factors and the joint conditions.

Chapter 7 Results

7.1 Sample Demographics

The six sites assessed in this study comprised a population of 187 adult skeletons, which was reduced to 175 individuals suitably preserved to allow for full data analysis (Table 7.1/ Figure 7.1). This information is descriptive in nature, and the statistical analyses will come below. The sites from which this sample of individuals derived were divided into two categories: urban and rural. The two site categories comprised roughly equal numbers of individuals: 90 individuals (51.4%) were assessed from urban sites and 85 (48.6%) individuals were assessed from the rural site.

Table 7.1 The sites used within this research with information regarding site name, location, category, and sample size.

SITE #	SITE NAME	ORIGINAL LOCATION	URBAN/ RURAL	SAMPLE SIZE
1	CHURCH OF ST HILDA, CORONATION STREET	SOUTH SHIELDS, NEWCASTLE-UPON-TYNE	URBAN	46 (26.3%)
2	CARVER STREET METHODIST CHURCH	SHEFFIELD	URBAN	4 (2.3%)
3	ST PETER'S COLLEGIATE CHURCH	WOLVERHAMPTON	URBAN	20 (11.4%)
4	QUAKER BURIAL GROUND	KINGSTON-UPON-THAMES	URBAN	18 (10.3%)
5	ST AUGUSTINE-THE-LESS	BRISTOL	URBAN	2 (1.1%)
6	ST PETER'S CHURCH	BARTON-UPON-HUMBER	RURAL	85 (48.6%)

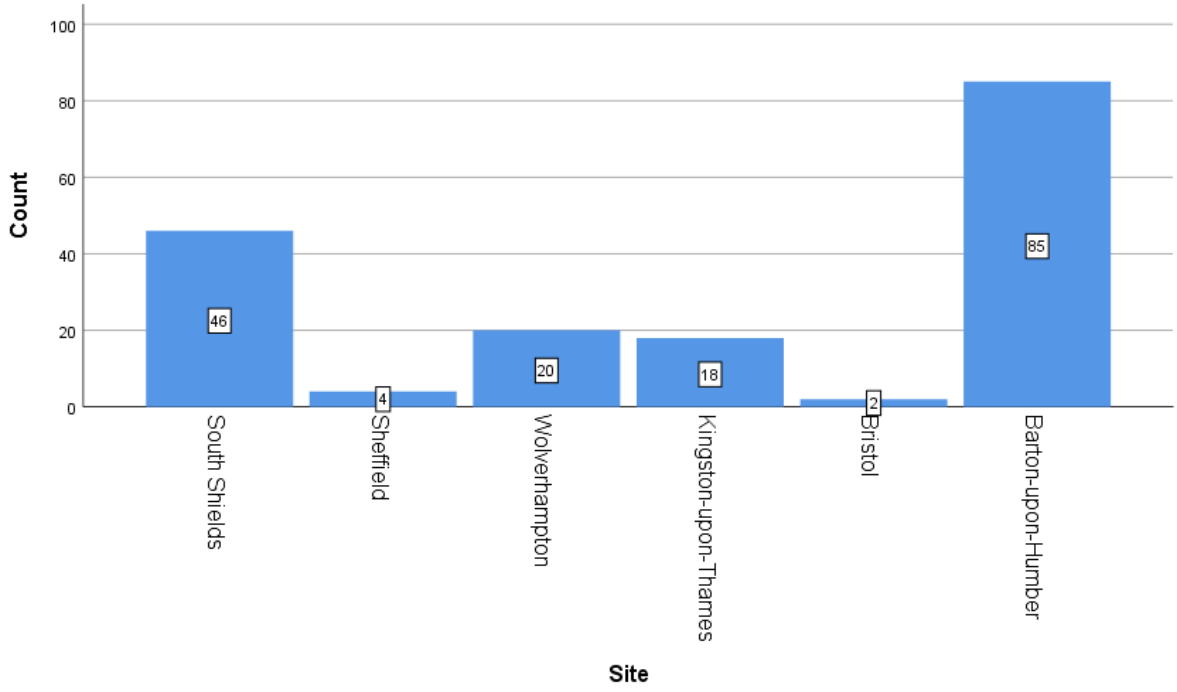

Figure 7.1 Proportional distribution of individuals analyzed in this study by site. Count is indicated within the bars.

7.1.1 Biological Sex

The study sample comprised 58.9% males (n = 103) and 41.1% females (n = 72) (see Table 7.2/ Figure 7.2). For the urban samples, 62.2% (n = 56) were male and 37.8% (n = 34) were female (see Table 7.3). For the rural samples, 55.3% (n = 47) were male and 44.7% (n = 38) were female. Of the male individuals, the rural site represented 54.4% (n=56) and the urban sites represented 45.6% (n = 47). Of the female individuals, the rural site represented 47.2% (n = 34) and the urban sites represented 52.8% (n = 38).

While the overall male to female ratio is close to equal, females are still slightly underrepresented, especially so among the urban group. Males were also slightly over-represented at every site and the two sites with the smallest sample sizes were only males.

Table 7.2 Distribution of the sample population with count and proportion by site category and biological sex.

	SAMPLE SIZES				PERCENTAGES	
SITE	MALE	FEMALE	TOTAL SITE		MALE	FEMALE
South Shields	27	19	46		58.7	41.3
Sheffield	4	0	4		100.0	0.0
Wolverhampton	12	8	20		60.0	40.0
Kingston-upon--Thames	11	7	18		61.1	38.9
Bristol	2	0	2		100.0	0.0
Barton-upon-Humber	47	38	85		55.3	44.7
TOTAL SEX	103	72	175		58.9	41.1

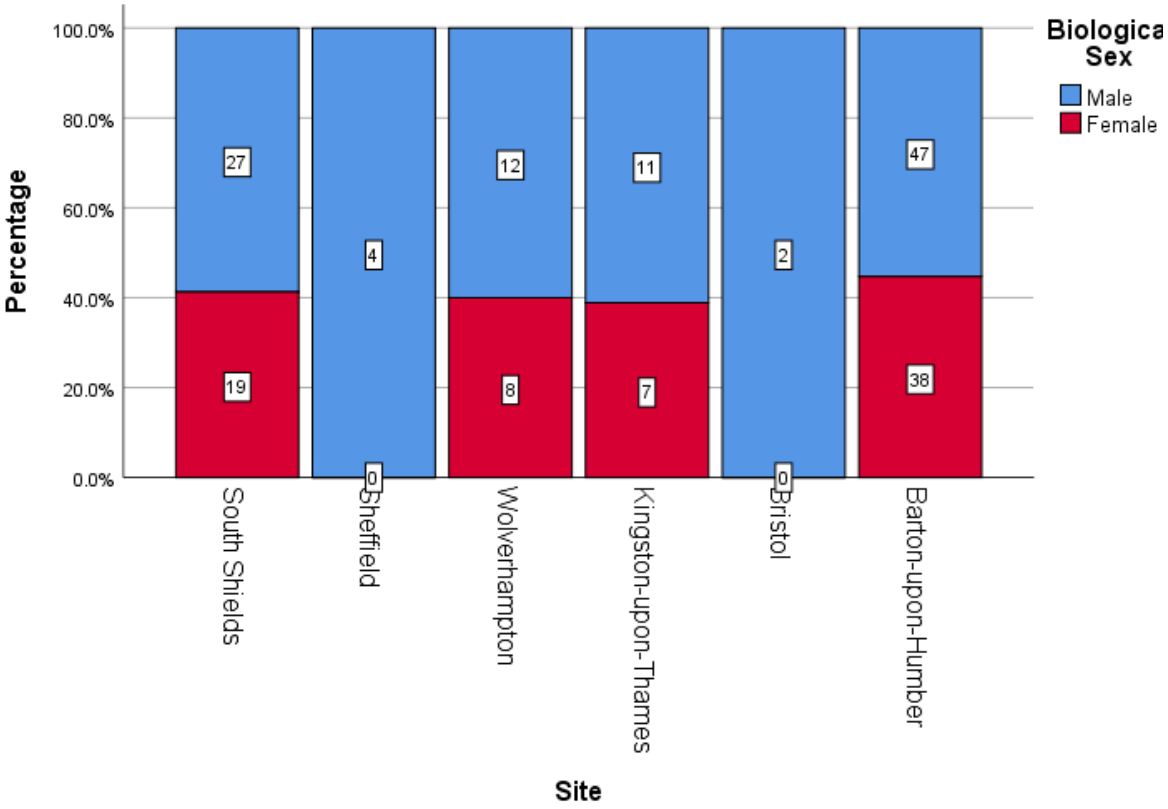

Figure 7.2 Proportional distribution of the sample population by site and biological sex. Count is indicated within the bars.

Table 7.3 Distribution of the sample population by site category and biological sex with count and percentage.

	SAMPLE SIZE			PERCENTAGE	
	MALES	FEMALES		MALES	FEMALES
URBAN	56	34		62.2	37.8
RURAL	47	38		55.3	44.7
PROPORTION BY SITE CATEGORY					
	SAMPLE SIZE			PERCENTAGE	
	RURAL	URBAN		RURAL	URBAN
MALES	56	47		54.4	45.6
FEMALES	34	38		47.2	52.8

The percentages total by row.

7.1.2 Age at Death

The age at death of the individuals was recorded in two ways, creating two different variables. The first was based on either the maximum likely age at death from the ADBOU data or the documented age at death, when available. This category is continuous data, with a single data point representing a single estimated age for each skeleton. The second was the age range was determined by the age of onset for the conditions, with each point value age placed into one of the range groupings. This variable grouped multiple individuals into one of three broad categories based on a combination of methods: early adults 18-35, middle adults 35.1 – 65, late adults 65.1+.

Of the entire sample population, 32.0% (n = 56) were early adults, 22.9% (n = 40) were middle adults, and 45.1% (n = 79) were late adults (see Table 7.4). Of the males (see Figure 7.3 - Figure 7.5), 27.2% (n = 28) were early adult, 21.4% (n = 22) were middle adult, and 51.5 (n = 53) were late adult. Of the females, 38.9 (n=28) were early adults, 25.0% (n = 18) were middle adults, and 36.1% (n = 26) were late adults. The distribution of the sample population favored the late adult age category, which was only slightly below half of the total population size. The males were an older population overall, with the females having a more equal distribution between the three age categories, though middle adults were still the least represented.

Table 7.4 Distribution of the sample population by biological and age at death with count and percentage.

BIOLOGICAL SEX	AGE AT DEATH CATEGORY	N	%
MALE	EARLY	28	27.2
	MIDDLE	22	21.4
	LATE	53	51.5
FEMALE	EARLY	28	38.9
	MIDDLE	18	25.0
	LATE	26	36.1
TOTAL	EARLY	56	32.0
	MIDDLE	40	22.9
	LATE	79	45.1

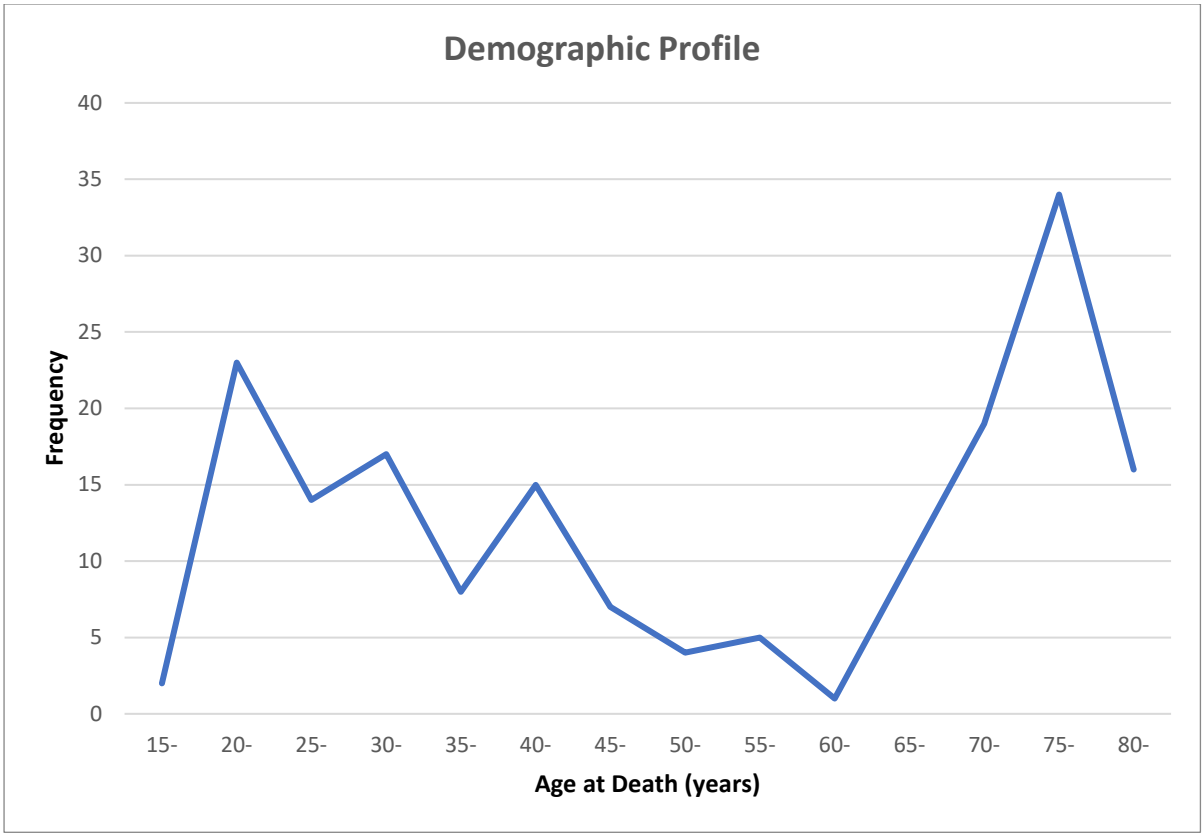

Figure 7.3 Frequency of all samples for the estimated age at death data. N = 175.

Figure 7.4 Proportional distribution of the sample population by biological sex and age at death. Count is indicated within the bars.

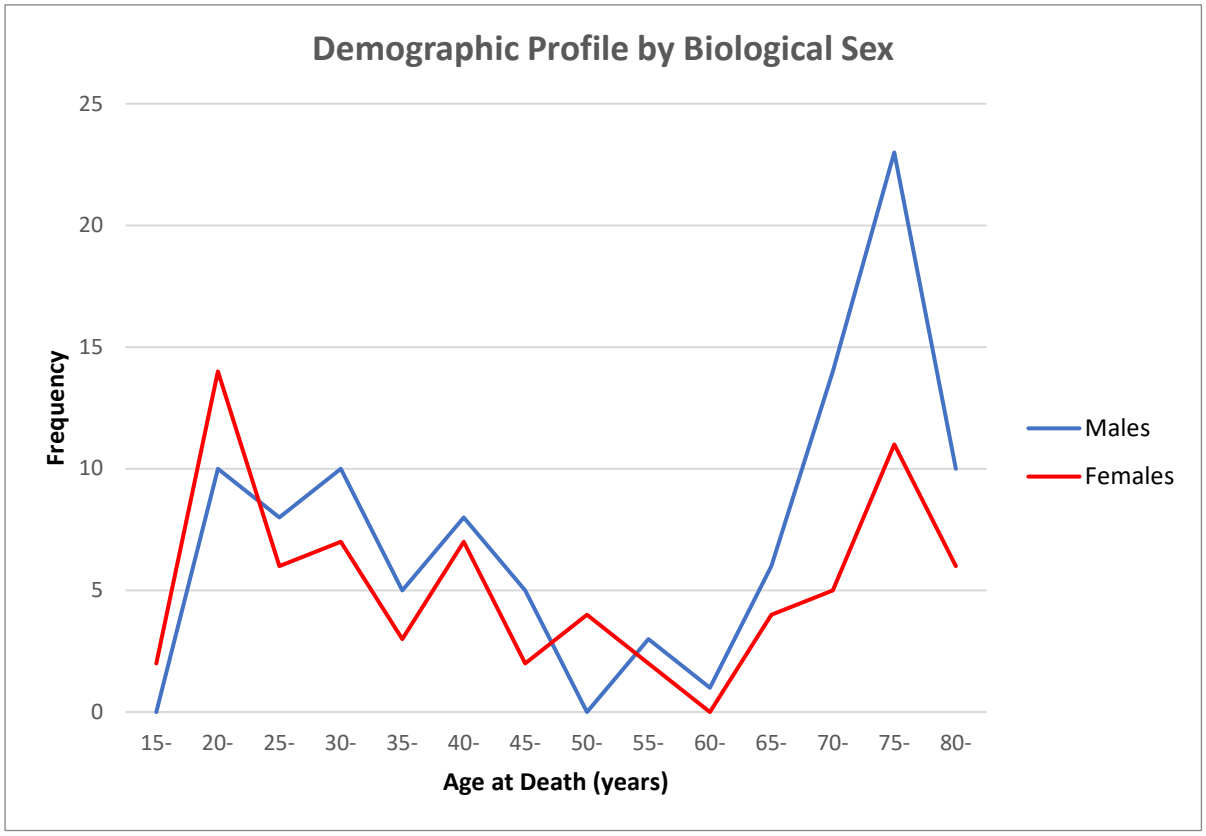

Figure 7.5 Frequency of all samples by estimated age at death data, divided by biological sex.

From the breakdown of age at death categories by site category (see Table 7.5), it was observed that the urban population had a higher proportion of early adults (37.8%, n = 34) than the rural population (28.2%, n = 24). Middle adults had a similar proportion with the urban population (21.1%, n = 19) and the rural population (21.2%, n = 18) (see Figure 7.6 and Figure 7.7). The rural population had the higher proportion of late adults (50.6%, n = 43) than the urban population (41.1%, n = 37).

Urban sites had higher proportion of early adult males (30.4%, n = 17) than rural (21.3%, n = 10). The rural site type had higher proportions of males within the middle (23.4%, n = 11) and late (53.3%, n = 26) adult categories than the urban populations (19.6%, n = 11 and 50.0%, n = 28). Urban females had a higher proportion of individuals within the early (50.0%, n = 17) and middle (23.5%, n = 8) adult categories than the rural females (38.8%, n = 14 and 18.4%, n = 7). Rural females had the higher proportion within the late adult categories (44.7%, n = 17) than the urban females (26.5%, n = 9).

Overall, the rural population was older than the urban population. The slight bias towards older individuals within the male cohort seen in the grouped data above was maintained in the urban and rural populations when divided, but it is notable that there is a disparity between the demographic profile of males and females at urban sites – the former being overrepresented among the late adults and the latter among the early adults.

Table 7.5 Distribution of sample population by site category, biological sex, and age at death with count and percentage.

URBAN SITE TYPE				RURAL SITE TYPE			
BIOLOGICAL SEX	**AGE AT DEATH CATEGORY**	**N**	**%**	**BIOLOGICAL SEX**	**AGE AT DEATH CATEGORY**	**N**	**%**
MALE	EARLY	17	30.4	MALE	EARLY	10	21.3
	MIDDLE	11	19.6		MIDDLE	11	23.4
	LATE	28	50.0		LATE	26	55.3
FEMALE	EARLY	17	50.0	FEMALE	EARLY	14	36.8
	MIDDLE	8	23.5		MIDDLE	7	18.4
	LATE	9	26.5		LATE	17	44.7
TOTAL	EARLY	34	37.8	TOTAL	EARLY	24	28.2
	MIDDLE	19	21.1		MIDDLE	18	21.2
	LATE	37	41.1		LATE	43	50.6

Note that values may not ≠ 100 as decimal point placement is rounded.

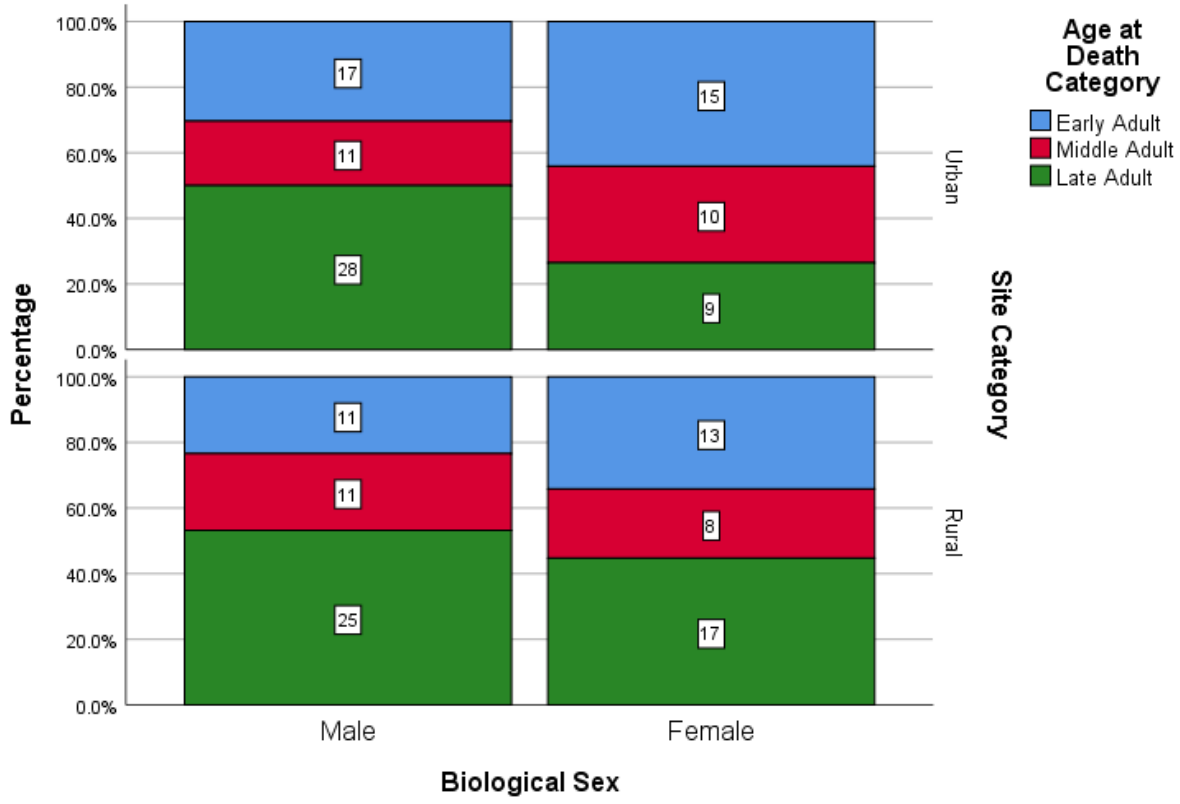

Figure 7.6 Proportional distribution of the sample population by site category, biological sex, and age at death. Count is indicated within the bars.

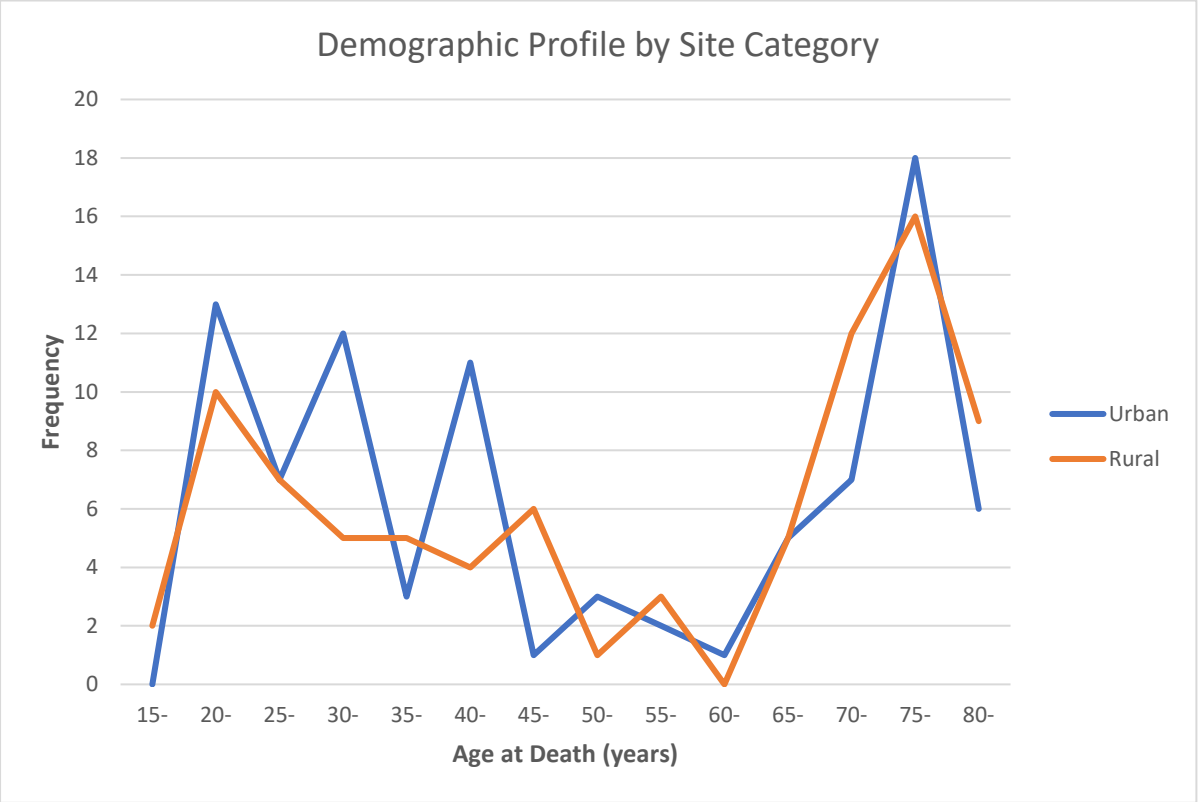

Figure 7.7 Frequency of samples by estimated age at death by site category.

7.1.3 Demographic Summary

The sample population was spread relatively evenly throughout the demographic categories, which should assist with the reduction of bias caused by uneven distributions and aid the forthcoming analysis of risk factors by providing a reasonable sample of individuals in each category. These risk factors are not caused by any other factor and are the only independent variables used within this research. The spread of the samples should help to create an accurate and viable sample population that is similar to clinical studies and will help us to understand living populations. Therefore, they must be understood in order to help assist with the testing and analyses with the dependent risk factor variables (body mass, entheseal changes and cross-sectional indices) which will be discussed later in the chapter.

7.2 Asymmetry

To ensure no bias in the forthcoming analysis arising from bilateral asymmetry relating to skeletal measurements, percent absolute (%AA) and percent directional (%DA) asymmetry was measured, and paired t-tests were conducted**.** Asymmetry was measured against variables of both (1) raw data from skeletal long bone measurements that was used within the equations for the more complex variables (e.g., femoral max length and femoral head diameter) and (2) the complex variables that were created using said equations (e.g., body mass estimation and the cross-sectional variables).

There was a measurable level of bilateral asymmetry when the left and right-side measurements were compared, which alternated by robusticity variable. By calculating percentage absolute asymmetry and directional asymmetry it became evident that individual values may be high with significant asymmetry variations, but the mean values are low. This can be explained by the fact that directional variation fluctuates between the left and right side with no side being dominant in all individuals (see Table 7.6 - Table 7.10. The range for percent absolute asymmetry was .43 – 10.68 and for percent directional asymmetry was -1.92 – 1.45. However, only six of the 26 (23.08%) variables exhibited significant difference, with $p \geq .02$, between the mean values for left and right sides. Femoral head diameter measurements (%DA .69/ %AA 2.16), femoral head body mass estimation (%DA 1.12/ %AA 3.47) and femoral midshaft shape (%DA 1.45/ %AA 4.94) displayed significant differences between the left and right sides with the right side being larger. Femoral midshaft mediolateral measurements (%DA -1.17/ %AA 4.08), and femoral midshaft robusticity (%DA -1.12/ %AA 3.23) and area (%DA -1.92/ %AA 5.93) displayed significance between the left and right side with the left side being larger. To standardize the measurements and calculations used, only the left-hand side was used.

The equations selected to determine the variables representing body mass index and the cross-sectional indices created values for both the left- and right-hand side of both. The differences between the sides did not exhibit a significant asymmetrical difference for body mass index. The robusticity values had three variables that displayed significance between the sides, all midshaft values: femoral midshaft shape (right), robusticity (left) and area (left).

Table 7.6 Abbreviations and the meanings used within Table 7.7 - Table 7.10.

ABBR	MEANING	ABBR	MEANING
FHD	FEMORAL HEAD DIAMETER	AP	ANTEROPOSTERIOR
FML	FEMORAL MAX LENGTH	ML	MEDIOLATERAL
FBL	FEMORAL BICONDYLAR LENGTH	NUT FOR	NUTRIENT FORAMEN
TAL	TIBIAL ANATOMICAL LENGTH	ST	STATURE
FEM	FEMUR/FEMORAL	TIB	TIBIA/TIBIAL

Table 7.7 Asymmetry data for raw skeletal data using paired t-tests.

MEASUREMENT	%DA	%AA	MEAN	STD DEV.	STD ERROR MEAN	95% CONFIDENCE INTERVAL OF THE DIFFERENCE		t	DF	SIG. (2-TAILED)
						LOWER	UPPER			
FHD	0.6903	2.1610	-0.3161	1.3357	0.1141	-0.5418	-0.0905	-2.77	136	0.006
FML	-0.0180	0.7859	0.0690	5.5820	0.5180	-0.9580	1.0960	0.133	115	0.894
FBL	-0.1679	0.7670	0.7500	4.9600	0.4610	-0.1620	1.6620	1.662	115	0.106
TAL	-0.1241	0.9119	0.4730	6.5740	0.5770	-0.6670	1.6140	0.821	129	0.413
TIB NUT FOR ML	-0.0093	4.5934	0.0264	1.6976	0.1395	-0.2494	0.3021	0.189	147	0.850
TIB NUT FOR AP	-0.8317	5.3283	0.2487	2.3233	0.1910	-0.1288	0.6261	0.1302	147	0.195
FEM SUBTROCH AP	0.8660	4.8162	-0.2407	1.9562	0.1659	-0.5687	0.0874	-1.45	138	0.149
FEM SUBTROCH ML	0.1920	4.4580	-0.0551	2.0369	0.1728	-0.3967	0.2865	-0.319	138	0.750
FEM MIDSHAFT AP	0.2365	3.8370	-0.0615	1.4765	0.1310	-0.3208	0.1978	-0.469	126	0.640
FEM MIDSHAFT ML	-1.1661	4.0825	0.3431	1.4924	0.1324	0.0810	0.6051	2.591	126	0.011

AP = anteroposterior. ML = mediolateral. Mean and standard deviation represent the individual of %DA across the sample population. Negative %DA values represent left > right and positive values represent right > left. Green = significant left leaning asymmetry. Orange = significant right leaning asymmetry.

Table 7.8 Asymmetry data for the cnemic/meric data using paired t-tests.

MEASUREMENT	%DA	%AA	MEAN	STD DEV.	STD ERROR MEAN	95% CONFIDENCE INTERVAL OF THE DIFFERENCE		t	DF	SIG. (2-TAILED)
						LOWER	UPPER			
CNEMIC	0.8244	5.7197	-0.5287	6.2261	0.5118	-1.5401	0.4828	-1.033	147	0.303
MERIC	0.6710	5.7786	-0.6527	7.1300	0.6048	-1.8485	0.5431	-1.079	138	0.282

Mean and standard deviation represent the changes of %DA across the sample population. Negative %DA values represent left > right and positive values represent right > left. Green = significant left leaning asymmetry. Orange = significant right leaning asymmetry.

Table 7.9 Asymmetry data for the stature and body mass data using paired t-tests.

MEASUREMENT	%DA	%AA	MEAN	STD DEV.	STD ERROR MEAN	95% CONFIDENCE INTERVAL OF THE DIFFERENCE		t	DF	SIG. (2-TAILED)
						LOWER	UPPER			
ST FEM	-0.1087	0.5185	0.1810	1.1913	0.1106	-0.0381	0.4001	1.637	115	0.104
ST TIB	-0.0545	0.6467	0.0913	1.8410	0.1634	-0.2320	0.4145	0.559	126	0.577
ST FEM/TIB	-0.0451	0.4347	-0.4946	5.8253	0.5855	-1.6564	0.6673	-0.845	98	0.400
ST (AVE)	-0.0926	0.7286	0.2320	18.6818	1.5305	-2.7925	3.2563	0.152	148	0.880
FH BME	1.1187	3.4692	-0.7349	3.0456	0.2602	-1.2495	-0.2203	-2.824	136	0.005
BMI	1.2637	4.0161	-0.1407	2.4339	0.2151	-0.5664	0.2850	-0.654	127	0.514

Mean and standard deviation represent the changes of %DA across the sample population. The rows in bold are the significant results. Negative %DA values represent left > right and positive values represent right > left.

Table 7.10 Asymmetry data for the robusticity data using paired t-tests.

MEASUREMENT	%DA	%AA	MEAN	STD DEV.	STD ERROR MEAN	95% CONFIDENCE INTERVAL OF THE DIFFERENCE		t	DF	SIG. (2-TAILED)
						LOWER	UPPER			
FSS	1.4466	4.9364	-0.0064	0.0706	0.0060	-0.0183	0.0054	-1.069	138	0.287
FSR	0.1378	3.8464	-0.1048	3.6706	0.3270	-0.7520	0.5423	-0.321	125	0.749
FSJ	1.2985	10.6770	-1.8906	35.7169	3.4856	-8.8027	5.0215	-0.542	104	0.589
FSA	0.5692	7.4001	-6.5312	115.6670	10.3044	-26.9250	13.8626	-0.634	125	0.527
FMS	1.4466	4.9354	-0.0157	0.0639	0.0057	-0.0269	-0.0045	-2.764	126	0.007
FMR	-1.1177	3.2268	0.7007	2.8777	0.2672	0.1714	1.2299	2.622	115	0.010
FMJ	-1.1643	10.1882	3.1839	24.5709	2.3535	-1.4811	7.8488	1.353	108	0.179
FMA	-1.9201	5.9286	18.5683	84.5686	7.8520	3.0150	34.1216	2.365	115	0.020

Mean and standard deviation represent the changes of %DA across the sample population. Negative %DA values represent left > right and positive values represent right > left. Green = significant left leaning asymmetry. Orange = significant right leaning asymmetry.

7.3 The Joint Conditions (JC)

To be recorded as having a joint condition for the purpose of this research, an individual must have at least one of the joint conditions being researched: general OA, OA of the spine, hip, and knee, AS, SI and DDD. Following full osteological assessment which recorded skeletal changes at the joints in detail and included differential diagnosis, if any, individuals were placed into one of three categories reflecting the extent of palaeopathological evidence for joint condition: no joint condition, possible diagnosis of joint condition, probable diagnosis of joint condition. The categories for diagnosis are further explained below:

Absent: No evidence for the joint conditions is present on the joint.

Possible diagnosis: An individual displayed evidence of skeletal change related to a joint condition, but not enough criteria were present for a definitive diagnosis. Differential diagnosis may have indicated two or more aetiologies that could be equally as likely. This category was used for the purposes of initial skeletal assessments. For statistical purposes and reporting, this category was merged into the absent category, as a possible diagnosis would have no bearing on the statistical testing. The exception to this rule is for ankylosing spondylitis, which will be further explained later in this section.

Probable diagnosis: An individual with enough criteria to conclude that a joint condition is present, with a single aetiology implicated by differential diagnosis.

The descriptions of these diagnoses are discussed as prevalence rates. The individual joint condition examined have an assumed 95% confidence interval and are point prevalence for the true count of the sample population as counted and diagnosed. The combined joint condition and osteoarthritis prevalences display an overall crude point prevalence and the individual joint conditions display prevalences that are more true point prevalences. However, as these samples are from assemblages that may have missing elements, not all skeletons may be counted for each condition, and so the prevalences describe non-paired extant elements present during the examinations.

In total, 49.7% of the individuals (n = 85) presented with evidence for a probable diagnosis for at least one of the joint conditions studied during this research (see Table 7.11) and 50.3% of individuals (n = 88) fell into the category of absent. Between the site categories

(see Figure 7.8), the rural population displayed the higher proportion of individuals with a probable diagnosis (50.6%, n = 43) than the urban population (48.9%, n = 44), however the difference was small. Males, overall, presented a higher prevalence of joint condition with 53.4% (n = 55) displaying enough criteria for a probable diagnosis in comparison to a total of 44.4% of female individuals (n = 32) (see Table 7.12/ Figure 7.9).

Table 7.11 Distribution of the diagnoses for all the joint conditions studied within this sample population by site category.

SITE CATEGORY	DIAGNOSIS	N	%
URBAN	ABSENT	46	51.1
	PROBABLE	44	48.9
RURAL	ABSENT	42	49.41
	PROBABLE	43	50.6
TOTAL	ABSENT	88	50.3
	PROBABLE	87	49.7

The possible diagnoses category has been reduced into the absent category.

Figure 7.8 Distribution of the diagnoses for all the joint conditions studied within this sample population by site category. The possible diagnoses category has been reduced into the absent category. Count is indicated within the bars.

Table 7.12 Distribution of the diagnoses for all the joint conditions studied within this sample population by biological sex.

BIOLOGICAL SEX	DIAGNOSIS	N	%
MALE	ABSENT	48	46.6
	PROBABLE	55	53.4
FEMALE	ABSENT	40	55.6
	PROBABLE	32	44.4

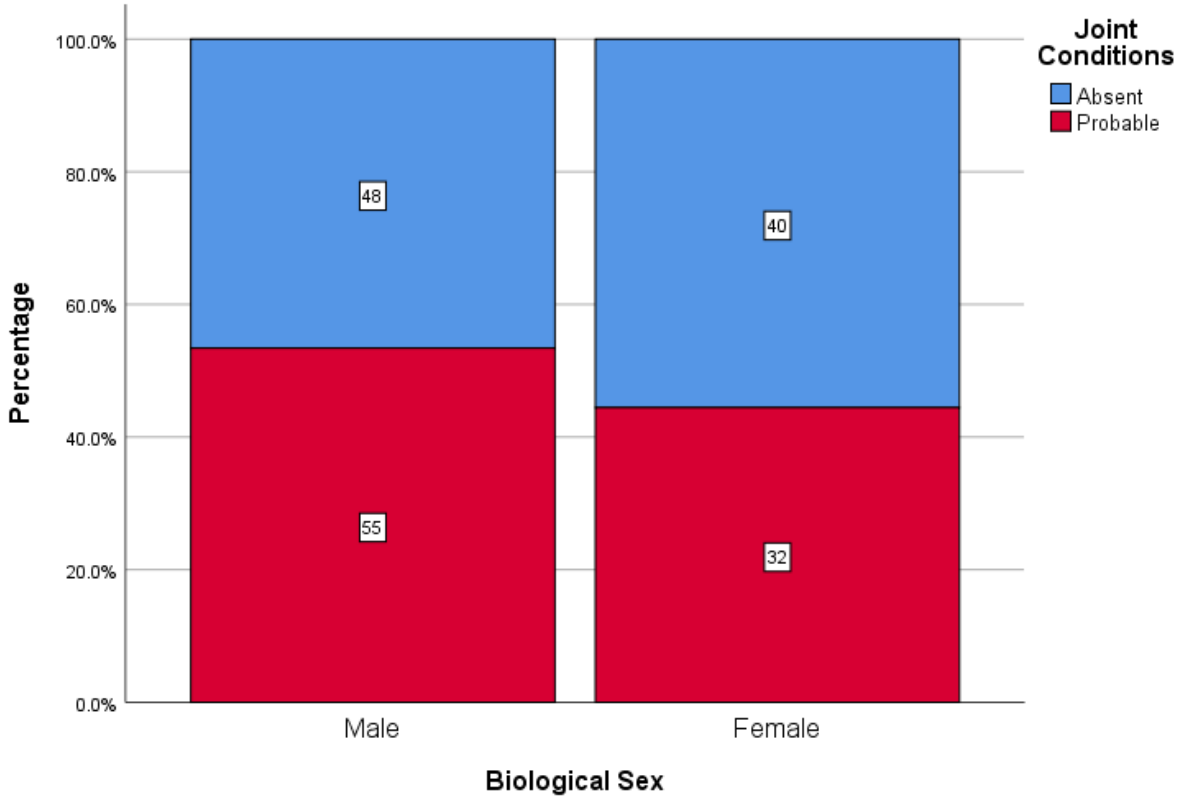

Figure 7.9 Distribution of all the joint conditions by biological sex. Count is indicated within the boxes.

Further break down of joint condition by age at death showed that early adults displayed a prevalence of 35.7% (n = 20) (see Table 7.13 and Table 7.14/ Figure 7.10 and Figure 7.11), 32.5% (n = 13) for middle adults and 68.4% (n = 54) were late adults. For the males, 39.3% (n = 11) of the early adults displayed a probable prevalence, 31.8% (n = 7) for middle adults and 69.8% (n = 37) for late adults. For the females, 32.1% (n = 9) early adults displayed a probable prevalence, 33.3% (n = 6) for middle adults, and late adults were 65.4% (n = 17).

Thus overall, while the distribution of probable diagnoses across site category and biological sex is relatively equal, cases are over-represented in the late adult category compared to both early and middle adult categories. Early adults tend to have slightly higher proportions

of probably cases than middle adults, and while the distinction is small compared to that between both these younger groups and the late adults, it is consistent between both males and females, and urban and rural groups. The exception to this is rural females, which have a more uniform prevalence for the early and middle age categories.

Table 7.13 Distribution of the diagnoses for all the joint conditions studied within this sample population by biological sex and age at death.

BIOLOGICAL SEX	AGE AT DEATH CATEGORY	ABSENT		PROBABLE	
		N	%	N	%
MALE	EARLY	17	60.7	11	39.3
	MIDDLE	15	68.2	7	31.8
	LATE	16	30.2	37	69.8
FEMALE	EARLY	19	67.9	9	32.1
	MIDDLE	12	66.7	6	33.3
	LATE	9	34.6	17	65.4
TOTAL	EARLY	36	64.3	20	35.7
	MIDDLE	27	67.5	13	32.5
	LATE	25	31.6	54	68.4

Note that values may not ≠ 100 as decimal point placement is rounded.

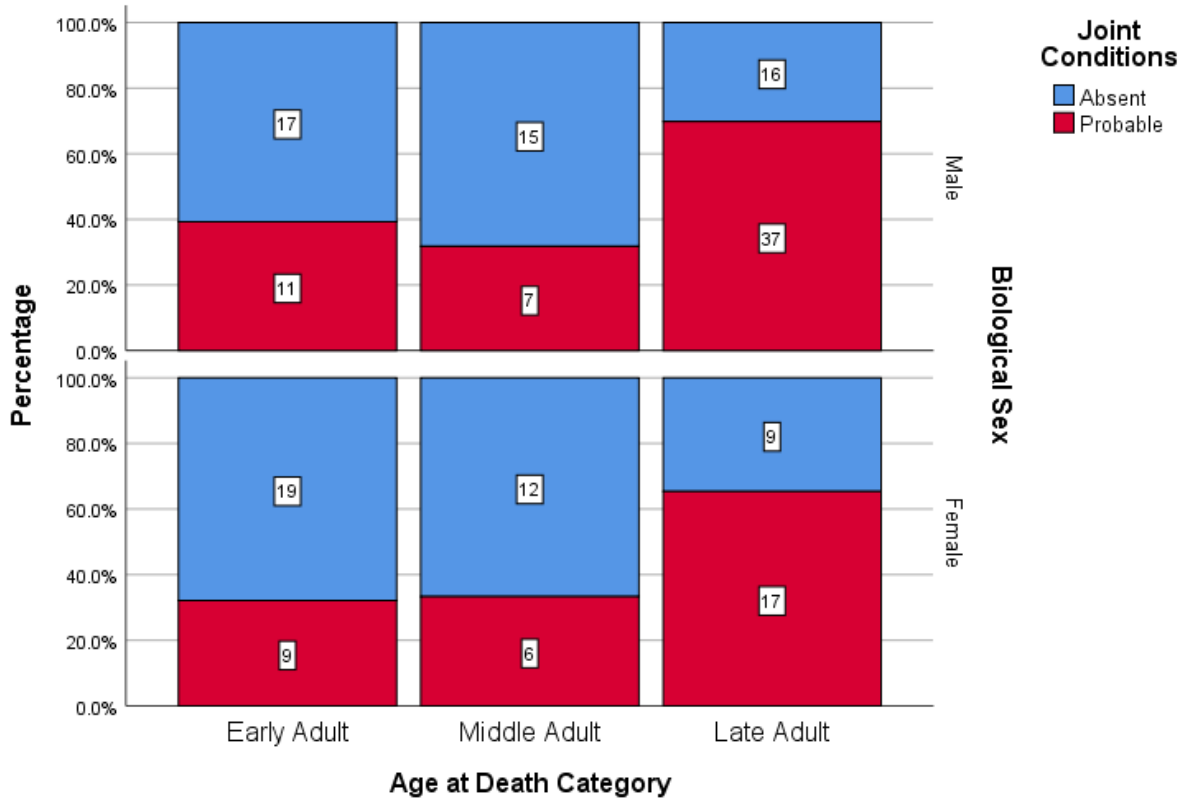

Figure 7.10 Proportional distribution of the diagnoses for all the joint conditions studied within this sample population by biological sex and age at death. Count is indicated in the bars.

Table 7.14 Distribution of the diagnoses for all the joint conditions studied within this sample population by site category and age at death.

BIOLOGICAL SEX	AGE AT DEATH CATEGORY	ABSENT		PROBABLE	
		N	%	N	%
URBAN	EARLY	22	68.8	10	31.3
	MIDDLE	13	61.9	8	38.1
	LATE	11	29.7	26	70.3
RURAL	EARLY	14	58.3	10	41.7
	MIDDLE	14	73.7	5	26.3
	LATE	14	33.3	28	66.7
TOTAL	EARLY	36	64.3	20	35.7
	MIDDLE	27	67.5	13	32.5
	LATE	25	31.6	54	68.4

Note that values may not ≠ 100 as decimal point placement is rounded.

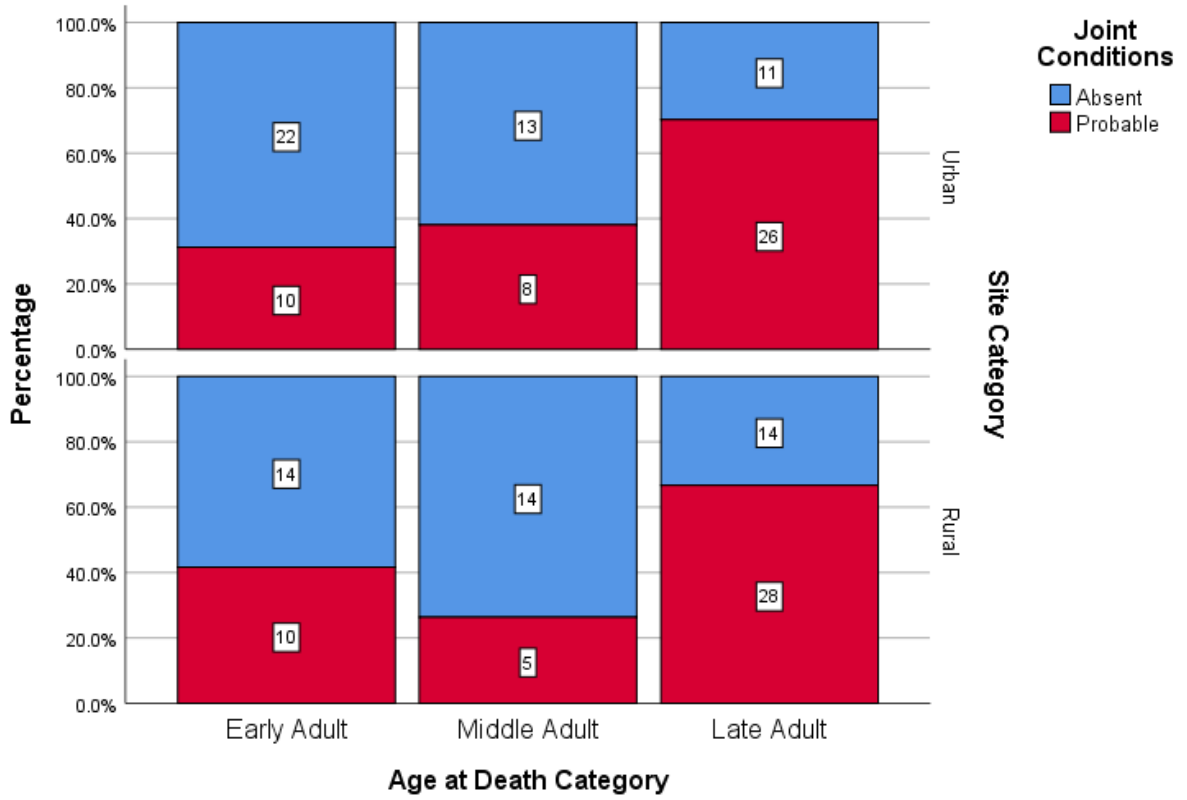

Figure 7.11 Distribution of the diagnoses for all the joint conditions studied within this sample population by site category, biological sex, and age at death. Count is indicated within the bars.

The diagnoses will for each joint condition will be explained in more detail in the following sections. Table 7.15 displays all of the joint conditions with the expanded diagnoses before reduction of possible into the absent category. The possible diagnoses may be cases of joint condition, however, due to either lack of definitive evidence or the presence of differential diagnoses, these possible diagnoses cannot offer any substantive value to the following evaluations.

Table 7.15 Diagnoses of the joint conditions researched within this study with possible diagnoses included.

		ABSENT		POSSIBLE		PROBABLE	
		N	%	N	%	N	%
JOINT CONDITIONS	OA	100	57.1	15	8.6	60	34.3
	SOA	120	71.4	7	4.2	41	24.4
	HOA	136	77.7	17	9.7	22	12.6
	KOA	148	86.0	7	4.1	17	9.9
	AS	149	90.9	15	9.1	0	0.0
	SI	152	93.3	0	0.0	11	6.7
	DDD	107	63.7	32	19.0	29	17.3

Note that values may not ≠ 100 as decimal point placement is rounded.

In summation, all cases of the joint conditions studied accounted for roughly half of the sample population. Both site categories, urban and rural, had a near even split of roughly 50% between absent and probable diagnoses. The distribution between males and females was also remarkably similar, with males presenting a slightly higher prevalence. Joint condition was found to be substantially more common in the late adult age category for the population as a whole and all sub-groups, with one exception. Urban females had a uniform distribution across the age categories, with the proportion of individuals with joint condition in the late adult category being only slightly larger than those in the younger age categories.

7.3.1 Osteoarthritis (OA)

Probable cases of osteoarthritis were observed in 34.3% (n = 60) of the total sample population (see Table 7.16/ Figure 7.12). These cases were spread throughout the joints assessed within this research: the spine, hip, knee. The patterns of OA in these joints are discussed separately below (see Section 7.3.2-4), but the following section combines them to reflect an overall prevalence of OA. Within the site categories, 30.0% (n = 27) of the urban population and 38.8% (n = 33) of the rural population were diagnosed as probable cases of OA. A total of 36.9% (n = 38) of the male sample population and 30.6% (n = 22) of the female sample population were diagnosed as probable cases of OA (see Table 7.17/ Figure 7.13).

Table 7.16 Distribution of the diagnoses for osteoarthritis within this sample population by site category.

SITE CATEGORY	DIAGNOSIS	N	%
URBAN	ABSENT	63	70.0
	PROBABLE	27	30.0
RURAL	ABSENT	52	61.2
	PROBABLE	33	38.8
TOTAL	ABSENT	115	65.7
	PROBABLE	60	34.3

Note that values may not ≠ 100 as decimal point placement is rounded.

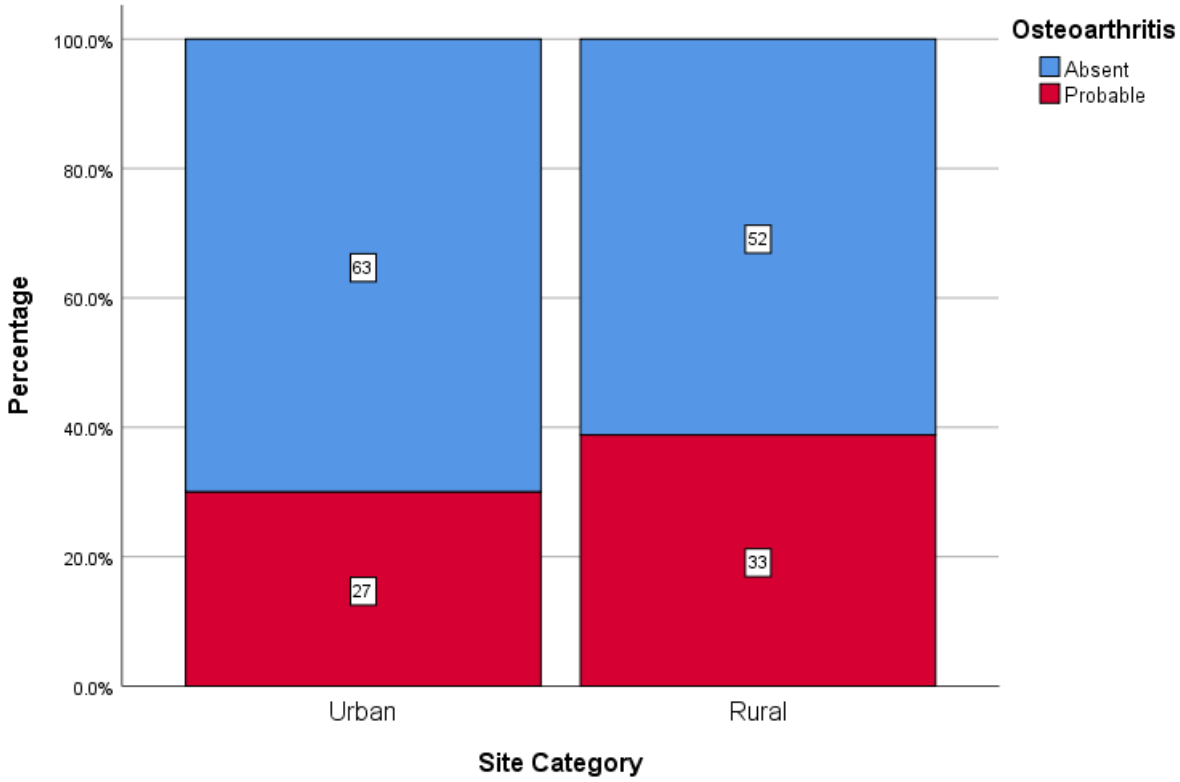

Figure 7.12 Distribution of the diagnoses for general osteoarthritis studied within this sample population by site category. Count is indicated within the bars.

Table 7.17 Distribution of the diagnoses for osteoarthritis within this sample population by biological sex.

BIOLOGICAL SEX	DIAGNOSIS	N	%
MALE	ABSENT	65	63.1
	PROBABLE	38	36.9
FEMALE	ABSENT	50	69.4
	PROBABLE	22	30.6

Note that values may not ≠ 100 as decimal point placement is rounded.

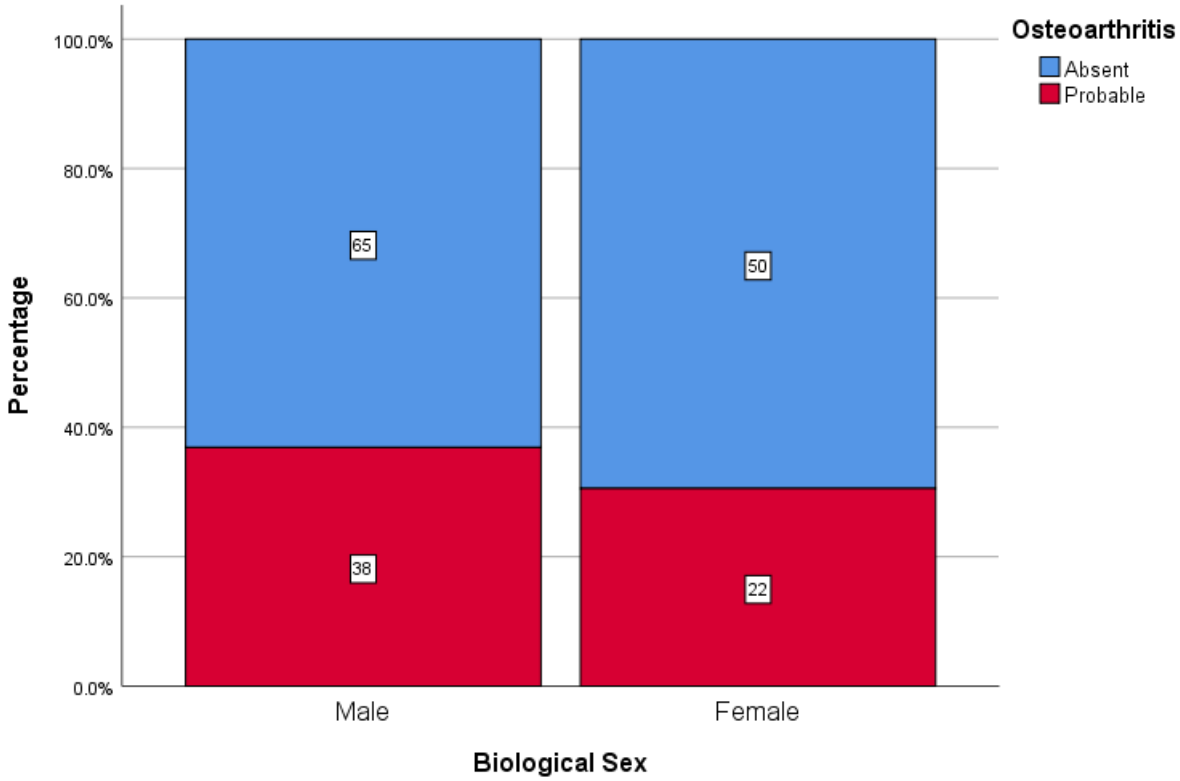

Figure 7.13 Distribution of the diagnoses for general osteoarthritis studied within this sample population by biological sex. Count is indicated within the bars.

Rural males and females had the higher proportion of osteoarthritis than their urban counterparts with prevalences of 42.6% (n = 20) and 34.2% (n = 13) respectively (see Table 7.17/ Figure 7.14). The urban males had a prevalence of 32.1% (n = 18) and urban females 26.5% (n = 9). Both males and females had the higher proportions of osteoarthritis in the late adult category (see Table 7.19/ Figure 7.15). Of the female individuals with a diagnosis of OA, a total of 22.7% (n = 5) fell within the early adult category, 18.18% (n = 4) fell within the middle adult category, and 59.1% (n = 13) fell within the late adult categories. Of the male individuals, 15.8% (n = 6) fell within the early adult category, 13.2% (n = 5) fell within the middle adult category, and 71.1% (n = 27) fell within the late adult category.

Table 7.18 Distribution of the diagnoses for osteoarthritis within this sample population by site category and biological sex.

			URBAN		RURAL	
FEMALE			N	%	N	%
	DIAGNOSIS	ABSENT	25	73.5	25	65.8
		PRESENT	9	26.5	13	34.2
			URBAN		RURAL	
MALE			N	%	N	%
	DIAGNOSIS	ABSENT	38	67.9	27	57.5
		PRESENT	18	32.1	20	42.6

Percentages are by subcategory (urban male, urban female, rural male, rural female). Note that values may not ≠ 100 as decimal point placement is rounded.

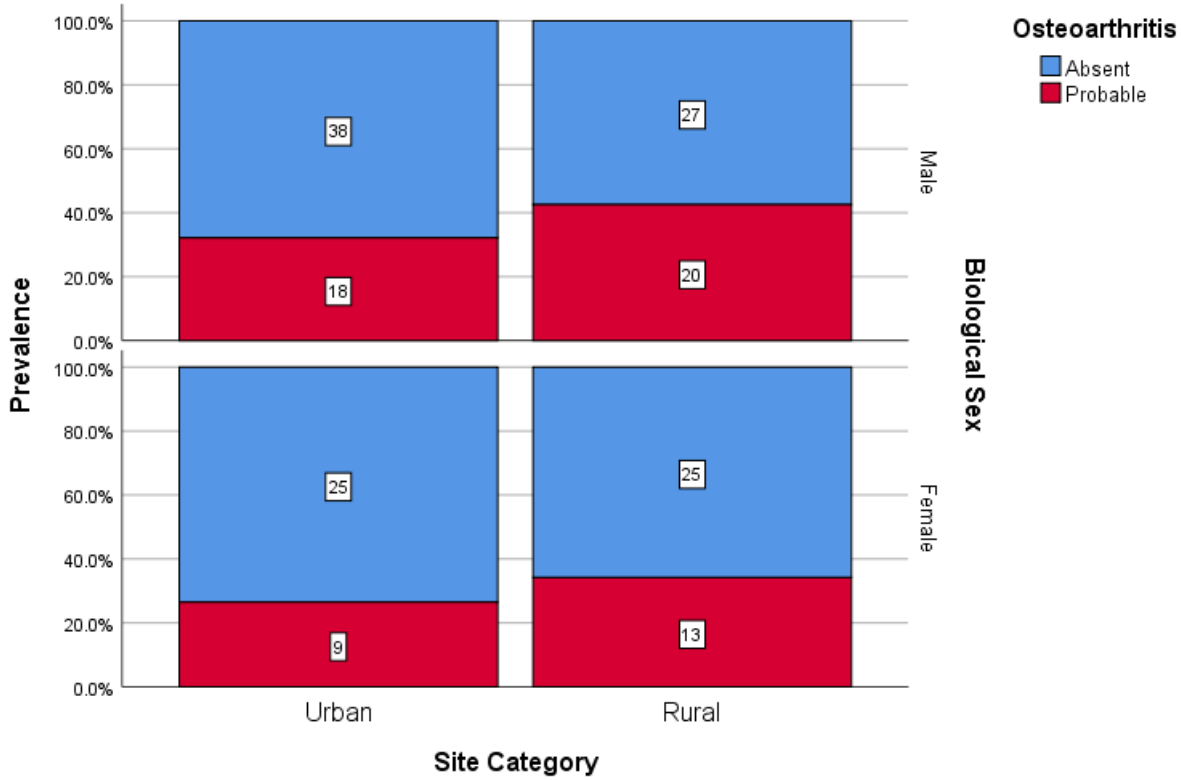

Figure 7.14 Distribution of the diagnoses for general osteoarthritis studied within this sample population by site category and biological sex. Count is indicated within the bars.

Table 7.19 Distribution of the diagnoses for osteoarthritis within this sample population by biological sex and age at death.

			ABSENT		PROBABLE	
			N	%	N	%
FEMALE	AGE AT DEATH CATEGORIES	EARLY	23	82.1	5	17.9
		MIDDLE	14	77.8	4	22.2
		LATE	13	50.0	13	50.0
			ABSENT		PROBABLE	
			N	%	N	%
MALE	AGE AT DEATH CATEGORIES	EARLY	22	78.6	6	21.4
		MIDDLE	17	77.3	5	22.7
		LATE	26	49.1%	27	50.9

Note that values may not ≠ 100 as decimal point placement is rounded.

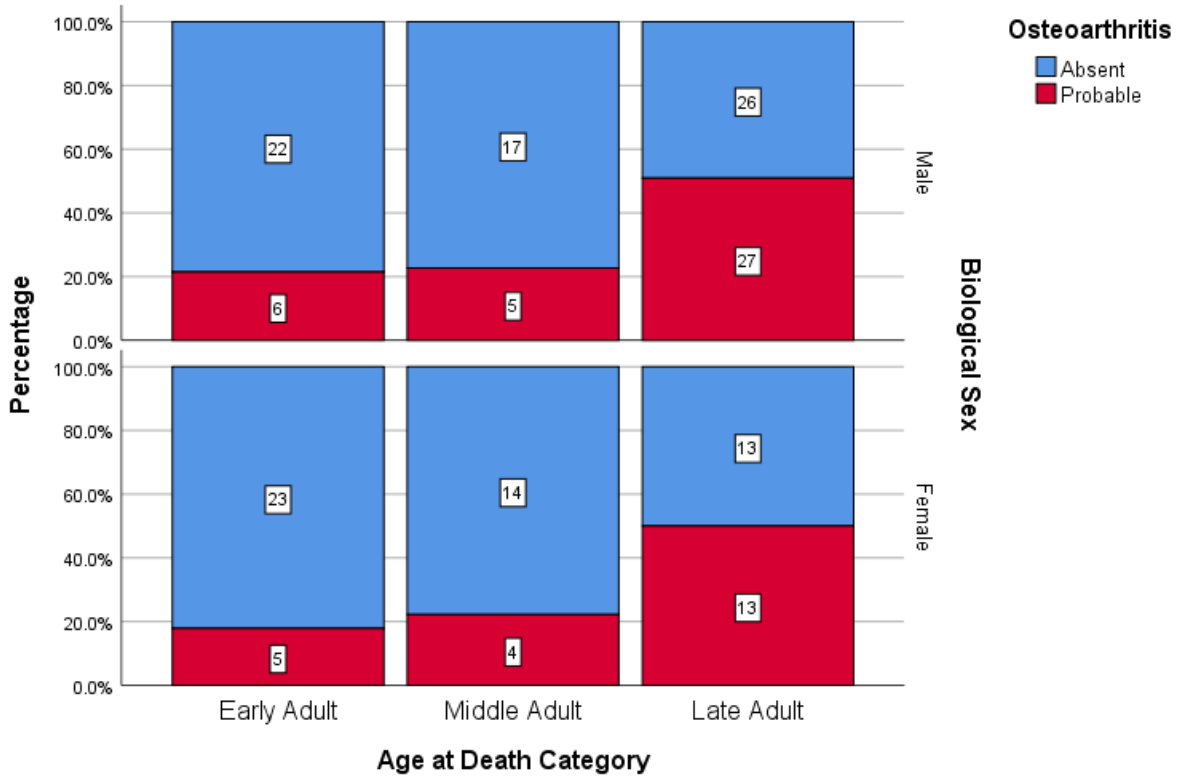

Figure 7.15 Proportional distribution of the diagnoses for osteoarthritis within this sample population by biological sex and age at death. Count is indicated within the bars.

Urban males displayed probable prevalences for OA at 11.8% (n = 2) within the early adults, 18.2% (n = 2) and the late category was 50.0% (n = 14) (see Table 7.19/ Figure 7.16). In stark contrast, the urban females displayed a more uniform distribution of probable diagnoses with early adults having a prevalence of 26.7% (n = 4), middle adults 30.0% (n = 3) and late adults 22.2% (n = 2). Early adult rural males displayed a prevalence of 36.4% (n = 4)

and middle adults at 27.3% (n = 3) and late adults at 52.0% (n = 13). Early adult rural females displayed a prevalence of 7.7% (n = 1), middle adults at 12.5% (n = 1), and late adults at 64.7% (n = 11). The overall distribution of probable diagnoses favors the late adults; however, the urban females did not follow that trend and instead had the lowest prevalence for late adults.

Table 7.20 Distribution of the diagnoses for osteoarthritis within this sample population by site category, biological sex, and age at death.

				ABSENT		PROBABLE	
				N	%	N	%
URBAN	FEMALE	AGE AT DEATH CATEGORIES	EARLY	11	73.3	4	26.7
			MIDDLE	7	70.0	3	30.0
			LATE	7	77.8	2	22.2
				ABSENT		PROBABLE	
				N	%	N	%
	MALE	AGE AT DEATH CATEGORIES	EARLY	15	88.2	2	11.8
			MIDDLE	9	81.8	2	18.2
			LATE	14	50.0	14	50.0
RURAL				ABSENT		PROBABLE	
				N	%	N	%
	FEMALE	AGE AT DEATH CATEGORIES	EARLY	12	92.3	1	7.7
			MIDDLE	7	87.5	1	12.5
			LATE	6	35.3	11	64.7
				ABSENT		PROBABLE	
				N	%	N	%
	MALE	AGE AT DEATH CATEGORIES	EARLY	7	63.6	4	36.4
			MIDDLE	8	72.7	3	27.3
			LATE	12	48.0	13	52.0

Note that values may not ≠ 100 as decimal point placement is rounded. Percentages are within each subcategory of age.

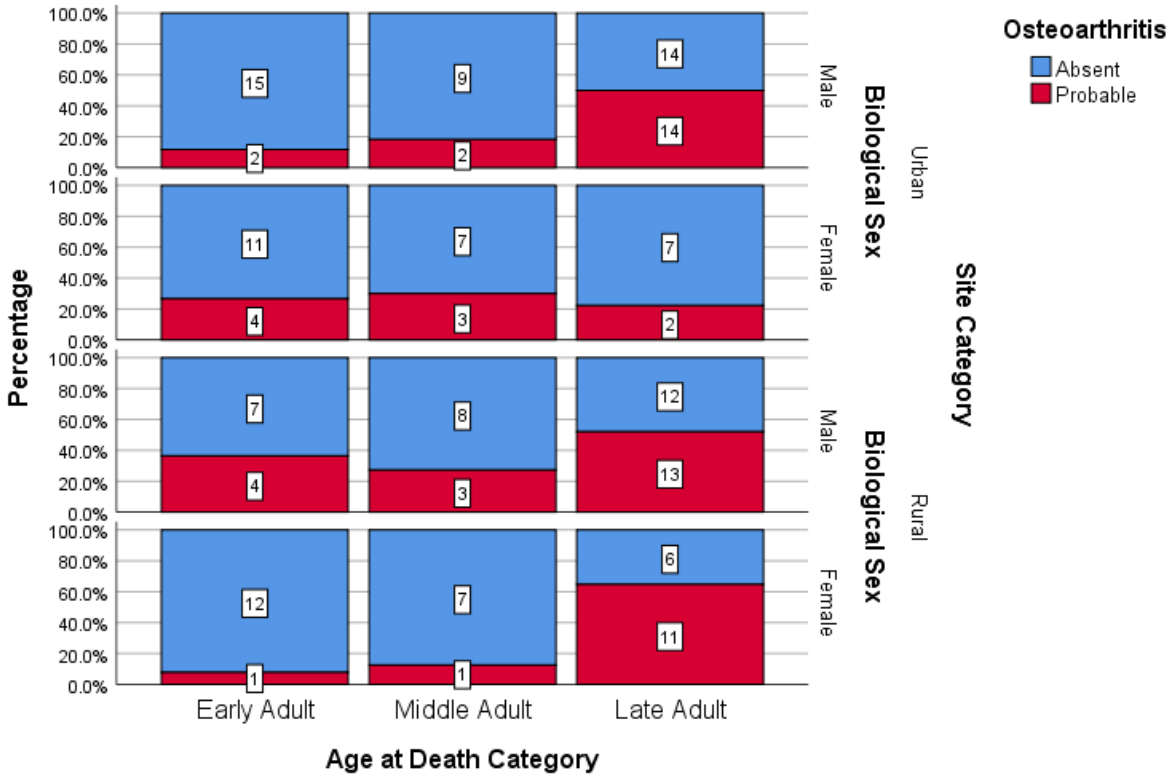

Figure 7.16 Proportional distribution of the diagnoses for osteoarthritis within this sample population by site category, biological sex, and age at death. Count is indicated within the bars.

In summation, osteoarthritis was present in one third of the sample population. Distribution between populations, was slightly higher among the rural group, and between the sexes was slightly higher among males. Late adults had the higher proportion of osteoarthritis, however, once more, urban females displayed a distinct distribution across the age at death categories. In this case, probable cases of OA decreased with advancing age and among late adult urban females alone, substantially more individuals did not show OA than did.

7.3.2 Spinal Osteoarthritis (SOA)

Overall, 24.4% (n = 41) of the total sample population displayed enough criteria for a probable diagnosis of spinal OA (see Table 7.21/ Figure 7.17). Individuals with a probable diagnosis constituted 19.3% (n=16) of the urban population and 29.4% (n=25) of the rural population. Males (25.5%, n = 25) displayed a higher proportion of probable diagnoses than females (22.9%, n = 16) within the population; however, the margin between the sexes is smaller than the other OA categories (see Table 7.22/ Figure 7.18).

Table 7.21 Distribution of the diagnoses for spinal osteoarthritis within this sample population by site category.

SITE CATEGORY	DIAGNOSIS	N	%
URBAN	ABSENT	67	80.7
	PROBABLE	16	19.3
RURAL	ABSENT	60	70.6
	PROBABLE	25	29.4
TOTAL	ABSENT	127	75.6
	PROBABLE	41	24.4

Note that values may not ≠ 100 as decimal point placement is rounded.

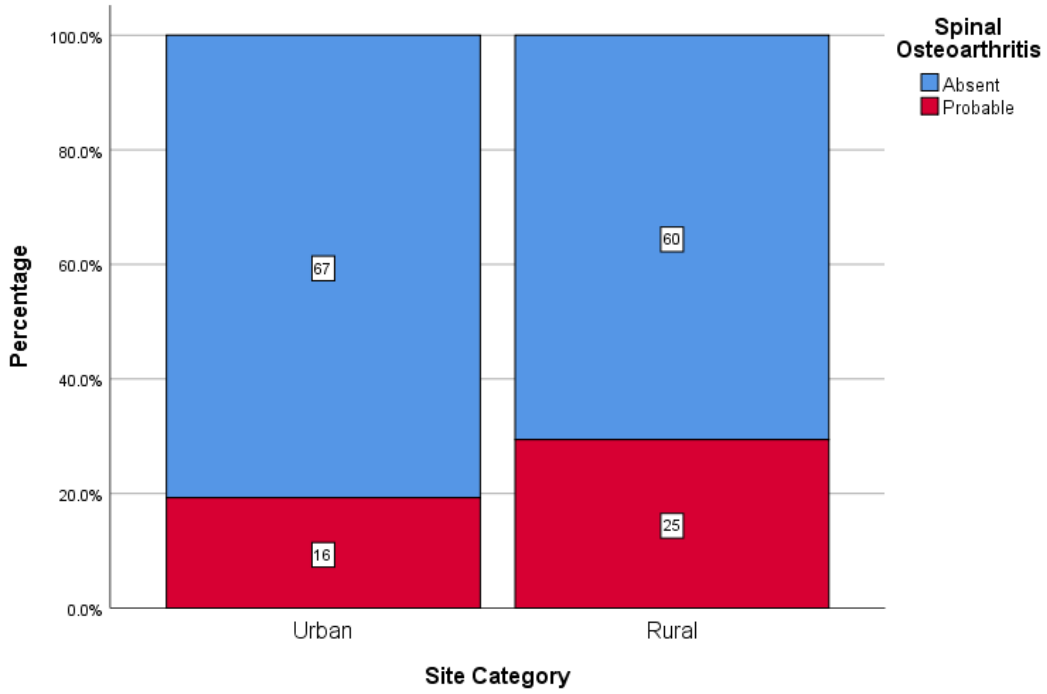

Figure 7.17 Distribution of the diagnoses for spinal osteoarthritis studied within this sample population by site category Count is indicated within the bars.

Table 7.22 Distribution of the diagnoses for spinal osteoarthritis within this sample population by biological sex.

BIOLOGICAL SEX	DIAGNOSIS	N	%
MALE	ABSENT	73	74.5
	PROBABLE	25	25.5
FEMALE	ABSENT	54	77.1
	PROBABLE	16	22.9

Note that values may not ≠ 100 as decimal point placement is rounded.

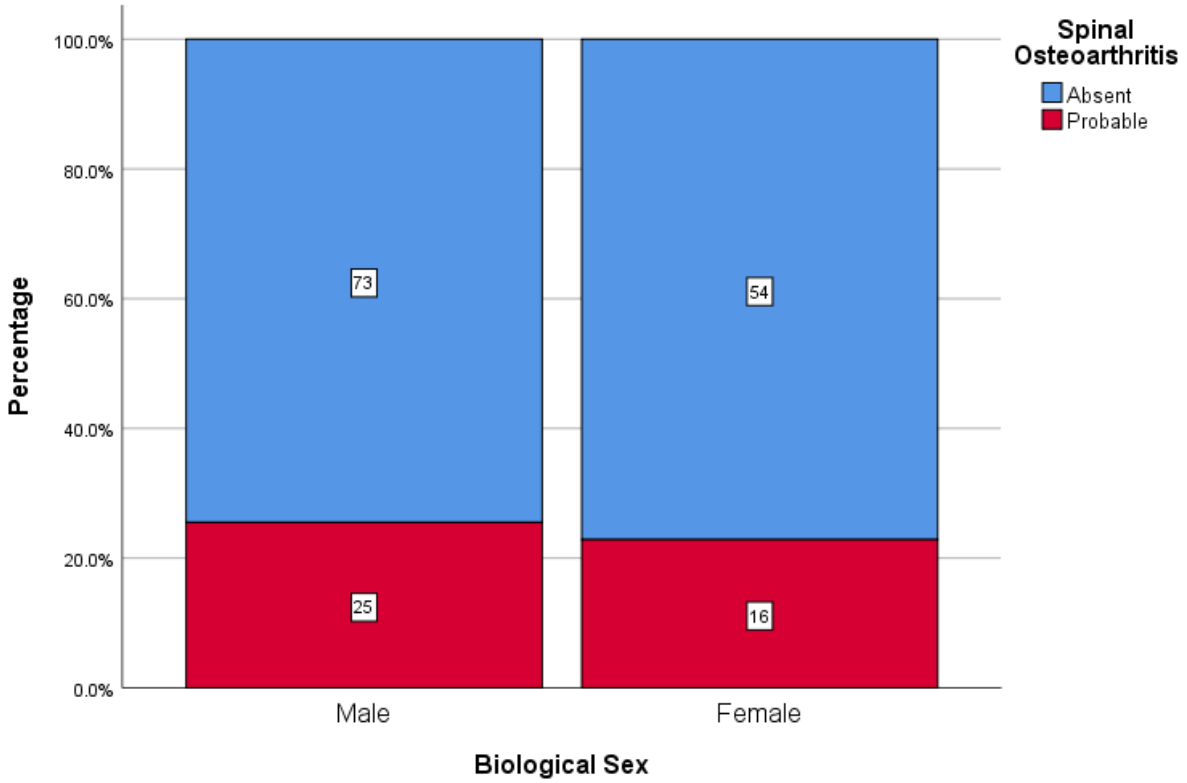

Figure 7.18 Distribution of the diagnoses for spinal osteoarthritis studied within this sample population by biological sex. Count is indicated within the bars.

Rural females and males had higher proportions of probable diagnoses of spinal osteoarthritis than urban female and male populations (see Table 7.23/ Figure 7.19): 29.0% (n = 11) of rural females and 29.8% (n = 14) of rural males, compared with 15.6% (n = 5) of urban females and 21.6% (n = 11) of urban males presented with probable diagnoses of spinal osteoarthritis. Both the males and females showed a gradual increase of SOA as age increased (see Table 7.24/ Figure 7.20). Of the female population with a probable diagnosis of spinal osteoarthritis, 10.7% (n = 3) fell within the early adult category, 15.8% (n = 3) fell within the middle adult category, and 23.8.0% (n = 10) fell within the late adult category. Of the total male population with a probable diagnosis of spinal osteoarthritis, 10.7% (n = 3) fell within the early adult category, 20.0% (n = 4) fell within the middle adult category, and 36.0% (n = 18) fell within the early adult category.

Table 7.23 Distribution of the diagnoses for spinal osteoarthritis within this sample population by site category and biological sex.

			URBAN		RURAL	
			N	%	N	%
FEMALE	DIAGNOSIS	ABSENT	27	84.4	27	71.1
		PRESENT	5	15.6	11	29.0
			URBAN		RURAL	
			N	%	N	%
MALE	DIAGNOSIS	ABSENT	40	85.7	27	80.9
		PRESENT	14	14.3	11	19.2

Percentages are by subcategory (urban male, urban female, rural male, rural female). Note that values may not ≠ 100 as decimal point placement is rounded.

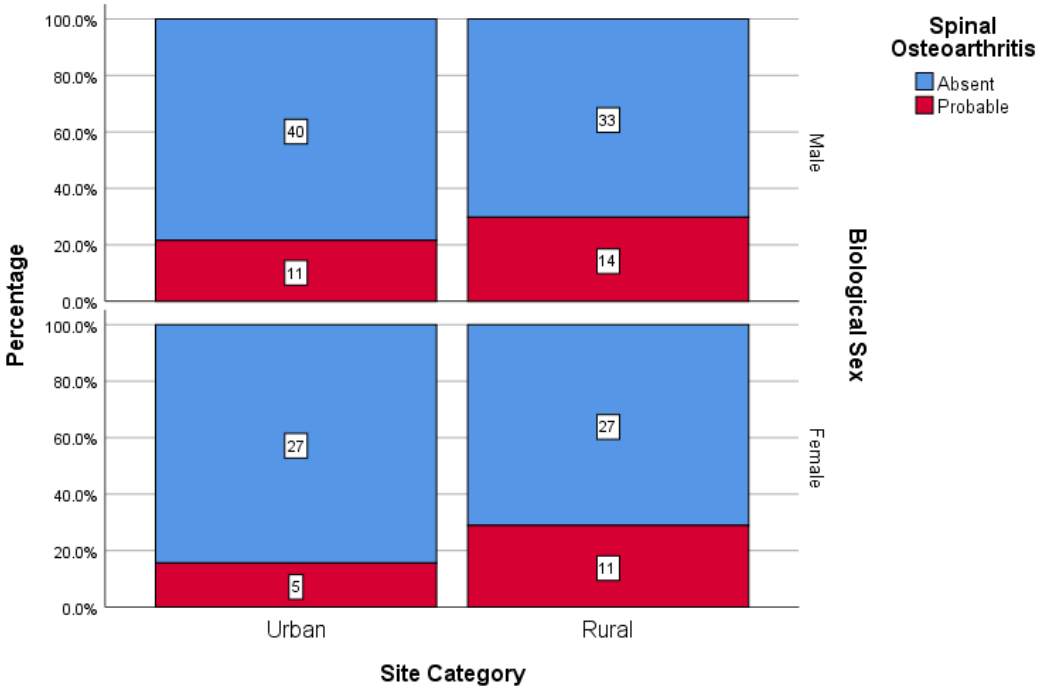

Figure 7.19 Distribution of the diagnoses for spinal osteoarthritis studied within this sample population by site type and biological sex. Count is indicated within the bars.

Table 7.24 Distribution of the diagnoses for spinal osteoarthritis within this sample population by biological sex and age at death category.

			ABSENT		PROBABLE	
			N	%	N	%
FEMALE	AGE AT DEATH CATEGORIES	EARLY	25	89.3	3	10.7
		MIDDLE	16	84.2	3	15.8
		LATE	32	76.2	10	23.8
			ABSENT		PROBABLE	
			N	%	N	%
MALE	AGE AT DEATH CATEGORIES	EARLY	25	89.3	3	10.7
		MIDDLE	16	80.0	4	20.0
		LATE	32	64.0	18	36.0

Note that values may not ≠ 100 as decimal point placement is rounded.

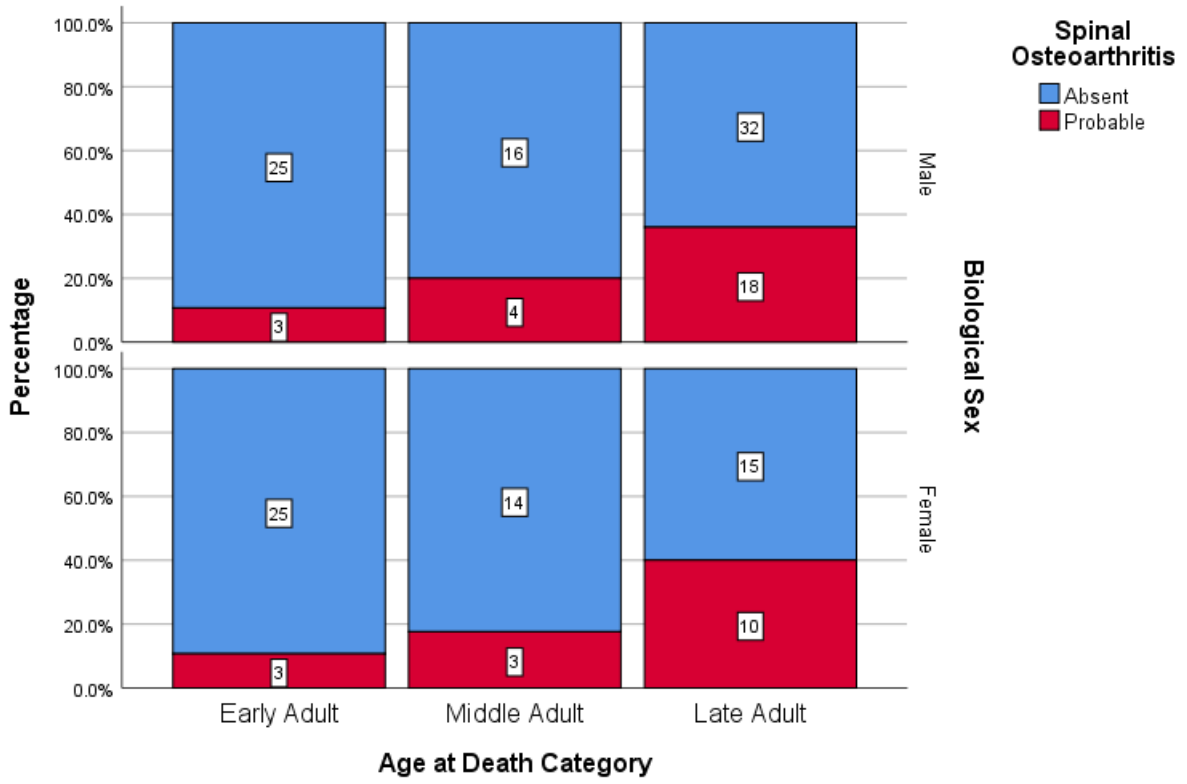

Figure 7.20 Proportional distribution of the diagnoses for spinal osteoarthritis within this sample population by biological sex and age at death. Count is indicated within the box.

Urban males displayed a prevalence of 5.9% (n = 1) for early adults, 11.1% (n = 1) for middle adults and 36.0% (n = 9) for late adults (see Table 7.25/ Figure 7.21). Rural males displayed a prevalence of 18.2% (n = 2) for early adults, 27.3% (n = 3) for middle adults and 36.0% (n = 9) for late adults. Urban females displayed a prevalence of 13.3% (n = 2) for early adults, 22.2% (n = 2) middle adults and 12.5% (n = 1) for late adults. Rural females displayed

similar proportions to urban males with 7.7% (n = 1) for early adults, 12.5% (n = 1) middle adults and 52.9% (n = 9) for late adults. The urban females have continued their unique trend with a uniform distribution across the age categories and late adults having a lower proportion of spinal OA than younger adults.

Table 7.25 Distribution of the diagnoses for spinal osteoarthritis within this sample population by site category, biological sex, and age at death.

				ABSENT		PROBABLE	
				N	%	N	%
URBAN	FEMALE	AGE AT DEATH CATEGORIES	EARLY	13	86.7	2	13.3
			MIDDLE	7	77.8	2	22.2
			LATE	7	87.5	1	12.5
				ABSENT		PROBABLE	
				N	%	N	%
	MALE	AGE AT DEATH CATEGORIES	EARLY	16	94.1	1	5.9
			MIDDLE	8	88.9	1	11.1
			LATE	16	64.0	9	36.0
RURAL				ABSENT		PROBABLE	
				N	%	N	%
	FEMALE	AGE AT DEATH CATEGORIES	EARLY	12	92.3	1	7.7
			MIDDLE	7	87.5	1	12.5
			LATE	8	47.1	9	52.9
				ABSENT		PROBABLE	
				N	%	N	%
	MALE	AGE AT DEATH CATEGORIES	EARLY	9	81.8	2	18.2
			MIDDLE	8	72.7	3	27.3
			LATE	16	64.0	9	36.0

Note that values may not ≠ 100 as decimal point placement is rounded. Percentages are within each subcategory of age.

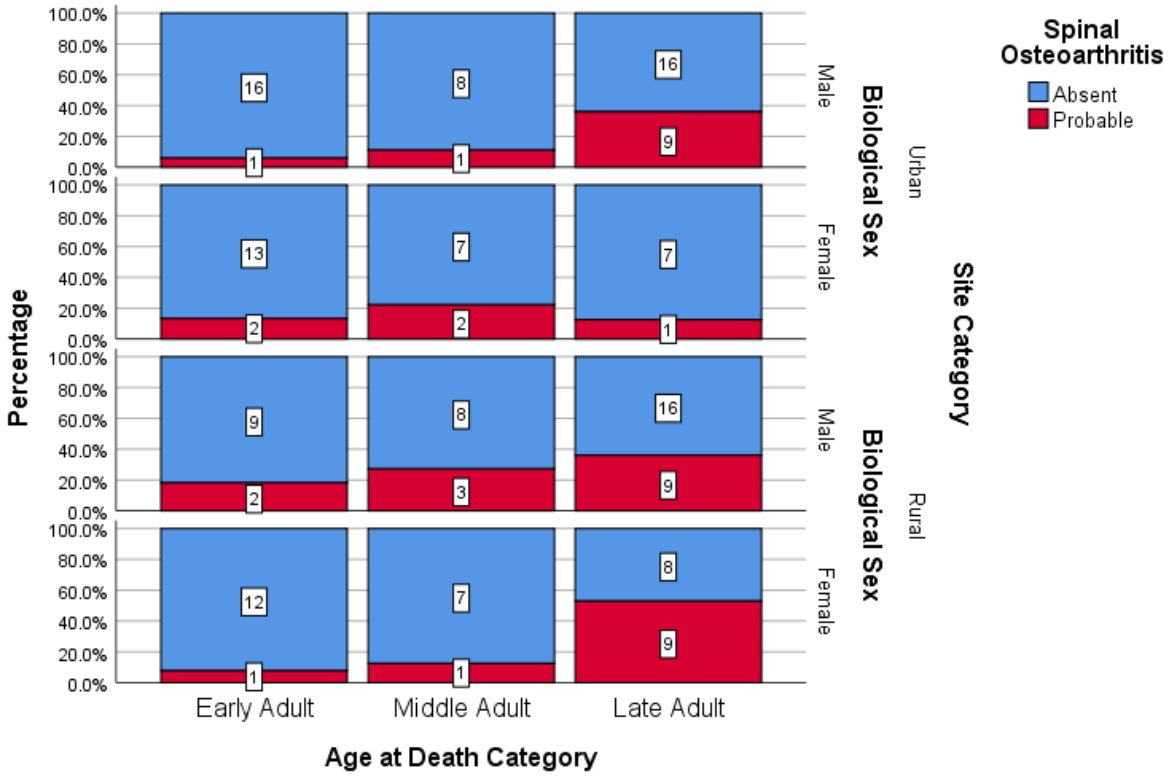

Figure 7.21 Distribution of the diagnoses for spinal osteoarthritis within this sample population by site category, biological sex, and age at death. Count is indicated within the bars.

In summation, spinal osteoarthritis was present in roughly one quarter of the sample population. The condition was found in one fifth of the urban population and almost one third of the rural population, therefore substantially more common in the latter overall. Males and females had a similar distribution and roughly one quarter of each group had spinal osteoarthritis. Rural males had a higher proportion of individuals with the condition than the urban cohort and the females were similar between both groups. Spinal osteoarthritis was found to be more common in late adults, however, urban females once again presented a different distribution across the age categories.

7.3.3 Hip Osteoarthritis (HOA)

Probable cases of hip osteoarthritis were observed in 12.6% (n = 22) of the sample population (see Table 7.26/ Figure 7.22). The prevalence of hip OA was found to be similar for both the urban and rural populations with 12.2%/12.9% (n = 11 each). Males (n =19, 17.9%) had a larger proportion of probable diagnoses for hip osteoarthritis than females (n = 5, 6.2%). Of the male sample population, 16.5% (n = 19) were diagnosed with probable hip OA and

83.5% (n = 85) did not meet the criteria for diagnosis (see Table 7.27/ Figure 7.23). Of the female sample population, 6.9% (n = 5) were diagnosed with probable hip OA and the final 93.1% (n = 67) did not meet the criteria for a diagnosis.

Table 7.26 Distribution of the diagnoses for hip osteoarthritis within this sample population by site category.

SITE CATEGORY	DIAGNOSIS	N	%
URBAN	ABSENT	79	87.8
	PROBABLE	11	12.2
RURAL	ABSENT	74	87.1
	PROBABLE	11	12.9
TOTAL	ABSENT	153	87.4
	PROBABLE	22	12.6

Note that values may not ≠ 100 as decimal point placement is rounded.

Figure 7.22 Distribution of the diagnoses for hip osteoarthritis within this sample population by site category and biological sex. Count is indicated within the bars.

Table 7.27 Distribution of the diagnoses for hip osteoarthritis within this sample population by site category.

BIOLOGICAL SEX	DIAGNOSIS	N	%
MALE	ABSENT	86	83.5
	PROBABLE	17	16.5
FEMALE	ABSENT	67	93.1
	PROBABLE	5	6.9

Note that values may not ≠ 100 as decimal point placement is rounded.

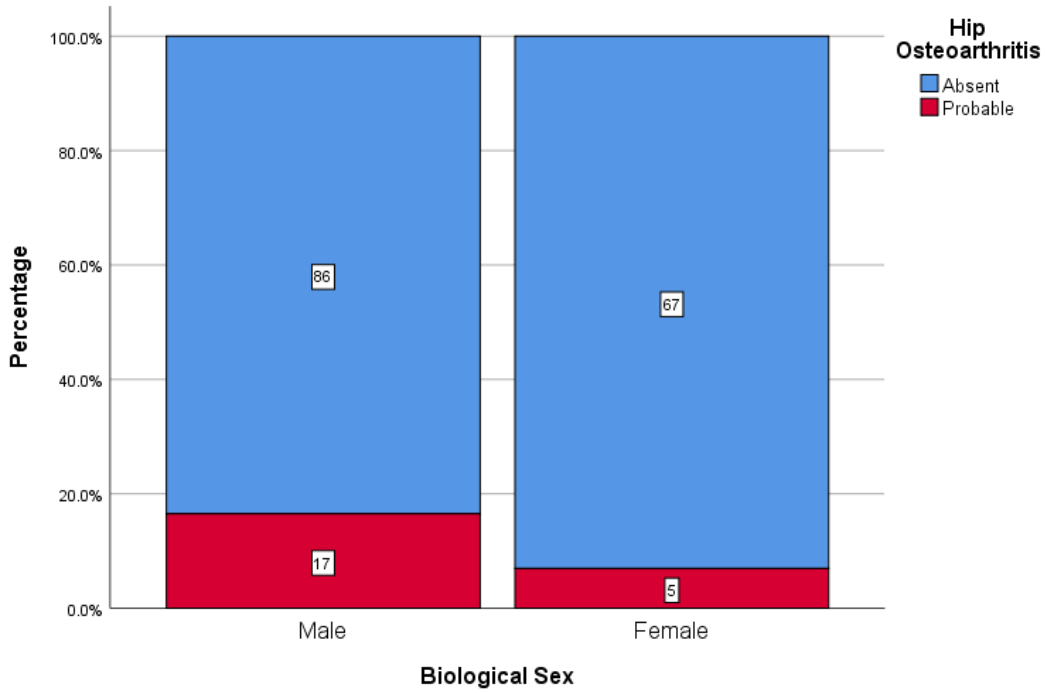

Figure 7.23 Distribution of the diagnoses for hip osteoarthritis studied within this sample population by biological sex. The possible diagnoses category has been reduced into the absent category. Count is indicated within the bars.

The distribution of probable diagnoses by biological sex across the site categories display similar prevalence rates (see Table 7.28/ Figure 7.24), however, the males displayed higher prevalence rates across the site categories. The urban males displayed a prevalence 14.3% (n = 8) and the females displayed a lower rate of 8.8% (n = 3). The rural males displayed a prevalence of 19.2% (n = 9) and the females displayed a lower rate of 5.3% (n = 2). Of the female population with a probable diagnosis of hip osteoarthritis, 3.6% (n = 1) fell within the early adult category, 5.6% (n = 1) fell within the middle adult category, and 11.5% (n = 3) fell within the late adult category (see Table 7.29/ Figure 7.25). Of the male population with a probable diagnosis of hip osteoarthritis, 10.7% (n = 3) fell within the early adult category, 4.5% (n = 1) fell within the middle adult category, 24.5% (n = 13) fell within the late adult category.

Table 7.28 Distribution of the diagnoses for hip osteoarthritis within this sample population by biological sex.

			URBAN		RURAL	
FEMALE			N	%	N	%
	DIAGNOSIS	ABSENT	31	91.2	36	94.7
		PRESENT	3	8.8	2	5.3
			URBAN		RURAL	
MALE			N	%	N	%
	DIAGNOSIS	ABSENT	48	85.7	38	80.9
		PRESENT	8	14.3	9	19.2

Note that values may not ≠ 100 as decimal point placement is rounded.

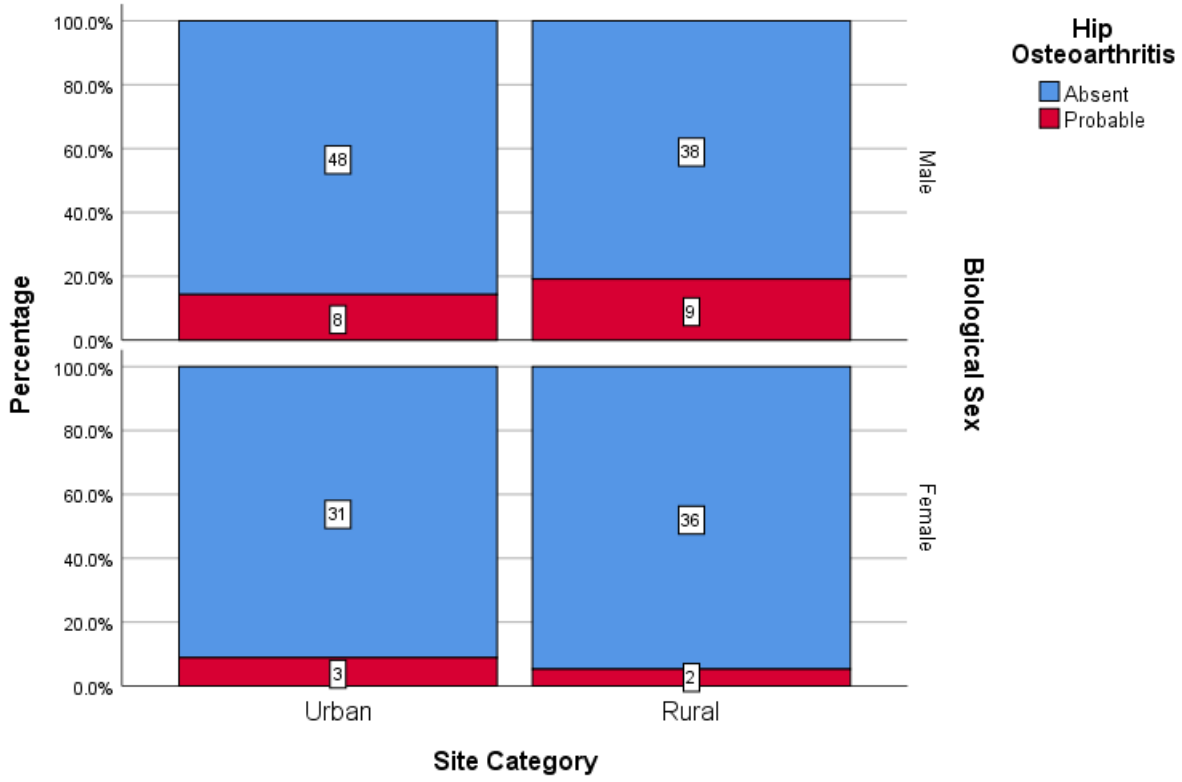

Figure 7.24 Distribution of the diagnoses for hip osteoarthritis studied within this sample population by site category. The possible diagnoses category has been reduced into the absent category. Count is indicated within the bars.

Table 7.29 Distribution of the diagnoses for hip osteoarthritis within this sample population by biological sex and age at death.

			ABSENT		PROBABLE	
			N	%	N	%
FEMALE	AGE AT DEATH CATEGORIES	EARLY	27	96.4	1	3.6
		MIDDLE	17	94.4	1	5.6
		LATE	23	88.5	3	11.5
			ABSENT		PROBABLE	
			N	%	N	%
MALE	AGE AT DEATH CATEGORIES	EARLY	25	89.3	3	10.7
		MIDDLE	21	95.5	1	4.5
		LATE	40	75.5	13	24.5

Note that values may not ≠ 100 as decimal point placement is rounded.

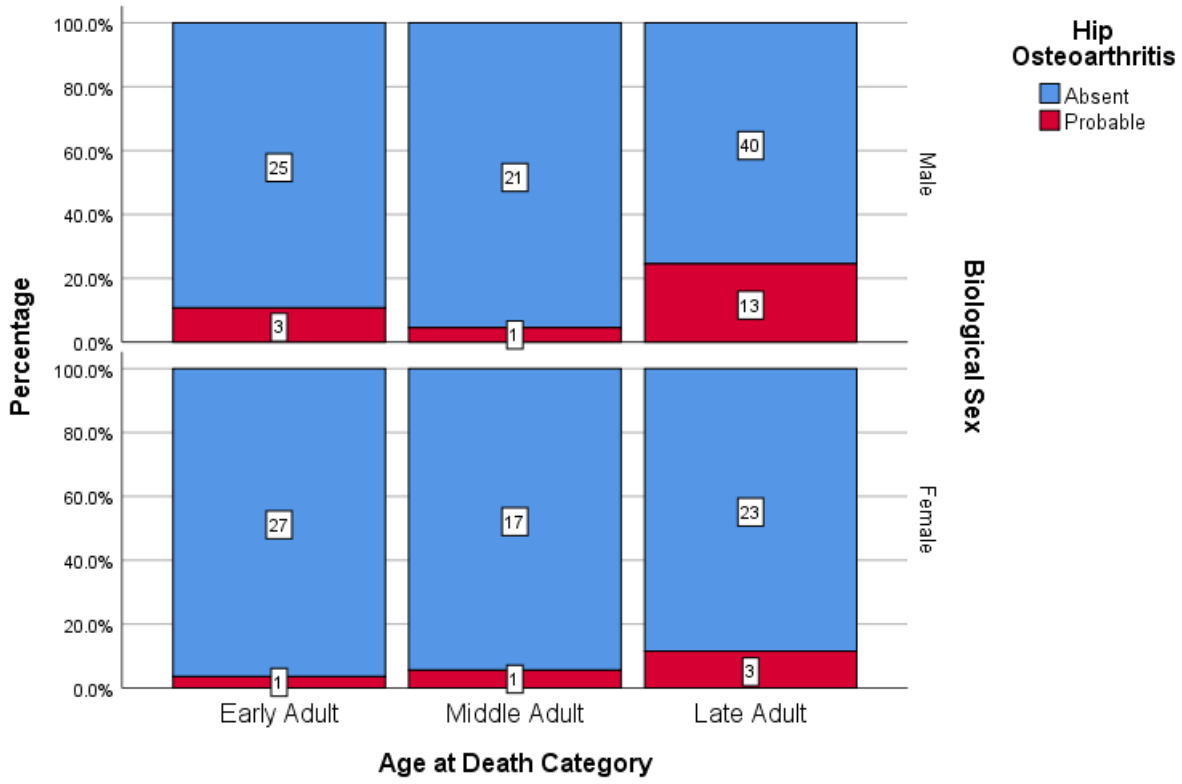

Figure 7.25 Distribution of the diagnoses for hip osteoarthritis within this sample population by biological sex and age at death. Count is indicated within the bars.

Urban males exhibited a prevalence of 5.7% (n = 1) in early adults and 25% (n = 7) in late adults (see Table 7.30/ Figure 7.26). The urban females displayed similar prevalences across all age categories with 6.7% (n = 1) for early adults, 10.0% (n = 1) for middle adults, and 11.1% (n = 1) for late adults. The rural males displayed prevalences of18.2^% (n = 2) in early adults, 9.1% (n = 1) in middle adults, and 24.0% (n = 6) in late adults. Rural females did not display prevalences in the early or middle adult categories and 11.8% (n = 2) in the late adult category.

Table 7.30 Distribution of the diagnoses for hip osteoarthritis within this sample population by site category, biological sex, and age at death.

				ABSENT		PROBABLE	
				N	%	N	%
URBAN	FEMALE	AGE AT DEATH CATEGORIES	EARLY	14	93.3	1	6.7
			MIDDLE	9	90.0	1	10.0
			LATE	8	88.9	1	11.1
	MALE			ABSENT		PROBABLE	
				N	%	N	%
		AGE AT DEATH CATEGORIES	EARLY	16	94.1	1	5.9
			MIDDLE	11	100.0	0	0.0
			LATE	21	75.0	7	25.0
RURAL	FEMALE			ABSENT		PROBABLE	
				N	%	N	%
		AGE AT DEATH CATEGORIES	EARLY	13	100.0	0	0.0
			MIDDLE	8	100.0	0	0.0
			LATE	15	88.2	2	11.8
	MALE			ABSENT		PROBABLE	
				N	%	N	%
		AGE AT DEATH CATEGORIES	EARLY	9	81.8	2	18.2
			MIDDLE	10	90.9	1	9.1
			LATE	19	76.0	6	24.0

Note that values may not ≠ 100 as decimal point placement is rounded. Percentages are within each subcategory of age.

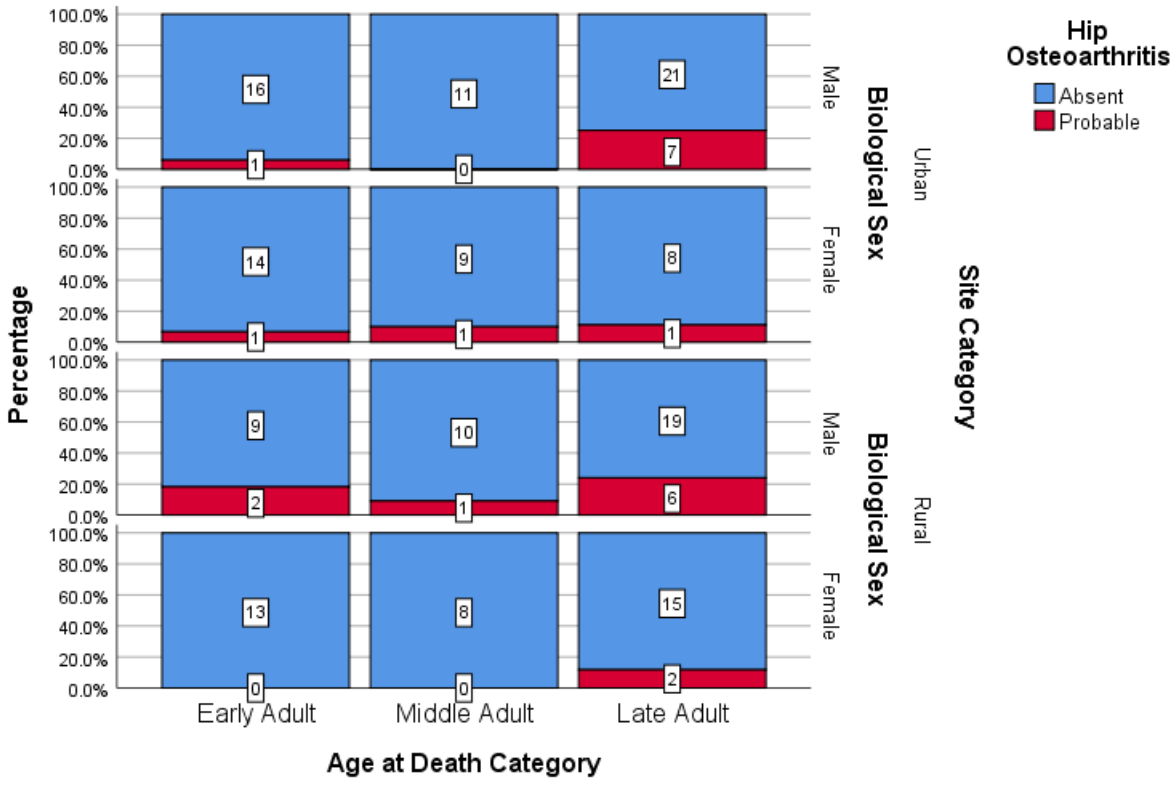

Figure 7.26 Distribution of the diagnoses for hip osteoarthritis within this sample population by site category, biological sex, and age at death. Count is indicated within the bars.

Hip osteoarthritis had a lower prevalence than the previous conditions at just over one tenth of the sample population. The site categories had similar proportions to the total sample. Males presented a higher proportion than females of probable diagnoses and late adults had the larger proportion that younger age categories. The exception to the latter, once again, was urban females however, the number of probable cases was too small to make any conclusive judgements about the reliability of this pattern.

7.3.4 Knee Osteoarthritis (KOA)

Probable cases of knee OA were observed in 9.9% (n = 17) of the total sample population (see Table 7.31/ Figure 7.27). The urban and rural populations displayed similar prevalences with 10.3%/9.4% (n = 9/ n = 8). Females had a slightly higher prevalence rate of hip OA at 12.7% (n = 9) than the males 7.9% (n =8) (see Table 7.32/ Figure 7.28).

Table 7.31 Distribution of the diagnoses for knee osteoarthritis within this sample population by site category.

SITE CATEGORY	DIAGNOSIS	N	%
URBAN	ABSENT	78	89.7
	PROBABLE	9	10.3
RURAL	ABSENT	77	90.6
	PROBABLE	8	9.4
TOTAL	ABSENT	155	90.1
	PROBABLE	17	9.9

Note that values may not ≠ 100 as decimal point placement is rounded.

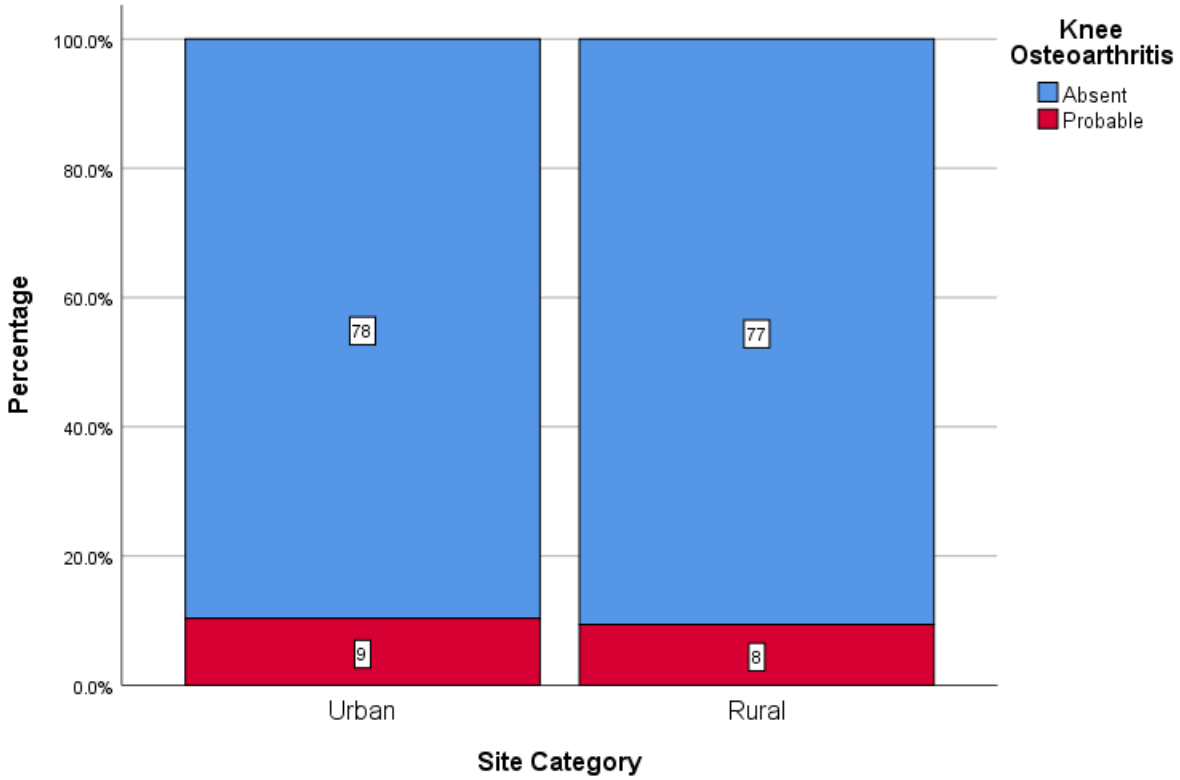

Figure 7.27 Distribution of the diagnoses for hip osteoarthritis within this sample population by site category. Count is indicated in the box.

Table 7.32 Distribution of the diagnoses for knee osteoarthritis within this sample population by biological sex.

BIOLOGICAL SEX	DIAGNOSIS	N	%
MALE	ABSENT	93	92.1
	PROBABLE	8	7.9
FEMALE	ABSENT	62	87.3
	PROBABLE	9	12.7

Note that values may not ≠ 100 as decimal point placement is rounded.

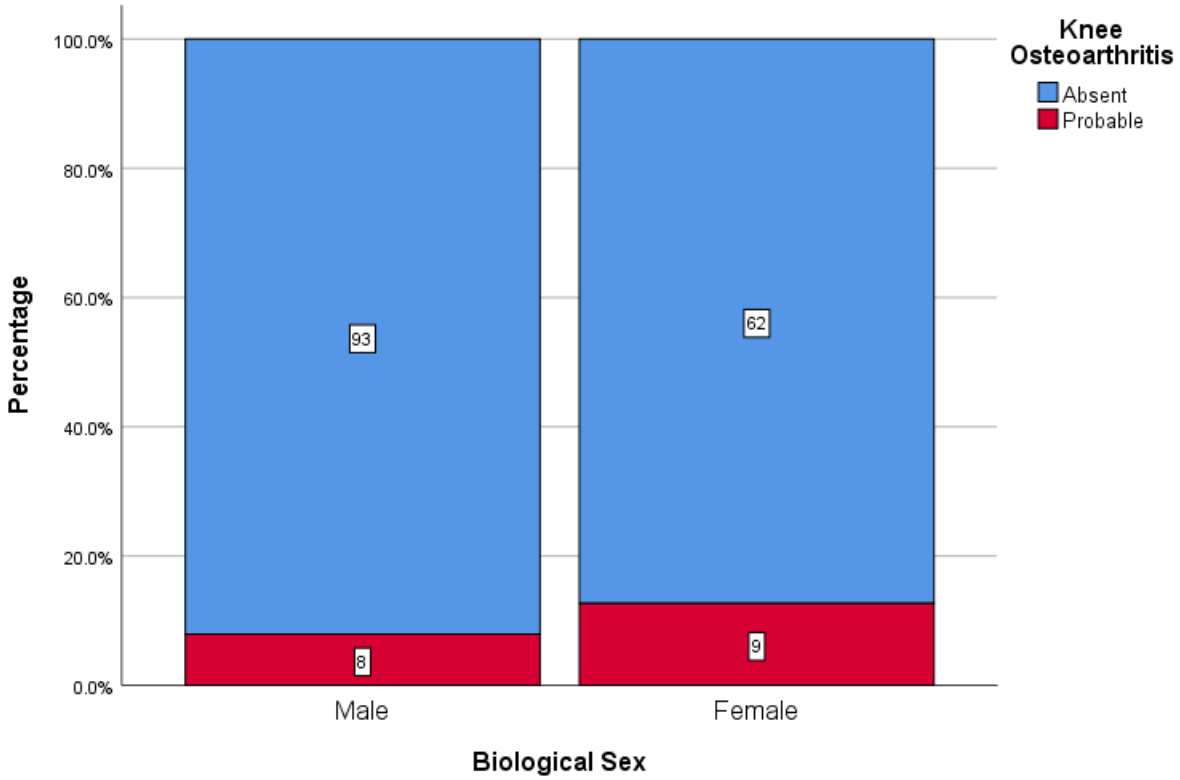

Figure 7.28 Distribution of the diagnoses for knee osteoarthritis within this sample population by biological sex. Count is indicated within the bars.

The female population had a similar prevalence, but the urban male population had a slightly higher prevalence than the rural male population (see Table 7.33/ Figure 7.29). Urban females displayed a prevalence of 12.1% (n = 4) and the males displayed a prevalence of 9.3% (n = 5). The rural female population displayed a prevalence two times larger (13.2%, n = 5) than the rural male population (6.4%, n = 3). Female individuals had a higher prevalence of knee arthritis in the early adult category, while male individuals had a higher prevalence in the late adult category, with the prevalence among the middle adult categories being roughly similar between the sexes (see Table 7.34/ Figure 7.30). Of the female population with a probable diagnosis of knee osteoarthritis 7.4% (n = 2) fell within the early adult category, 11.1% (n = 2) fell within the middle adult category, and 19.2% (n = 5) fell within the late adult category. Of the male population with a probable diagnosis of knee osteoarthritis, 3.6% (n = 1) fell within the early adult category, 4.5% (n = 1) fell within the middle adult category, and 27.3% (n = 6) fell within the late adult category.

Table 7.33 Distribution of the diagnoses for knee osteoarthritis within this sample population by biological sex.

			URBAN		RURAL	
			N	%	N	%
FEMALE	DIAGNOSIS	ABSENT	29	87.9	33	86.8
		PRESENT	4	12.1	5	13.2
			URBAN		RURAL	
			N	%	N	%
MALE	DIAGNOSIS	ABSENT	49	90.7	44	93.6
		PRESENT	5	9.3	3	6.4

Note that values may not ≠ 100 as decimal point placement is rounded.

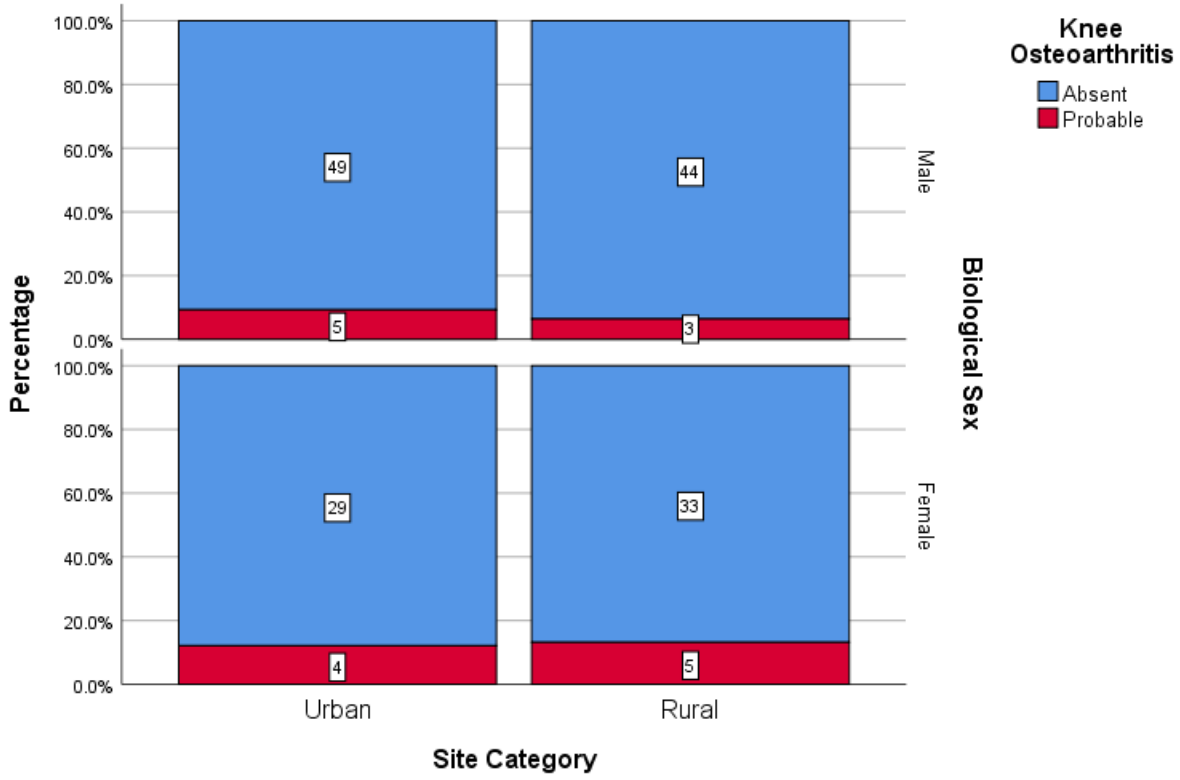

Figure 7.29 Distribution of the diagnoses for knee osteoarthritis studied within this sample population by site category. The possible diagnoses category has been reduced into the absent category. Count is indicated within the bars.

Table 7.34 Distribution of the diagnoses for hip osteoarthritis within this sample population by biological sex and age at death category.

			ABSENT		PROBABLE	
			N	%	N	%
FEMALE	AGE AT DEATH CATEGORIES	EARLY	25	92.6	2	7.4
		MIDDLE	16	88.9	2	11.1
		LATE	21	80.8	5	19.2
			ABSENT		PROBABLE	
			N	%	N	%
MALE	AGE AT DEATH CATEGORIES	EARLY	27	96.4	1	3.6
		MIDDLE	21	95.5	1	4.5
		LATE	16	72.7	6	27.3

Note that values may not ≠ 100 as decimal point placement is rounded.

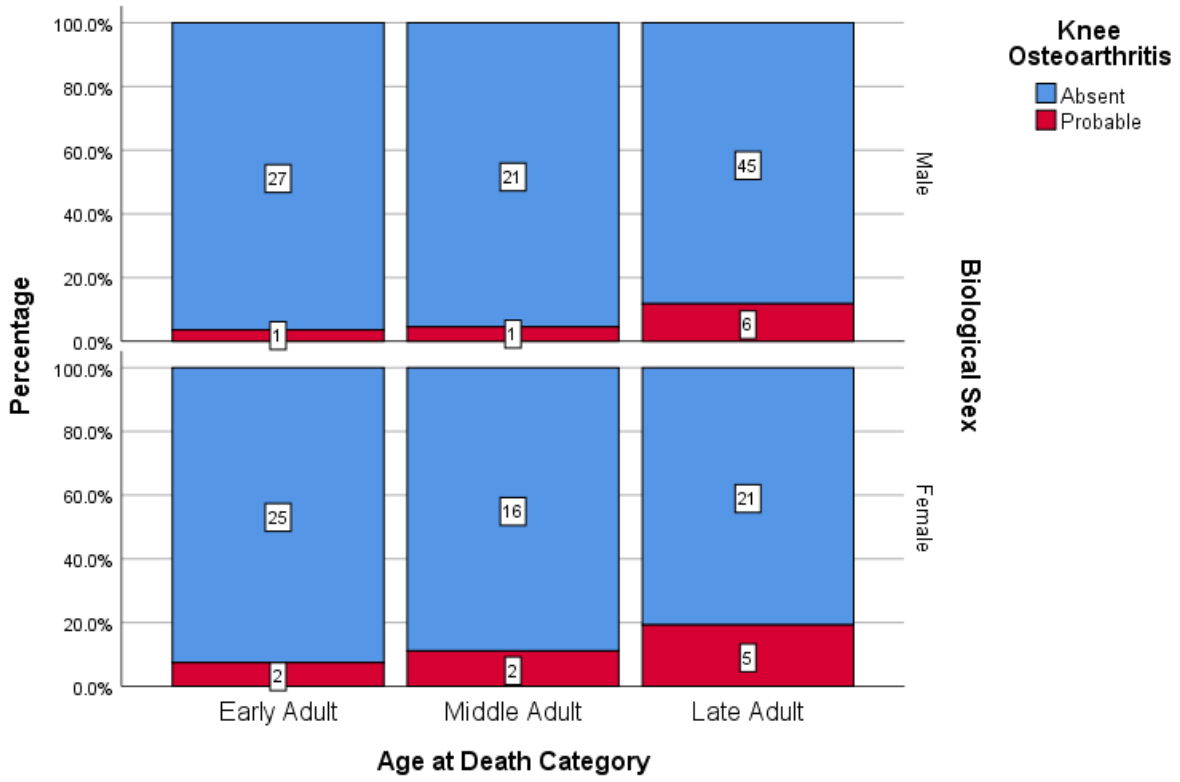

Figure 7.30 Distribution of the diagnoses for hip osteoarthritis within this sample population by biological sex and age at death. Count is indicated within the bars.

Urban males presented no cases of knee OA within early adults but had a prevalence of 9.1% (n = 1) in middle adults and 15.4% (n = 4) in late adults (see Table 7.34/ Figure 7.31). Rural males had no cases among middle adults and the remaining age at death categories displayed a prevalence of 9.1% (n = 1) for early adults and 8.0% (n = 2) for late adults. Urban females had a prevalence of 14.3% (n= 2) for early adults and 10.0% (n = 1) for middle and

11.1% (n = 1) for late adults. Rural females had no probable diagnoses for the early adult age category and the remaining age at death categories displayed a prevalence of 12.5% (n = 1) for middle adults and 23.5% (n = 4) for late adults. The trend seen among the other joint locations and disease categories for urban females to present a distinctive distribution was also evidenced in the knee OA data, with a slightly higher proportion in the early adults than middle or older adults.

Table 7.35 Distribution of the diagnoses for hip osteoarthritis within this sample population by site category, biological sex, and age at death.

				ABSENT		PROBABLE	
				N	%	N	%
URBAN	FEMALE	AGE AT DEATH CATEGORIES	EARLY	12	85.7	2	14.3
			MIDDLE	9	90.0	1	10.0
			LATE	8	88.9	1	11.1
	MALE			ABSENT		PROBABLE	
				N	%	N	%
		AGE AT DEATH CATEGORIES	EARLY	17	100.0	0	0.0
			MIDDLE	10	90.9	1	9.1
			LATE	22	84.6	4	15.4
RURAL	FEMALE			ABSENT		PROBABLE	
				N	%	N	%
		AGE AT DEATH CATEGORIES	EARLY	13	100.0	0	0.0
			MIDDLE	7	87.5	1	12.5
			LATE	13	76.5	4	23.5
	MALE			ABSENT		PROBABLE	
				N	%	N	%
		AGE AT DEATH CATEGORIES	EARLY	10	90.9	1	9.1
			MIDDLE	11	100.0	0	0.0
			LATE	23	92.0	2	8.0

Note that values may not ≠ 100 as decimal point placement is rounded. Percentages are within each subcategory of age.

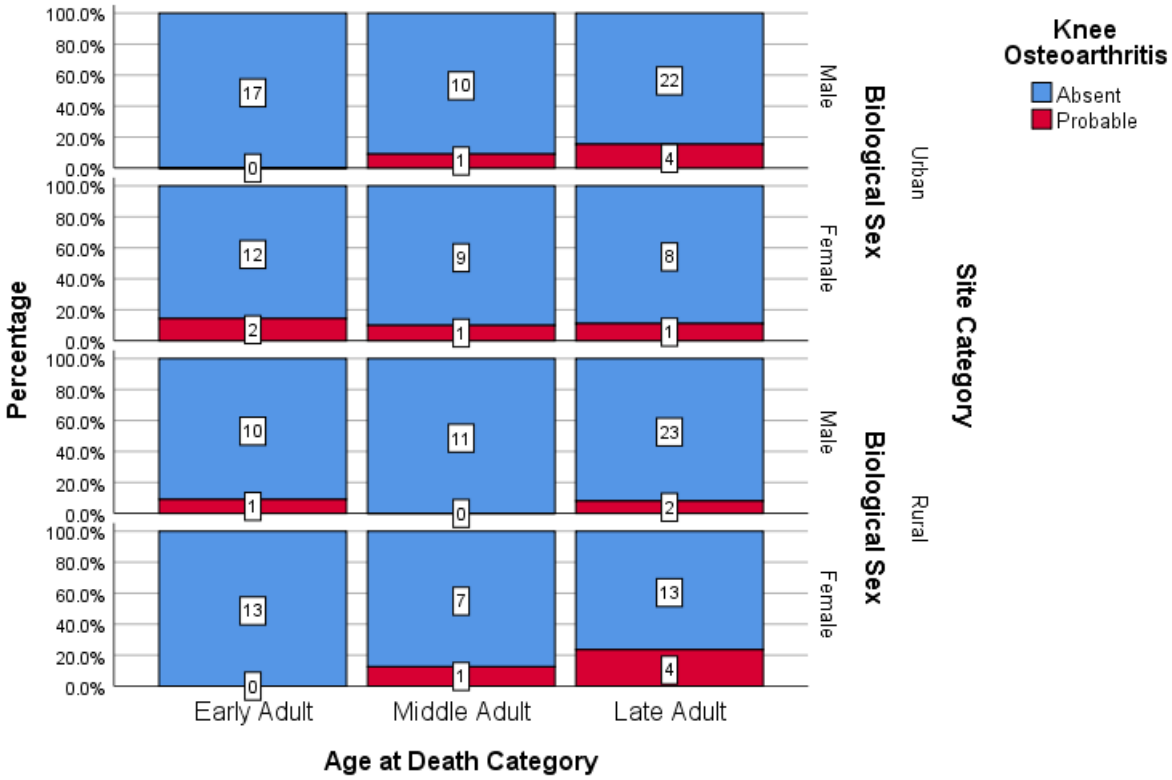

Figure 7.31 Distribution of the diagnoses for hip osteoarthritis within this sample population by site category, biological sex, and age at death. Count is indicated within the bars.

In summation, knee osteoarthritis was present in nearly one tenth of the population and the female population displayed a higher prevalence than the male population. Site category did not display differences for the females but did exhibit a difference between the males. Moreover, the difference between the rural females and males was larger (2:1) than with the urban females and males (4:3). Late adults had the highest prevalence rates of knee OA except for urban females which had a more uniform spread with early adults having a higher prevalence.

7.3.5 Ankylosing Spondylitis (AS)

No individuals were assessed having a probable diagnosis for ankylosing spondylitis (see Table 7.36/ Figure 7.31). However, 8.02% (n = 15) of the sample population were given a possible diagnosis due to having skeletal markers and evidence for the condition, but due to poor preservation of the remains and presence of possible alternative differential diagnoses, these were unable to be definitively diagnosed as present. A total of 12.3% (n = 13) of the male and 2.5% (n = 2) of the female individuals were afforded possible diagnoses (see Table 7.37/

Figure 7.32). Due to the lack of any probable diagnoses, following this section presenting the basic prevalence rates ankylosing spondylitis was not used for further analyses and testing.

Table 7.36 Distribution of the diagnoses for ankylosing spondylitis within this sample population by site category.

BIOLOGICAL SEX	DIAGNOSIS	N	%
URBAN	ABSENT	66	83.5
	POSSIBLE	13	16.5
RURAL	ABSENT	83	97.7
	POSSIBLE	2	2.4
TOTAL	ABSENT	149	90.9
	PROBABLE	15	9.2

Note that unlike the previous joint conditions, probable has been replaced with possible diagnoses. Note that values may not ≠ 100 as decimal point placement is rounded.

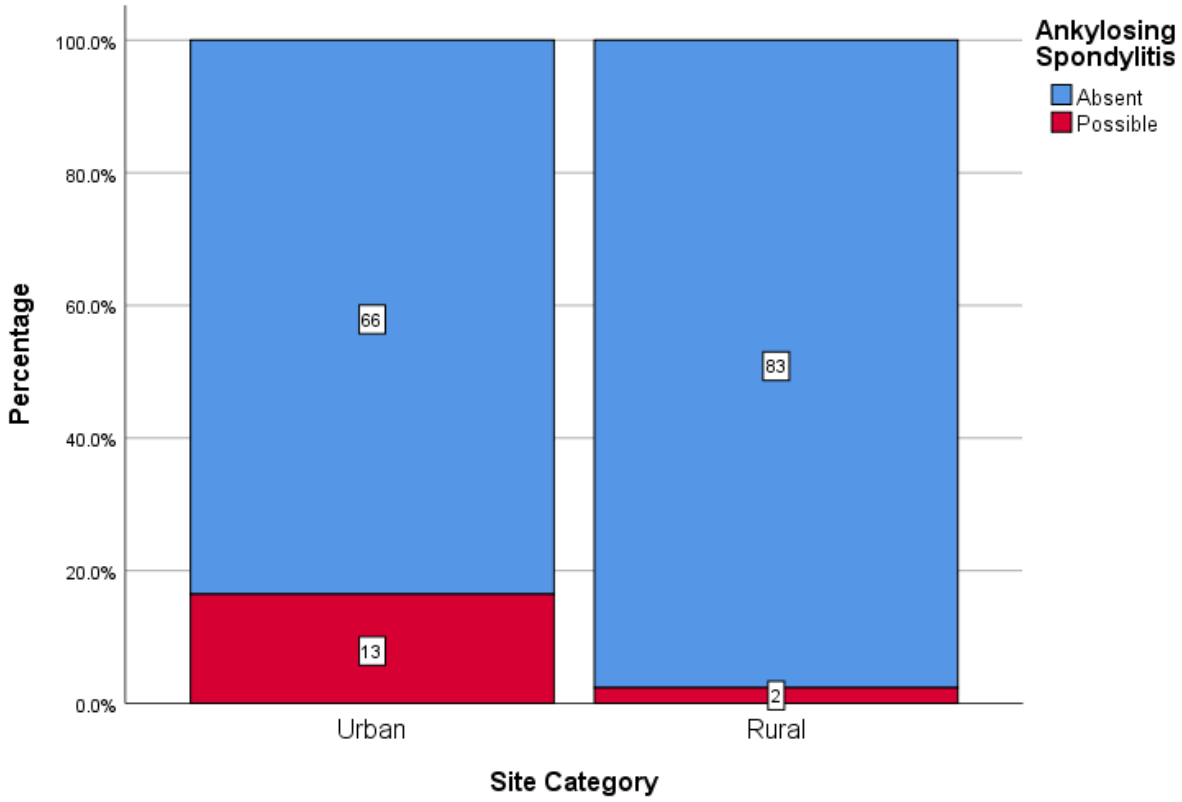

Figure 7.32 Distribution of the diagnoses for ankylosing spondylitis within this sample population by site category. Count is indicated within the bars.

Table 7.37 Distribution of the diagnoses for ankylosing spondylitis within this sample population by biological sex.

BIOLOGICAL SEX	DIAGNOSIS	N	%
MALE	ABSENT	83	86.5
	POSSIBLE	13	13.5
FEMALE	ABSENT	66	97.1
	POSSIBLE	2	2.9

Note that unlike the previous joint conditions, probable has been replaced with possible diagnoses. Note that values may not ≠ 100 as decimal point placement is rounded.

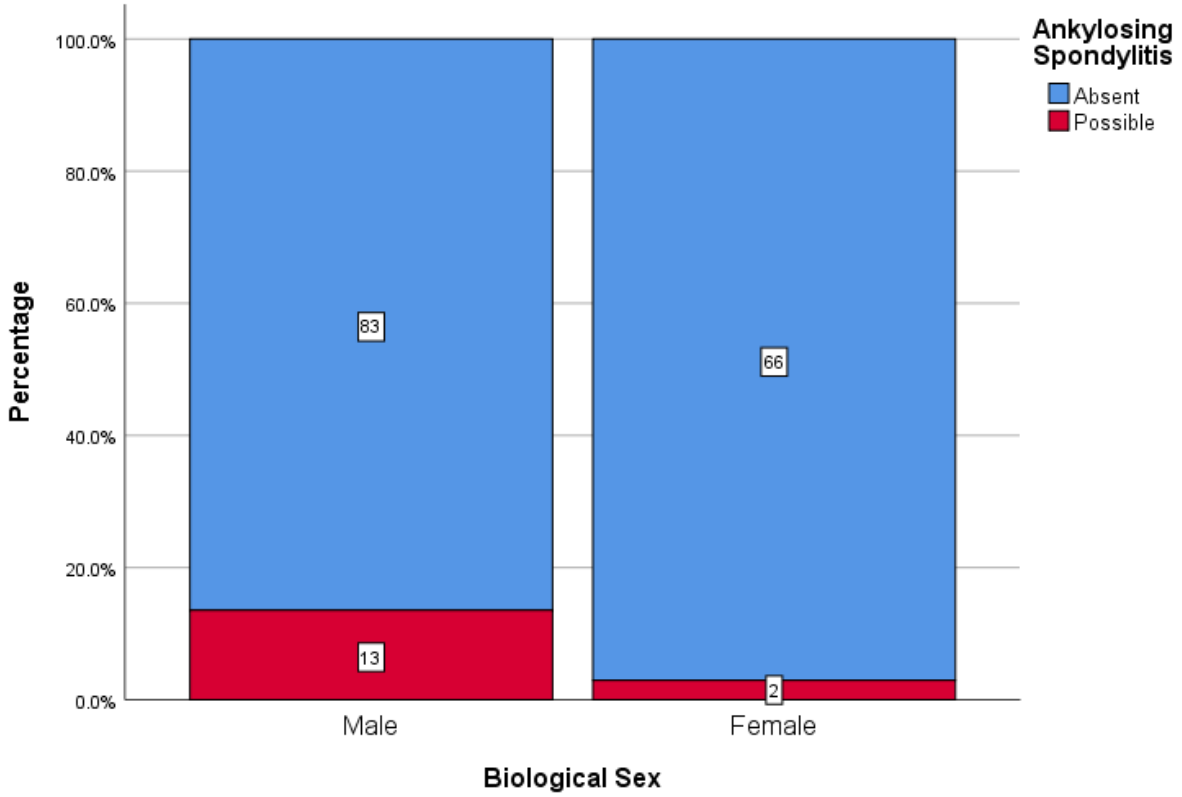

Figure 7.33 Distribution of the diagnoses for ankylosing spondylitis within this sample population by biological sex. Count is indicated within the bars.

The urban population for both females and males had a higher prevalence for possible cases of ankylosing spondylitis than the rural site category (see Table 7.38/ Figure 7.33). No females were found to have evidence of the condition within the rural site category and 6.67% (n = 2) of the female urban population was afforded a possible diagnosis. Of the male population with a possible diagnosis, 22.45% (n = 11) fell within the urban site category and 4.26% (n = 2) fell within the rural site category.

Table 7.38 Distribution of the diagnoses for ankylosing spondylitis within this sample population by site category and biological sex.

			URBAN		RURAL	
FEMALE			N	%	N	%
	DIAGNOSIS	ABSENT	28	93.3	38	100.0
		POSSIBLE	2	6.7	0	0.0
MALE			URBAN		RURAL	
			N	%	N	%
	DIAGNOSIS	ABSENT	38	77.6	45	95.7
		POSSIBLE	11	22.5	2	4.3

Note that unlike the previous joint conditions, probable has been replaced with possible diagnoses. Note that values may not ≠ 100 as decimal point placement is rounded.

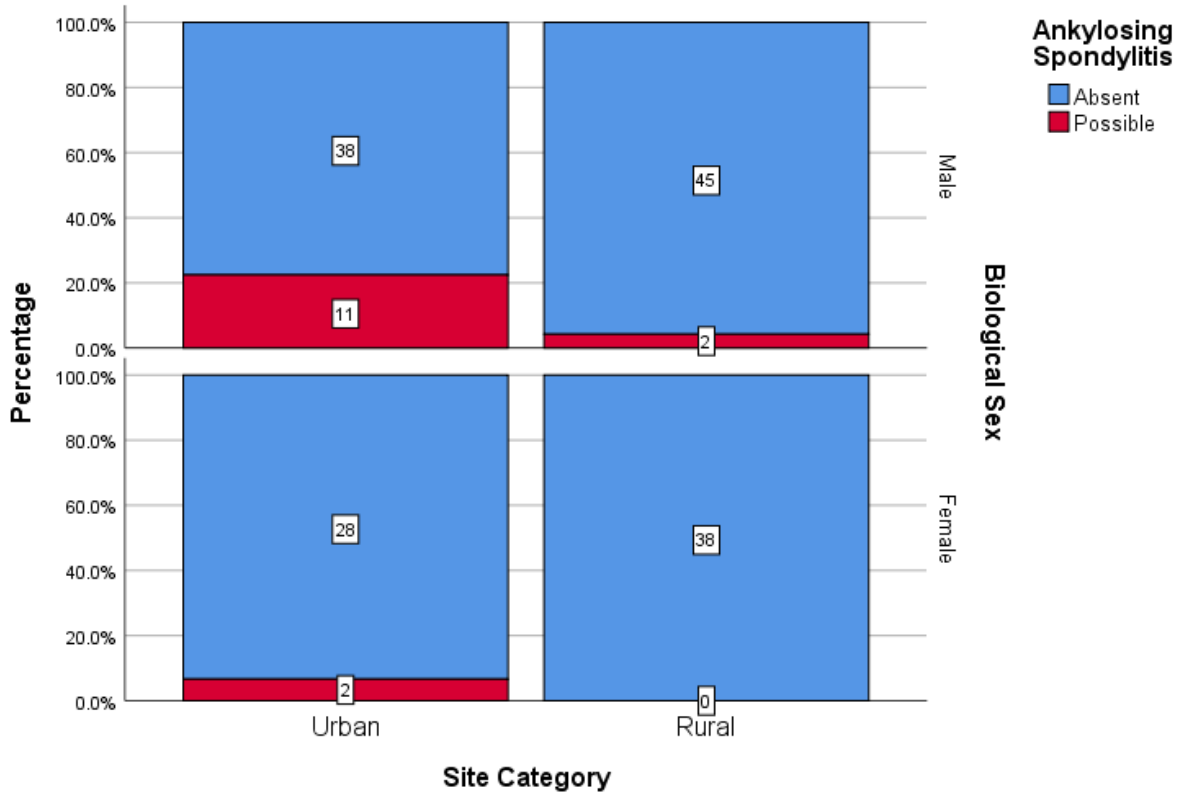

Figure 7.34 Distribution of the diagnoses for ankylosing spondylitis within this sample population by site category and biological sex. Note that unlike the previous joint conditions, probable has been replaced with possible diagnoses. Count is indicated within the bars.

Prevalence of possible ankylosing spondylitis within the age categories was skewed due to the low numbers and lack of probable cases (see Table 7.39/ Figure 7.35). Of the female population with a possible diagnosis of ankylosing spondylitis, 50.0% (n = 1) fell within the middle adult category and 50.0% (n = 1) fell within the late adult category. Of the male population with a possible diagnosis of ankylosing spondylitis, 23.1% (n = 3) fell within the

early adult category, 7.7% (n = 1) fell within the middle adult category, and 69.2% (n = 9) fell within the late adult category.

Table 7.39 Distribution of the diagnoses for ankylosing spondylitis within this sample population by biological sex and age at death.

			ABSENT		POSSIBLE	
			N	%	N	%
FEMALE	AGE AT DEATH CATEGORIES	EARLY	27	100.0	0	0.0
		MIDDLE	15	93.8	1	6.3
		LATE	24	96.0	1	4.0
			ABSENT		POSSIBLE	
			N	%	N	%
MALE	AGE AT DEATH CATEGORIES	EARLY	24	88.9	3	11.1
		MIDDLE	19	95.0	1	5.0
		LATE	40	81.6	9	18.4

Note that values may not ≠ 100 as decimal point placement is rounded.

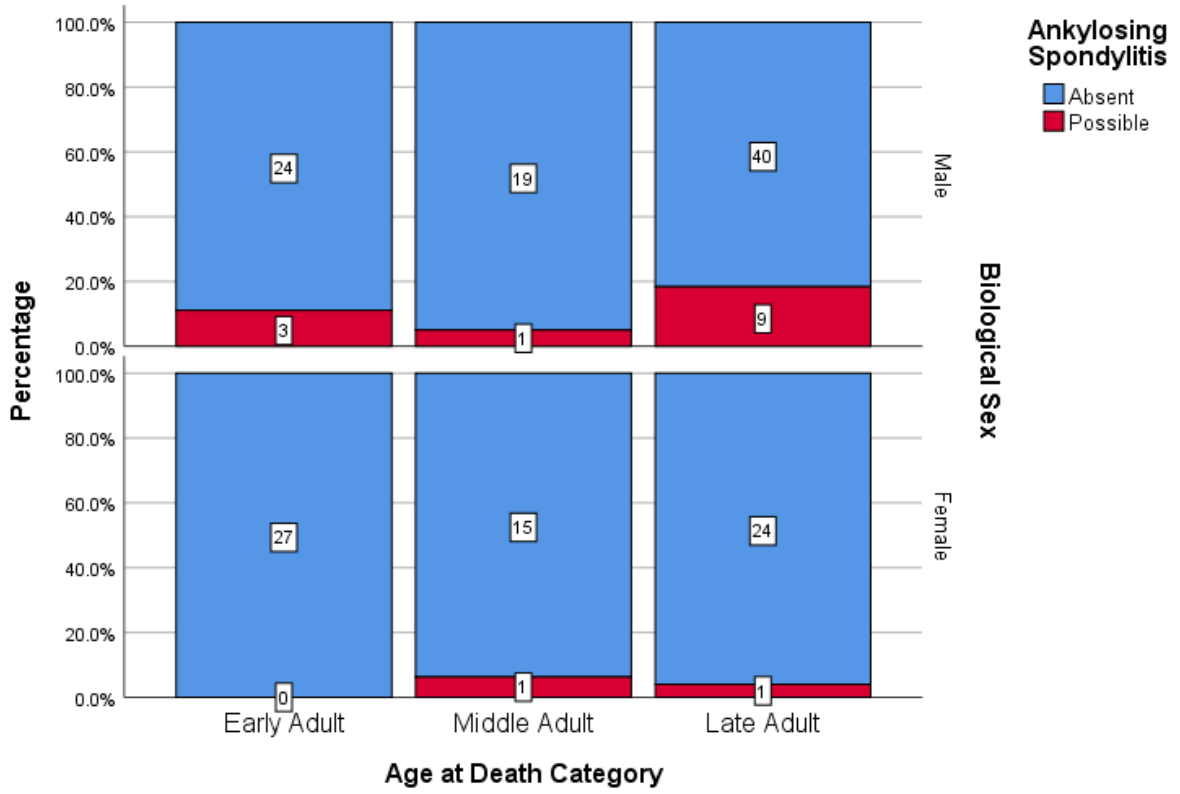

Figure 7.35 Distribution of the diagnoses for ankylosing spondylitis within this sample population by biological sex and age at death. Note that unlike the previous joint conditions, probable has been replaced with possible diagnoses. Count is indicated within the bars.

Urban males displayed a possible prevalence of 18.8% (n = 3) for early adults, 11.1% (n = 1) for middle adults and 12.5% (n = 7) for late adults (see Table 7.40/ Figure 7.36). Rural males only displayed a possible prevalence for late adults (8.0%; n = 2). Urban females had no possible diagnoses within early adults and the remaining possible prevalence was 12.5% (n = 1) for both middle and late adults. No possible diagnoses were found within the rural females.

Table 7.40 Distribution of the diagnoses for ankylosing spondylitis within this sample population by site category, biological sex, and age at death.

				ABSENT		POSSIBLE	
				N	%	N	%
URBAN	FEMALE	AGE AT DEATH CATEGORIES	EARLY	14	100.0	0	0.0
			MIDDLE	7	87.5	1	12.5
			LATE	7	87.5	1	12.5
				ABSENT		POSSIBLE	
				N	%	N	%
	MALE	AGE AT DEATH CATEGORIES	EARLY	13	81.3	3	18.8
			MIDDLE	8	88.9	1	11.1
			LATE	17	70.8	7	29.2
RURAL				ABSENT		POSSIBLE	
				N	%	N	%
	FEMALE	AGE AT DEATH CATEGORIES	EARLY	13	100.0	0	0.0
			MIDDLE	8	100.0	0	0.0
			LATE	17	100.0	0	0.0
				ABSENT		POSSIBLE	
				N	%	N	%
	MALE	AGE AT DEATH CATEGORIES	EARLY	11	100.0	0	0.0
			MIDDLE	11	100.0	0	0.0
			LATE	23	92.0	2	8.0

Note that unlike the previous joint conditions, probable has been replaced with possible diagnoses. Percentages are within each subcategory of age.

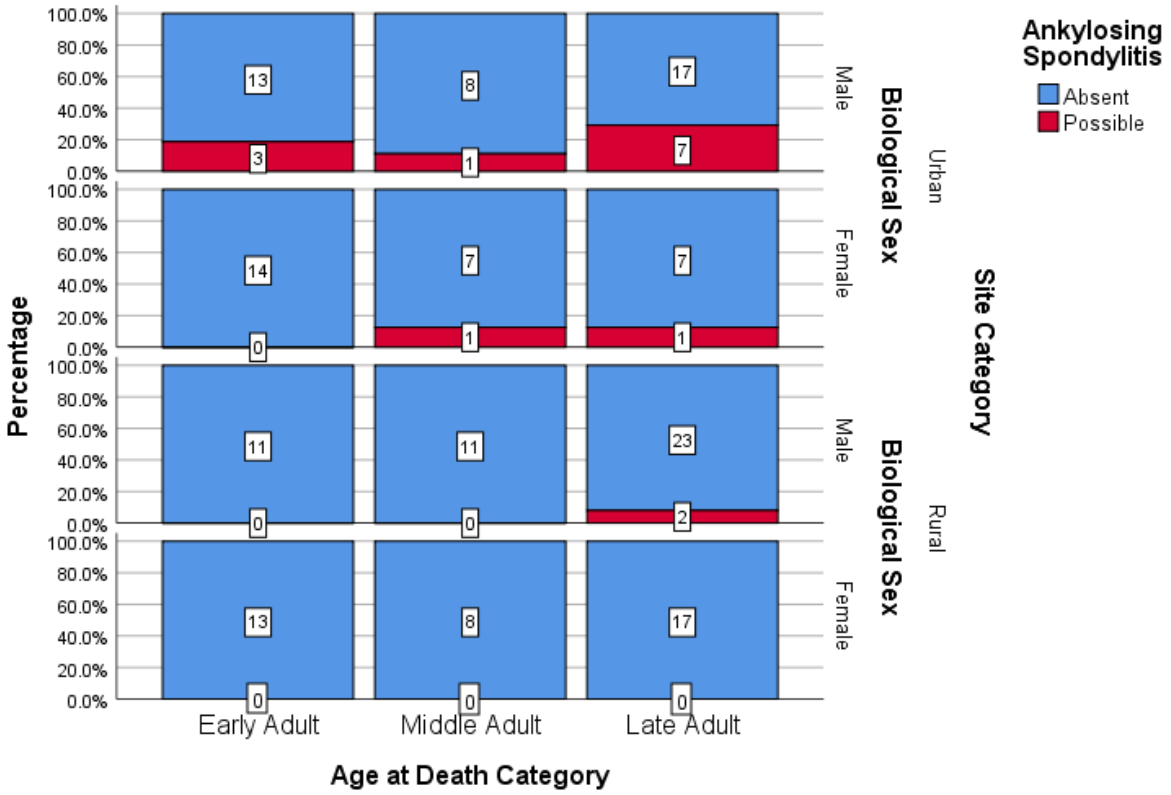

Figure 7.36 Distribution of the diagnoses for ankylosing spondylitis within this sample population by site category, biological sex, and age at death. Note that unlike the previous joint conditions, probable has been replaced with possible diagnoses. Count is indicated within the bars.

Ankylosing spondylitis was unable to be completely or accurately assessed, as there were no probable diagnoses. The possible diagnoses are not conclusive as they do not represent definitive cases of the condition. Males had more possible diagnoses than the females and the urban sites also contained the higher proportion of numbers.

7.3.6 Sacroiliitis (SI)

Overall, 6.8% (n = 11) of the sample population were given a probable diagnosis of sacroiliitis (see Table 7.41/ Figure 7.37). The urban and rural population had a uniform distribution of probable diagnoses (6.4%, n = 5 and 7.1%, n = 6). A total of 9.4% (n = 9) of the male individuals were afforded a probable diagnosis and of the female population, only 3.0% (n = 2) were afforded a probable diagnosis (see Table 7.42/ Figure 7.38).

Table 7.41 Distribution of the diagnoses for sacroiliitis within this sample population by site category.

BIOLOGICAL SEX	DIAGNOSIS	N	%
URBAN	ABSENT	73	93.6
	PROBABLE	5	6.4
RURAL	ABSENT	79	92.9
	PROBABLE	6	7.1
TOTAL	ABSENT	152	93.3
	PROBABLE	11	6.8

Note that values may add up to >100 as decimal point placement is rounded. Note that values may not ≠ 100 as decimal point placement is rounded.

Figure 7.37 Distribution of the diagnoses for sacroiliitis within this sample population by site category, biological sex, and age at death. Count is indicated within the bars.

Table 7.42 Distribution of the diagnoses for sacroiliitis within this sample population by biological sex.

BIOLOGICAL SEX	DIAGNOSIS	N	%
MALE	ABSENT	87	90.6
	PROBABLE	9	9.4
FEMALE	ABSENT	65	97.01
	PROBABLE	2	3.0

Note that values may add up to >100 as decimal point placement is rounded. Note that values may not ≠ 100 as decimal point placement is rounded.

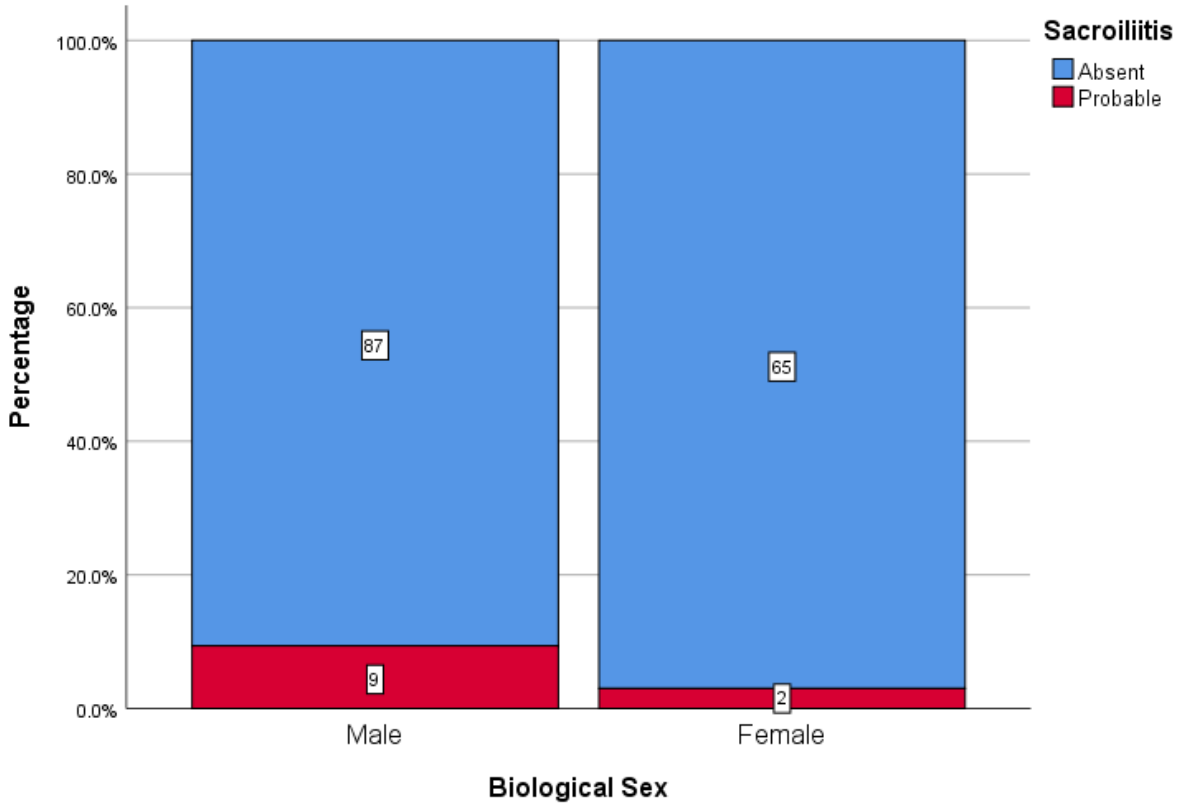

Figure 7.38 Distribution of the diagnoses for sacroiliitis within this sample population by biological sex. Count is indicated within the bars.

No females from the urban site category were diagnosed with sacroiliitis, whereas 5.3% (n = 2) of the female rural population were given a probable diagnosis (see Table 7.42/ Figure 7.39). Of the male population with probable diagnoses, 10.2% (n=5) fell within the urban population and 8.5% (n = 4) fell within the rural population.

Table 7.43 Distribution of the diagnoses for sacroiliitis within this sample population by site category and biological sex.

			URBAN		RURAL	
FEMALE			N	%	N	%
	DIAGNOSIS	ABSENT	29	100.0	36	94.7
		PRESENT	0	0.0	2	5.3
			URBAN		RURAL	
MALE			N	%	N	%
	DIAGNOSIS	ABSENT	44	89.8	43	91.5
		PRESENT	5	10.2	4	8.5

Note that values may not ≠ 100 as decimal point placement is rounded.

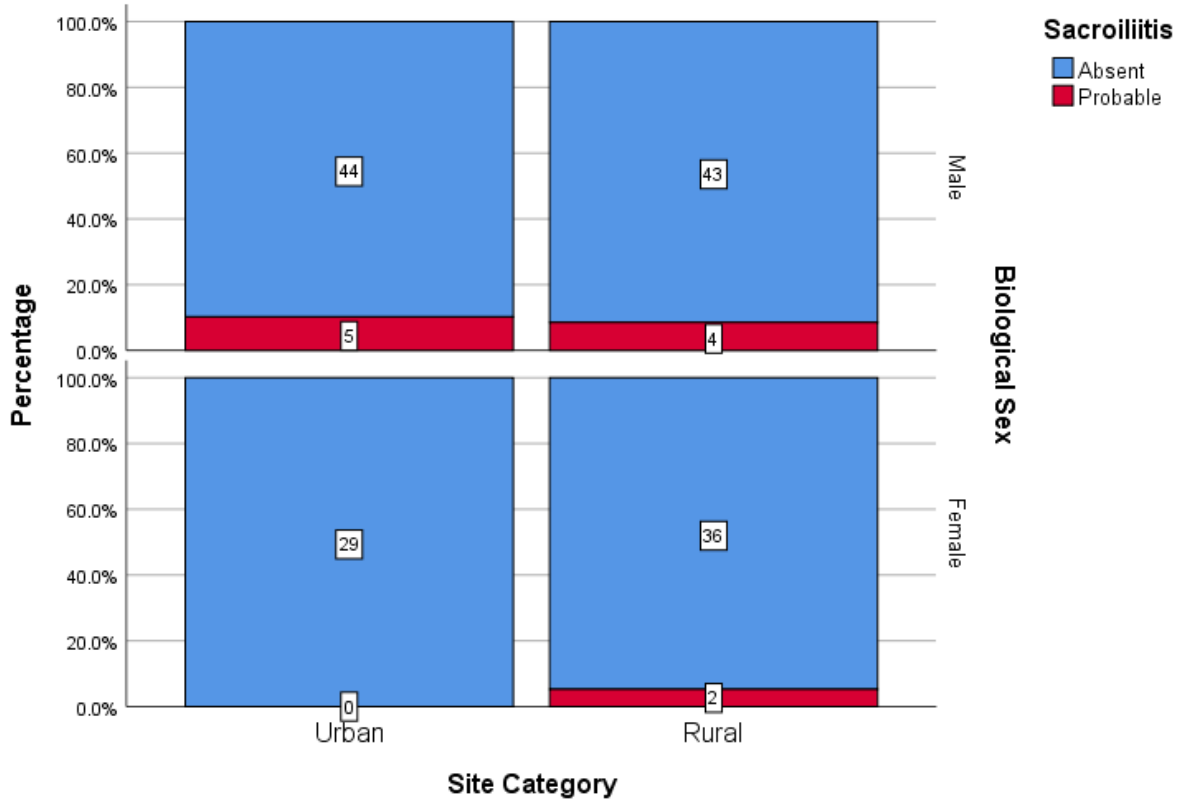

Figure 7.39 Distribution of the diagnoses for sacroiliitis within this sample population by site category and biological sex. Count is indicated within the bars.

No cases of sacroiliitis fell within the early adult age category for females (see Table 7.44/ Figure 7.40). Of the female population with a probable diagnosis of sacroiliitis, 6.7% (n = 1) fell within the middle adult category and 4.0% (n = 1) were within the late adult category. Of the male population with a probable diagnosis of sacroiliitis, 3.7% (n=1) fell within the early adult category, 5.6% (n = 1) fell within the middle adult category and 13.7% (n = 7) fell within the late adult category.

Table 7.44 Distribution of the diagnoses for sacroiliitis within this sample population by biological sex and age at death.

			ABSENT		PROBABLE	
			N	%	N	%
FEMALE	AGE AT DEATH CATEGORIES	EARLY	27	100.0	0	0.0
		MIDDLE	14	93.3	1	6.7
		LATE	24	96.0	1	4.0
			ABSENT		PROBABLE	
			N	%	N	%
MALE	AGE AT DEATH CATEGORIES	EARLY	26	96.3	1	3.7
		MIDDLE	17	94.4	1	5.6
		LATE	44	86.3	7	13.7

Note that values may not ≠ 100 as decimal point placement is rounded.

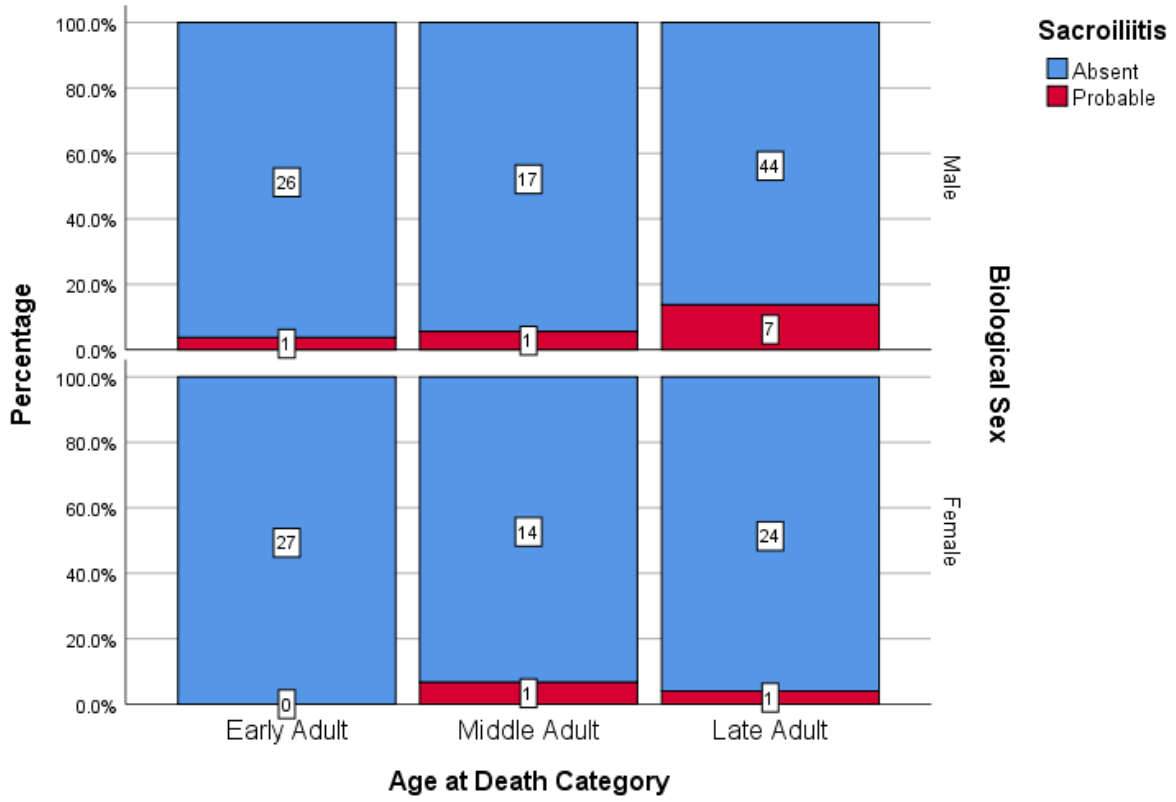

Figure 7.40 Distribution of the diagnoses for sacroiliitis within this sample population by biological sex and age at death. Count is indicated in the bars.

Urban males had no probable diagnoses for early adults and the remaining prevalence rates between the age categories displayed 25.0% (n = 1) for middle adults and 75.0% (n = 3) for late adults (see Table 7.45/ Figure 7.41). Rural males had no probable diagnoses for early adults also, and the remaining prevalence rates between the age categories were split at 50.0% (n = 1) for middle and late adults. Urban females had no probable diagnoses within middle

adults and the remaining prevalence rates for probable diagnoses displayed 20.0% (n = 1) for early adults and 80.0% (n = 4) for late adults.

Table 7.45 Distribution of the diagnoses for sacroiliitis within this sample population by site category, biological sex, and age at death.

				ABSENT		PROBABLE	
				N	%	N	%
URBAN	FEMALE	AGE AT DEATH CATEGORIES	EARLY	14	100.0	0	0.0
			MIDDLE	7	100.0	0	0.0
			LATE	8	100.0	0	0.0
				ABSENT		PROBABLE	
				N	%	N	%
	MALE	AGE AT DEATH CATEGORIES	EARLY	15	93.8	1	6.3
			MIDDLE	7	100.0	0	0.0
			LATE	22	84.6	4	15.4
RURAL				ABSENT		PROBABLE	
				N	%	N	%
	FEMALE	AGE AT DEATH CATEGORIES	EARLY	13	100.0	0	0.0
			MIDDLE	7	87.5	1	12.5
			LATE	16	94.1	1	5.9
				ABSENT		PROBABLE	
				N	%	N	%
	MALE	AGE AT DEATH CATEGORIES	EARLY	11	100.0	0	0.0
			MIDDLE	10	90.9	1	9.1
			LATE	22	88.0	3	12.0

Note that values may not ≠ 100 as decimal point placement is rounded. Percentages are within each subcategory of age.

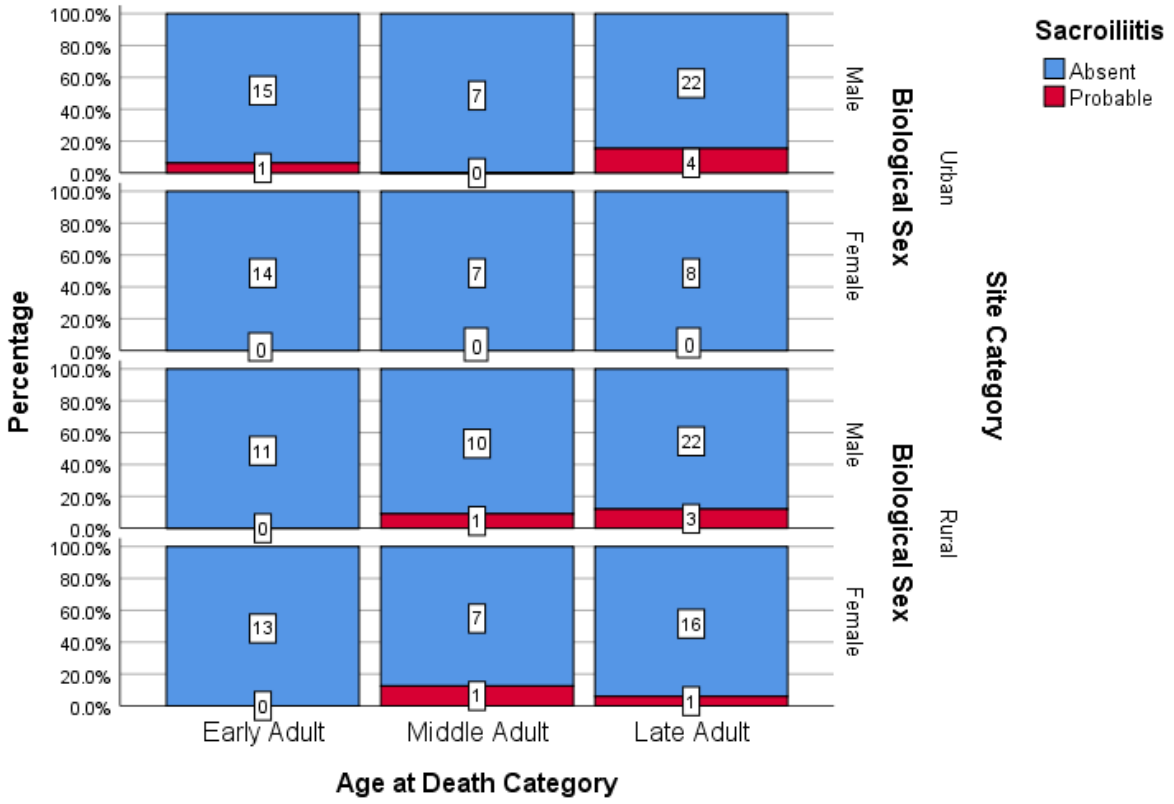

Figure 7.41 Distribution of the diagnoses for sacroiliitis within this sample population by site category, biological sex, and age at death. Count is indicated within the bars.

Sacroiliitis was rare in the sample, resulting in an exceptionally low proportion of probable diagnoses. Males had a larger proportion of probable diagnoses, and between site categories the distribution is similar. Late adults had the higher proportion of cases. The low number of probable cases makes any further assessment unreliable.

7.3.7 Degenerative Disc Disease (DDD)

Probable cases of degenerative disc disease were observed in 17.3% (n = 29) of the sample population (see Table 7.46/ Figure 7.42). The urban site category (22.9%, n = 19) had almost twice the overall prevalence of DDD than the rural site category (11.8%, n = 10). Males (19.4%, n = 19) had a slightly higher proportion of degenerative disc disease than females (14.3%, n = 10) within the sample population (see Table 7.47/ Figure 7.43).

Table 7.46 Distribution of the diagnoses for degenerative disc disease within this sample population by site category.

BIOLOGICAL SEX	DIAGNOSIS	N	%
URBAN	ABSENT	64	77.1
	PROBABLE	19	22.9
RURAL	ABSENT	75	88.2
	PROBABLE	10	11.8
TOTAL	ABSENT	139	82.7
	PROBABLE	29	17.3

Note that values may not ≠ 100 as decimal point placement is rounded.

Figure 7.42 Distribution of the diagnoses for degenerative disc disease within this sample population by site category. Count is indicated within the bars.

Table 7.47 Distribution of the diagnoses for degenerative disc disease within this sample population by biological sex.

BIOLOGICAL SEX	DIAGNOSIS	N	%
MALE	ABSENT	79	80.6
	PROBABLE	19	19.4
FEMALE	ABSENT	60	85.
	PROBABLE	10	14.3

Note that values may not ≠ 100 as decimal point placement is rounded.

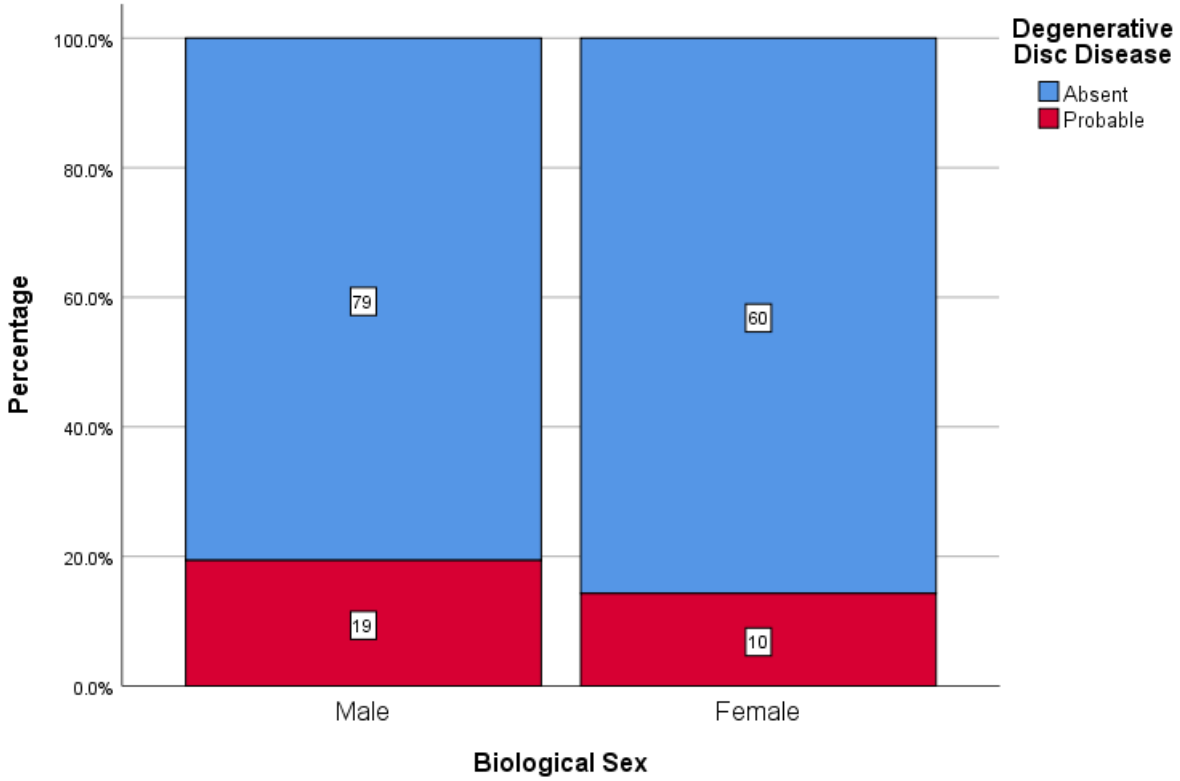

Figure 7.43 Distribution of the diagnoses for degenerative disc disease studied within this sample population by biological sex. The possible diagnoses category has been reduced into the absent category. Count is indicated within the bars.

The female population had a similar distribution of prevalence for DDD, while the males had an almost 3:1 difference between the site categories (see Table 7.48/ Figure 7.44). Probable cases of DDD were found to be 15.6% (n = 5) of the urban females and 13.2% (n = 5) of the rural females. Probable cases of DDD were found to be 27.5% (n = 14) in urban males and 10.6% (n = 5) in the rural males.

Table 7.48 Distribution of the diagnoses for degenerative disc disease within this sample population by site category and biological sex.

			URBAN		RURAL	
			N	%	N	%
FEMALE	DIAGNOSIS	ABSENT	27	84.4	33	86.8
		PRESENT	5	15.6	5	13.2
			URBAN		RURAL	
			N	%	N	%
MALE	DIAGNOSIS	ABSENT	37	72.6	42	89.4
		PRESENT	14	27.5	5	10.6

Note that values may not ≠ 100 as decimal point placement is rounded.

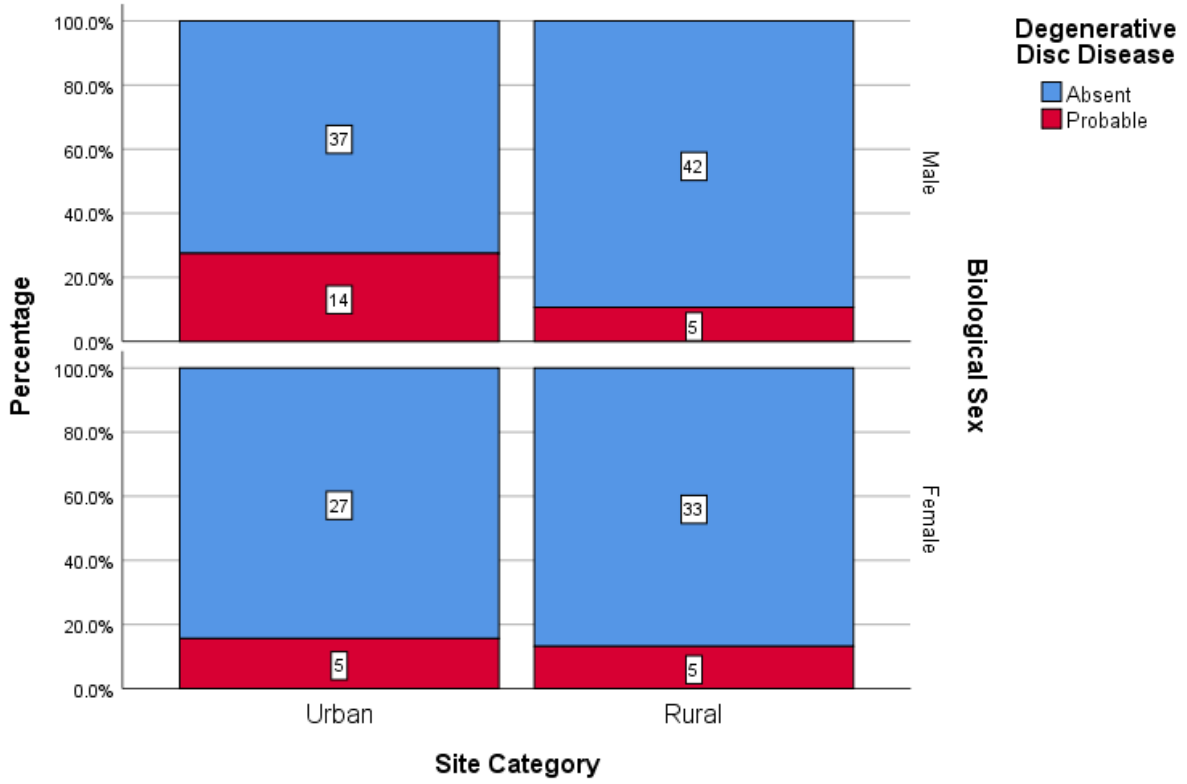

Figure 7.44 Distribution of the diagnoses for degenerative disc disease within this sample population by site category and biological sex.

Females had a higher prevalence of degenerative disc disease at the early adult category and males had a higher prevalence at the middle and late adult categories (see Table 7.47/ Figure 7.44). Of the female population with a probable diagnosis of degenerative disc disease, 40.0% (n = 4) fell within the early adult category, 10.0% (n = 1) fell within the middle adult category and 50.0% (n = 5) fell within the late adult category. Of the male population with a probable diagnosis of degenerative disc disease, 15.8% (n = 3) fell within the early adult

category, 21.0% (n = 4) fell within the middle adult category, and the remaining 63.2% (n =12) fell within the late adult category.

Table 7.49 Distribution of the diagnoses for degenerative disc disease within this sample population by biological sex and age at death.

			ABSENT		PROBABLE	
			N	%	N	%
FEMALE	AGE AT DEATH CATEGORIES	EARLY	24	85.7	4	14.3
		MIDDLE	16	94.1	1	5.9
		LATE	20	80.0	5	20.0
			ABSENT		PROBABLE	
			N	%	N	%
MALE	AGE AT DEATH CATEGORIES	EARLY	26	89.7	3	10.3
		MIDDLE	16	80.0	4	20.0
		LATE	38	76.0	12	24.0

Note that values may not ≠ 100 as decimal point placement is rounded.

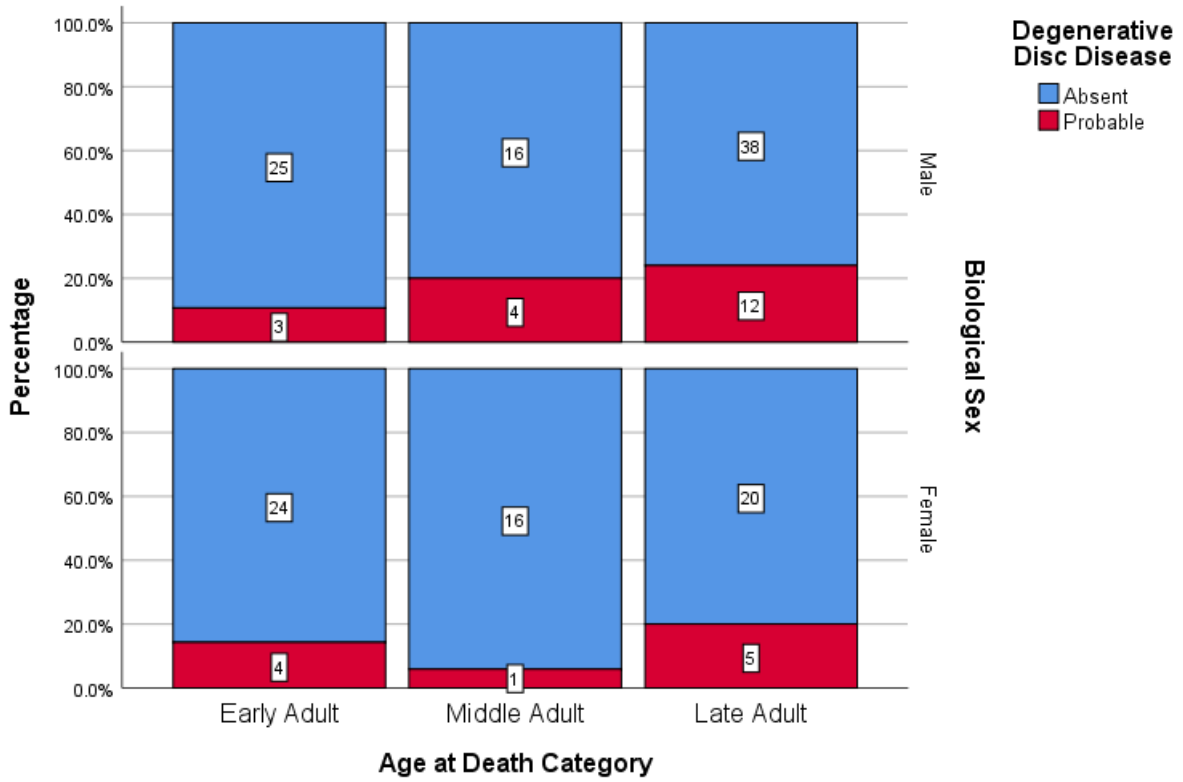

Figure 7.45 Distribution of the diagnoses for degenerative disc disease within this sample population by biological sex and age at death. Count is indicated within the bars.

Urban males with a probable diagnosis displayed a prevalence of 14.3% (n = 2) for early adults, 21.4% (n = 3) for middle adults and 64.3% (n = 9) for late adults (see Table 7.49/ Figure 7.45). Rural males with a probable diagnosis displayed prevalence of 20.0% (n = 1) for early and middle adults and 60.0% (n = 3) for late adults. Urban females had no probable diagnoses within early adults and the remaining probable diagnoses for the age at death categories displayed 20.0% (n = 1) for middle adults and 80.0% (n = 4) for late adults. Rurals had no probable diagnoses for early adults and the remaining probable diagnoses for age at death categories showed the inverse of the urban prevalence with 80.0% (n= 4) for early adults and 20.0% (n = 1) for late adults.

Table 7.50 Distribution of the diagnoses for degenerative disc disease within this sample population by site category, biological sex, and age at death.

				ABSENT		PROBABLE	
				N	%	N	%
URBAN	FEMALE	AGE AT DEATH CATEGORIES	EARLY	15	100.0	0	0.0
			MIDDLE	8	88.9	1	11.1
			LATE	4	50.0	4	50.0
	MALE			ABSENT		PROBABLE	
				N	%	N	%
		AGE AT DEATH CATEGORIES	EARLY	15	88.2	2	11.8
			MIDDLE	6	66.7	3	33.3
			LATE	16	64.0	9	36.0
RURAL	FEMALE			ABSENT		PROBABLE	
				N	%	N	%
		AGE AT DEATH CATEGORIES	EARLY	9	69.2	4	30.8
			MIDDLE	8	100.0	0	0.0
			LATE	16	94.1	1	5.9
	MALE			ABSENT		PROBABLE	
				N	%	N	%
		AGE AT DEATH CATEGORIES	EARLY	10	90.9	1	9.1
			MIDDLE	10	90.9	1	9.1
			LATE	22	88.0	3	12.0

Note that values may not ≠ 100 as decimal point placement is rounded. Percentages are within each subcategory of age.

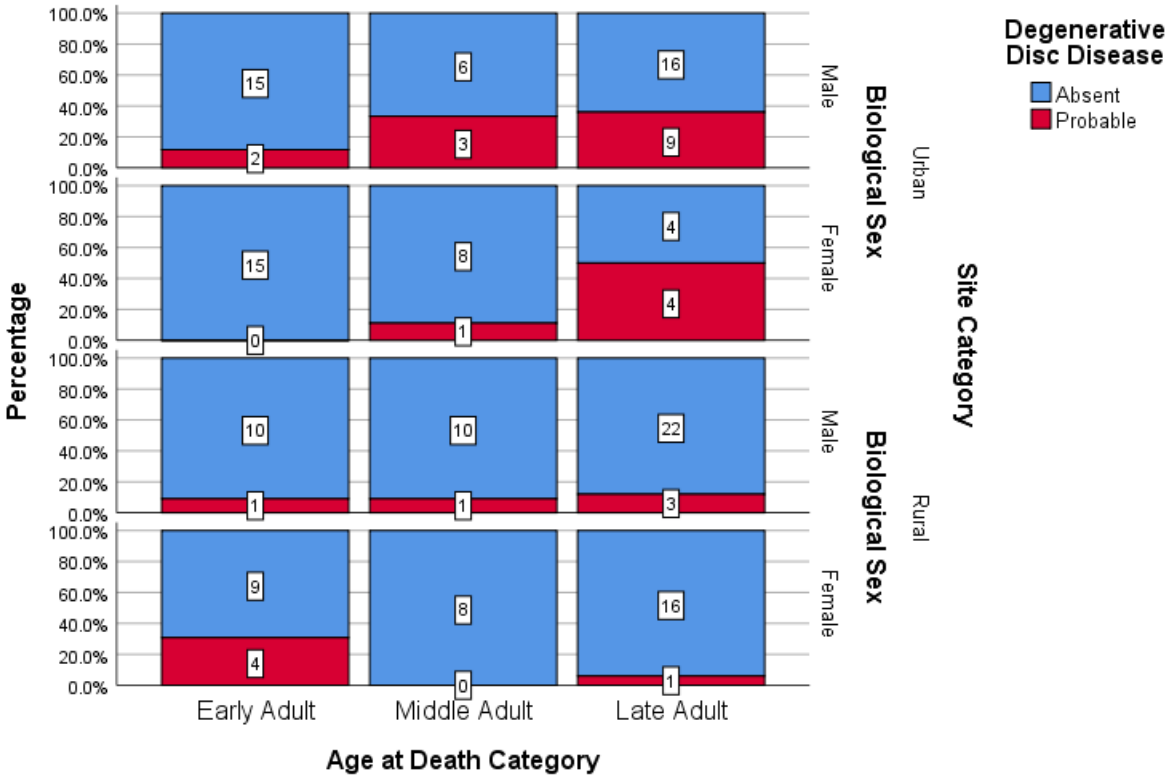

Figure 7.46 Distribution of the diagnoses for degenerative disc disease within this sample population by site category, biological sex, and age at death. Count is indicated within the bars.

In summation, degenerative disc disease was present in almost two fifths of the population and the proportions were higher in the urban population than the rural. Males had a high proportion, although the difference was not large. Urban males had 2.6 times the number of probable diagnoses than the rural males, while females were evenly divided. Degenerative disc disease had larger proportions within the late adult category, with exception of the rural females, which had the larger proportion in the middle adult category.

7.3.8 Summary

Four of the sample collections had enough of a sample size to be viable for further comparisons with data from the literature in the following discussion chapter (see Table 7.51). These four sites represent individuals from both rural (Barton-upon-Humber) and urban sites (South Shields, Wolverhampton, and Kingston-upon-Thames). The ratios for each joint condition (urban: rural) will be used for the discussions as well. One or more joint conditions was present in roughly half of the sample population and osteoarthritis the most common. Spinal osteoarthritis was the most common joint condition present within this sample. The

prevalence of joint conditions was distributed roughly equally between the site categories displayed, apart from spinal osteoarthritis and degenerative disc diseases (see Table 7.52). Spinal osteoarthritis was more prevalent in the rural population, while degenerative disc disease was more prevalent in the urban population. Late adults had the larger proportions of all the joint conditions; however, urban females exhibited a trend towards a more even distribution of probable diagnoses across the age at death categories which, in some cases, resulted in more young and middle adults presenting joint condition than late adults. For OA, SOA and HOA, rural males displayed a trend of having larger prevalences at younger ages. AS and SI were present in much smaller proportions that the other joint conditions, which results in too small a sample to draw any conclusions about their distribution with site category, sex, or age.

Table 7.51 The prevalences of the joint conditions by individual site.

	OA		SOA		HOA		KOA		DDD	
	N	%	N	%	N	%	N	%	N	%
SOUTH SHIELDS	16	34.8	9	20.0	7	15.2	7	15.2	10	22.7
WOLVERHAMPTON	2	10.0	2	11.8	1	5.0	0	0.0	4	25.0
KINGSTON-UPON-THAMES	7	38.9	4	26.7	2	11.1	1	6.3	2	11.8
BARTON-UPON-HUMBER	33	38.8	25	29.4	11	12.9	8	9.4	10	11.8

These are the sites with the most viable individual sample sizes. Note how Wolverhampton had the lowest prevalence rates across the board except for degenerative disc disease which was the highest.

Table 7.52 The prevalences of the joint conditions by site category from within this study.

	OA	SOA	HOA	KOA	DDD
URBAN	30.0	19.3	12.2	10.3	22.9
RURAL	38.8	29.4	12.9	9.4	11.8
RATIO	0.8	0.7	0.9	1.1	1.9

Note that DDD is higher for urban, HOA and KOA are more neutral, and OA and SOA are higher for rural. Ratio is urban/ rural.

7.4 Aetiological Factors

Site category, biological sex and age at death are only three of a wider range of variables that were assessed in this project to explore the risk factors underpinning joint condition in the past. The remaining variables include factors related to body mass and activity patterns that are thought to directly affect the onset and progression of the joint conditions discussed within this body of work. These variables were body mass index, entheseal changes and a grouping of eight separate but related cross-sectional indices.

7.4.1 Body Mass Index (BMI)

The data for BMI were reported in three formats: point values on a continuous scale, categorization based on interquartile range and binary categorization ('normal' and 'over' mass). The reason for the different scales for reporting the BMI data relates directly to the different type of analysis. The binary categorization helps to display the data in basic percentages and proportions in a way similar to the clinical categorizations. The binary categorization originally had 4 categories, however, there were no under mass individuals and only a very small number of extremely over mass which were then merged into the over mass category. The point-based data allowed for a better analysis of ranges and distribution within the population and with variables added. The interquartile range categorization was better suited to the statistical tests to determine association and correlation, as it provided a more nuanced distinction between mass levels than the binary categorization that would help to investigate the relationship between body mass and joint condition prevalence.

Overall, 65.3% (n = 98) of the sample population were found to be normal mass and 34.7% were found to be over massed (see Table 7.53). No individuals had a BMI that fell within the under-mass range. The urban site category displayed 63.5% (n = 47) with a normal mass BMI and 36.5% (n = 27) with an over mass BMI. The rural site category displayed 67.1% (n = 51) with a normal mass BMI and 32.9% (n = 25) with an over mass BMI. Of the males (see Table 7.54), 62.5% (n = 55) were normal mass and 37.5% (n = 33) were over mass. Of the females, 69.4% (n = 43) were normal mass and 30.7% (n = 19) were over mass. Thus, in general there was no difference in BMI between the site types, but a slightly higher proportion of males were over mass compared to females.

Table 7.53 Distribution of body mass by site category.

SITE CATEGORY	BMI CATEGORY	N	%
URBAN	NORMAL	47	63.5
	OVER	27	36.5
RURAL	NORMAL	51	67.1
	OVER	25	32.9
TOTAL	NORMAL	98	65.3
	OVER	52	34.7

Note that values may not ≠ 100 as decimal point placement is rounded.

Table 7.54 Distribution of body mass by biological sex.

BIOLOGICAL SEX	BMI CATEGORY	N	%
MALE	NORMAL	55	62.5
	OVER	33	37.5
FEMALE	NORMAL	43	69.4
	OVER	19	30.7

Note that values may not ≠ 100 as decimal point placement is rounded.

Body mass was further subdivided between age at death and biological sex (see Table 7.55/ Figure 7.47). The larger proportion of males were normal mass with 31.8% (n = 28) being late adults, 14.8% (n = 13) middle adults and 15.9% (n = 14) early adults. The over mass males represented 20.5% (n = 19) late adults, 9.1% (n = 8) middle adults and 8.0% (n = 7) early adults. The larger proportion of females were also normal mass with 25.8% (n = 16) late adults, 17.7% (n = 11) middle adults and 25.8% (n = 16) early adults. The over mass females represented 11.3% (n =7) late adults, 4.8% (n = 3) middle adults and 14.5% (n = 9) young adults.

Table 7.55 Distribution of body mass by biological sex and age at death.

			NORMAL MASS		OVER MASS	
FEMALE			N	%	N	%
	AGE AT DEATH CATEGORIES	EARLY	16	25.8	9	14.5
		MIDDLE	11	17.7	3	4.8
		LATE	16	25.8	7	11.3
			NORMAL MASS		OVER MASS	
MALE			N	%	N	%
	AGE AT DEATH CATEGORIES	EARLY	14	15.9	7	8.0
		MIDDLE	13	14.8	8	9.1
		LATE	28	31.8	18	20.5

Percentages are within the biological sex categories.

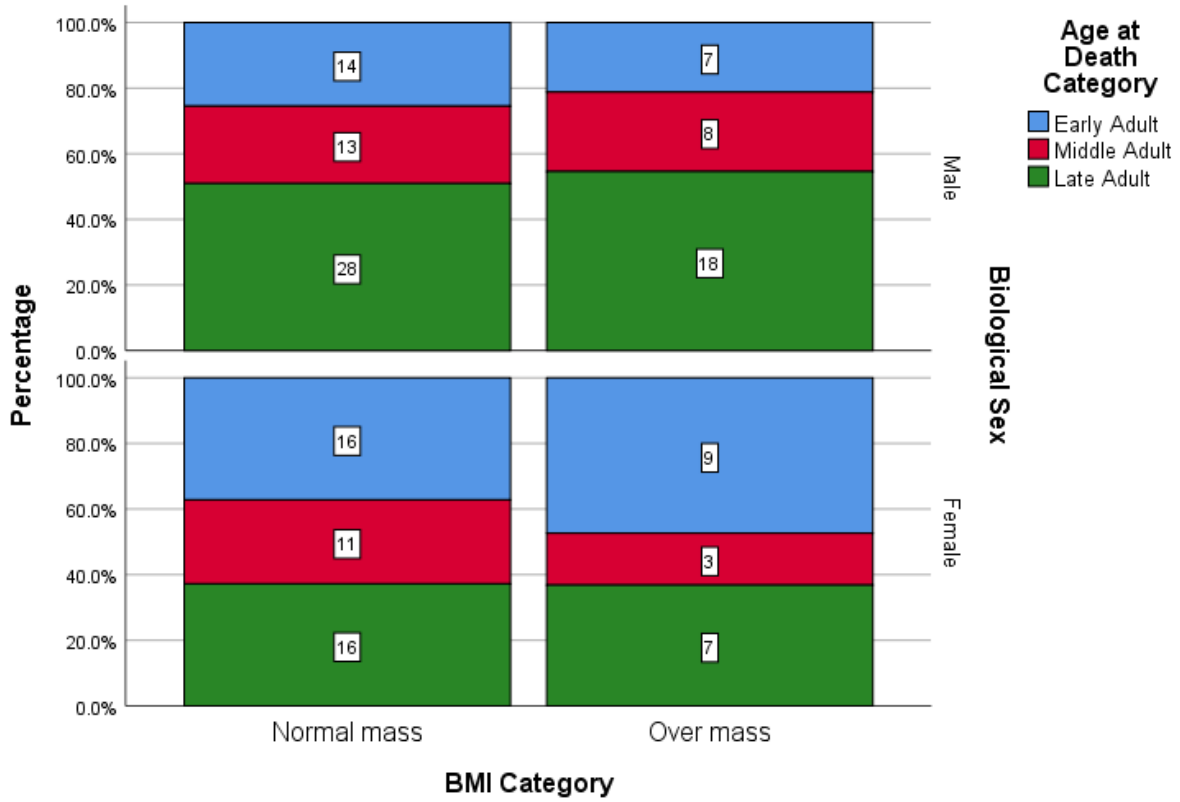

Figure 7.47 Proportional distribution of body mass by biological sex and age at death. Count is indicated within the bars.

Mean body mass is similar for males (24.25) and females (23.92) and between male and females age at death categories, however, the range of body masses among the males is greater at all ages than the females. This distinction between the sexes in most apparent among young individuals. While females have a comparatively restricted range of BMI in the young adult group, which increases with age, the males present greater variation at all ages. Indeed, both the maximum and minimum BMI in the population belong to young males (see Figure 7.48).

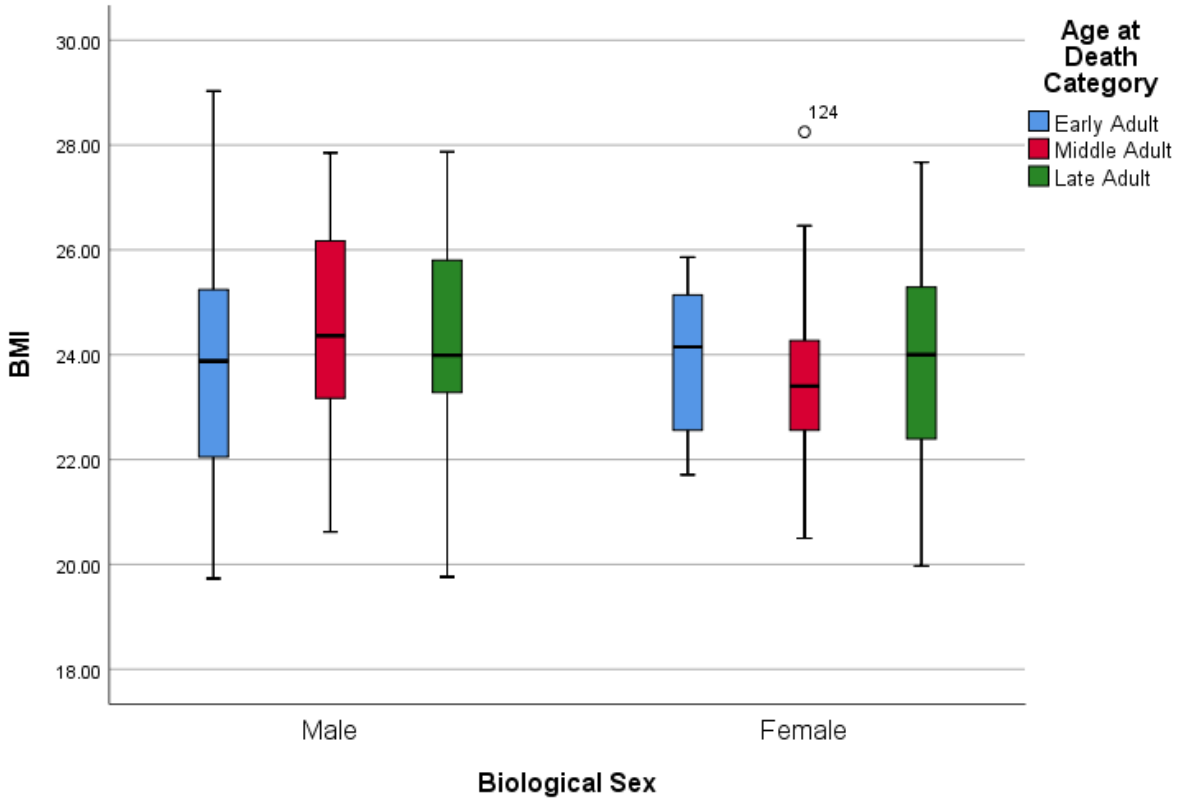

Figure 7.48 Boxplot of the sample population's body mass index, sorted by biological sex and age at death categories, which presents the mean and interquartile ranges.

Within the urban male population, normal massed individuals displayed percentages of 31.8% (n = 14) for late adults, 9.1% (n = 4) for middle adults, and 20.5% (n = 9) for early adults (see Table 7.56/ Figure 7.49). The over massed urban males displayed percentages of 18.2% (n = 8) for late adults, 11.4% (n = 5) for middle adults, and 9.1% (n = 4) for early adults. Within the urban female population, normal massed individuals displayed percentages of 20.7% (n = 6) for late adults, 20.7% (n = 6) for middle adults, and 24.1% (n = 7) for early adults. The over massed urban females displayed percentages of 10.3% (n = 3) for late adults, 6.9% (n = 2) for middle adults, and 17.2% (n = 5) for early adults.

Within the rural male population, normal massed individuals displayed percentages of 32.6% (n = 14) for late adults, 18.6% (n = 8) for middle adults, and 11.6% (n = 5) for early adults. The over massed rural males displayed percentages of 23.3% (n = 10) for late adults, 7.0% (n = 3) for middle adults, and 7.0% (n = 3) for early adults. Within the rural female population, normal massed individuals displayed percentages of 30.3% (n = 10) for late adults, 15.2% (n = 5) for middle adults, and 27.3% (n = 9) for early adults. The over massed rural

females displayed percentages of 12.1% (n = 4) for late adults,3.0% (n = 1) for middle adults, and 12.1% (n = 4) for early adults.

Table 7.56 Distribution of body mass by site category, biological sex, and age at death.

				NORMAL MASS		OVER MASS	
URBAN	MALE			N	%	N	%
		AGE AT DEATH CATEGORIES	EARLY	9	20.5%	4	9.1%
			MIDDLE	4	9.1%	5	11.4%
			LATE	14	31.8%	8	18.2%
	FEMALE			NORMAL MASS		OVER MASS	
				N	%	N	%
		AGE AT DEATH CATEGORIES	EARLY	7	24.1%	5	17.2%
			MIDDLE	6	20.7%	2	6.9%
			LATE	6	20.7%	3	10.3%
RURAL	MALE			NORMAL MASS		OVER MASS	
				N	%	N	%
		AGE AT DEATH CATEGORIES	EARLY	5	11.6%	3	7.0%
			MIDDLE	8	18.6%	3	7.0%
			LATE	14	32.6%	10	23.3%
	FEMALE			NORMAL MASS		OVER MASS	
				N	%	N	%
		AGE AT DEATH CATEGORIES	EARLY	9	27.3%	4	12.1%
			MIDDLE	5	15.2%	1	3.0%
			LATE	10	30.3%	4	12.1%

Percentages are by subcategory (urban males, urban females, rural males, rural females).

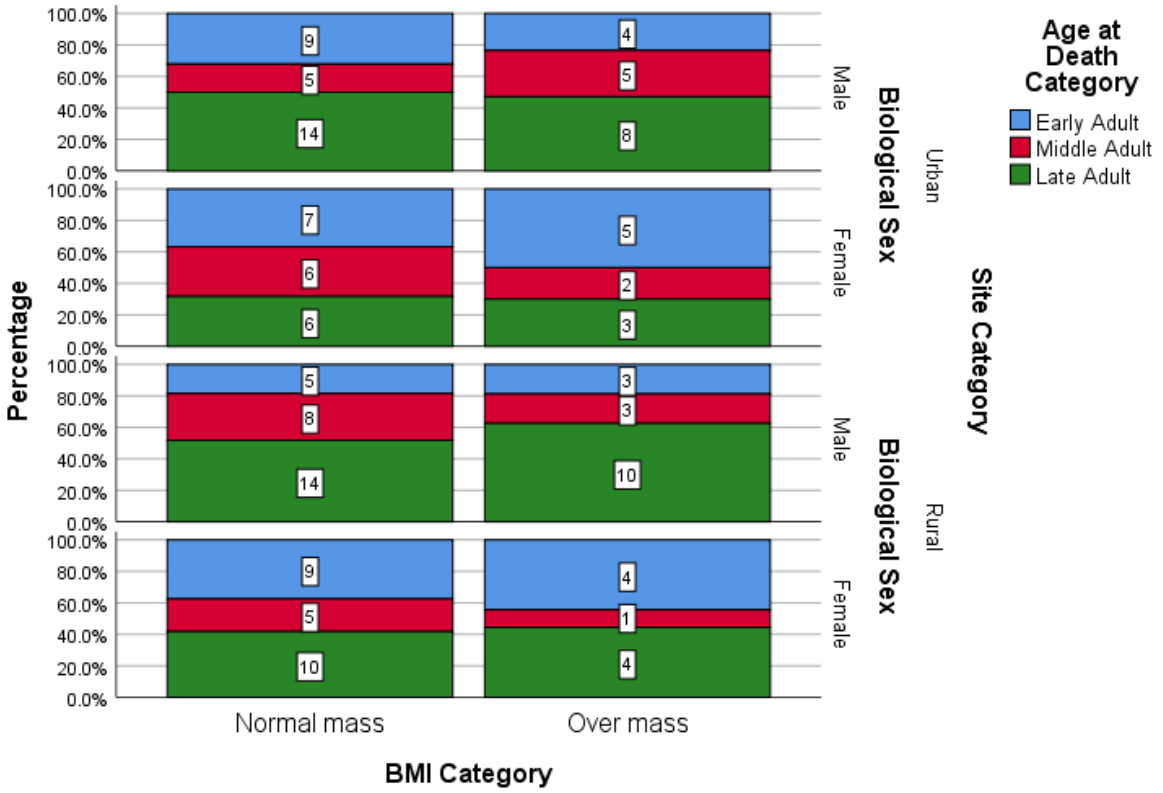

Figure 7.49 Distribution of body mass by site category, biological sex, and age at death. Actual count is in the box.

In total, 39.8% of normal mass and 30.8% of over mass individuals were diagnosed with probable general osteoarthritis (see Table 7.57/ Figure 7.50 - Figure 7.54). In the majority of cases, the difference between prevalence of joint condition in normal and over massed individuals is small, with exception of SOA which has a much greater difference. With the exception of DDD, the joint conditions display higher prevalences within the normal mass category.

Table 7.57 Distribution of diagnoses for the researched joint conditions by body mass category.

			DIAGNOSES			
			ABSENT		PROBABLE	
			N	%	N	%
JOINT CONDITIONS	OA	NORMAL	59	60.2	39	39.8
		OVER	36	69.2	16	30.8
	SOA	NORMAL	66	68.8	30	31.3
		OVER	40	81.6	9	18.4
	HOA	NORMAL	83	84.7	15	15.3
		OVER	48	92.3	4	7.7
	KOA	NORMAL	86	88.7	11	11.3
		OVER	47	90.4	5	9.6
	DDD	NORMAL	80	83.3	16	16.7
		OVER	38	80.9	9	19.1

Percentages based on body mass category.

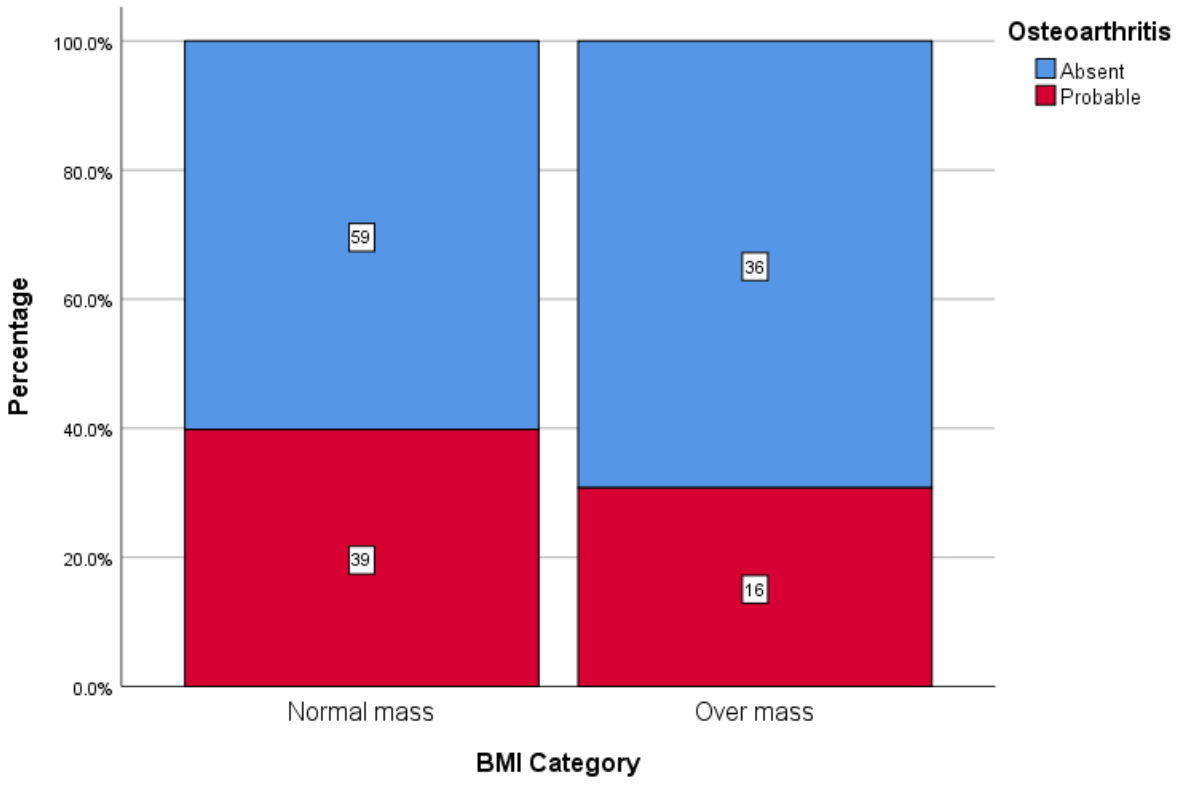

Figure 7.50 Distribution of diagnoses for osteoarthritis by BMI category. Count is indicated within the box.

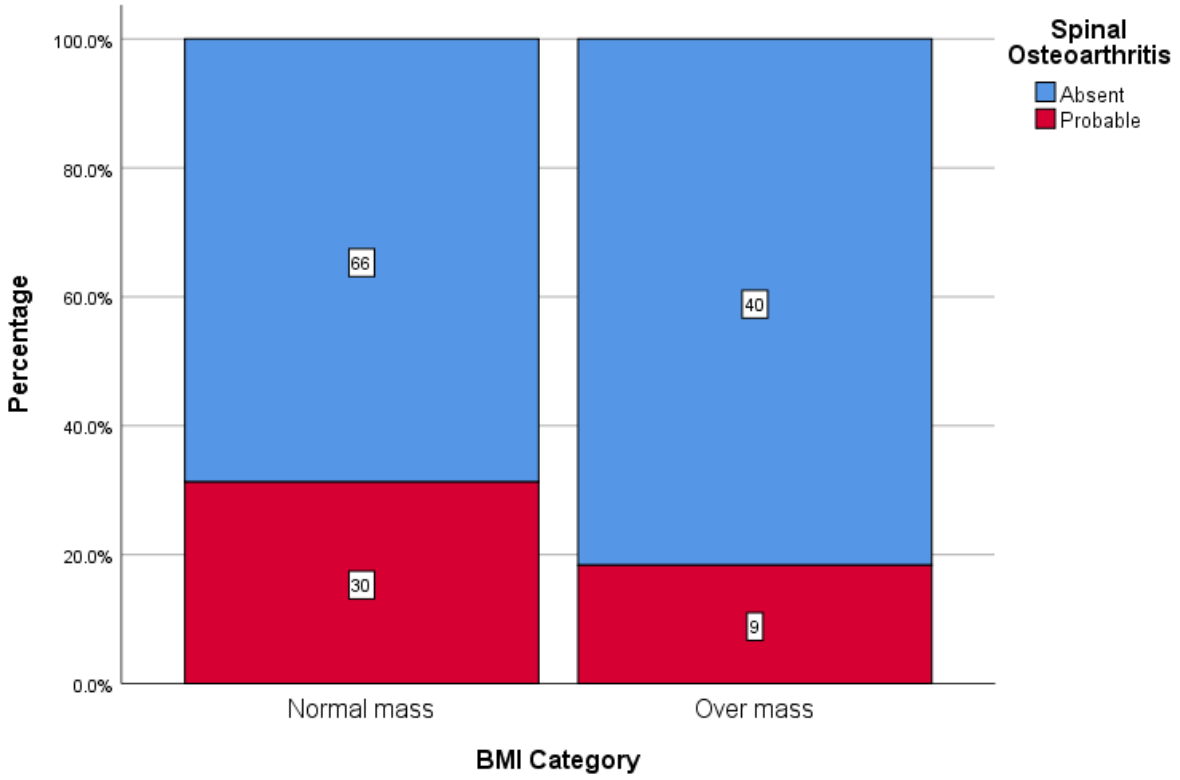

Figure 7.51 Distribution of diagnoses for spinal osteoarthritis by BMI category. Count is indicated within the box.

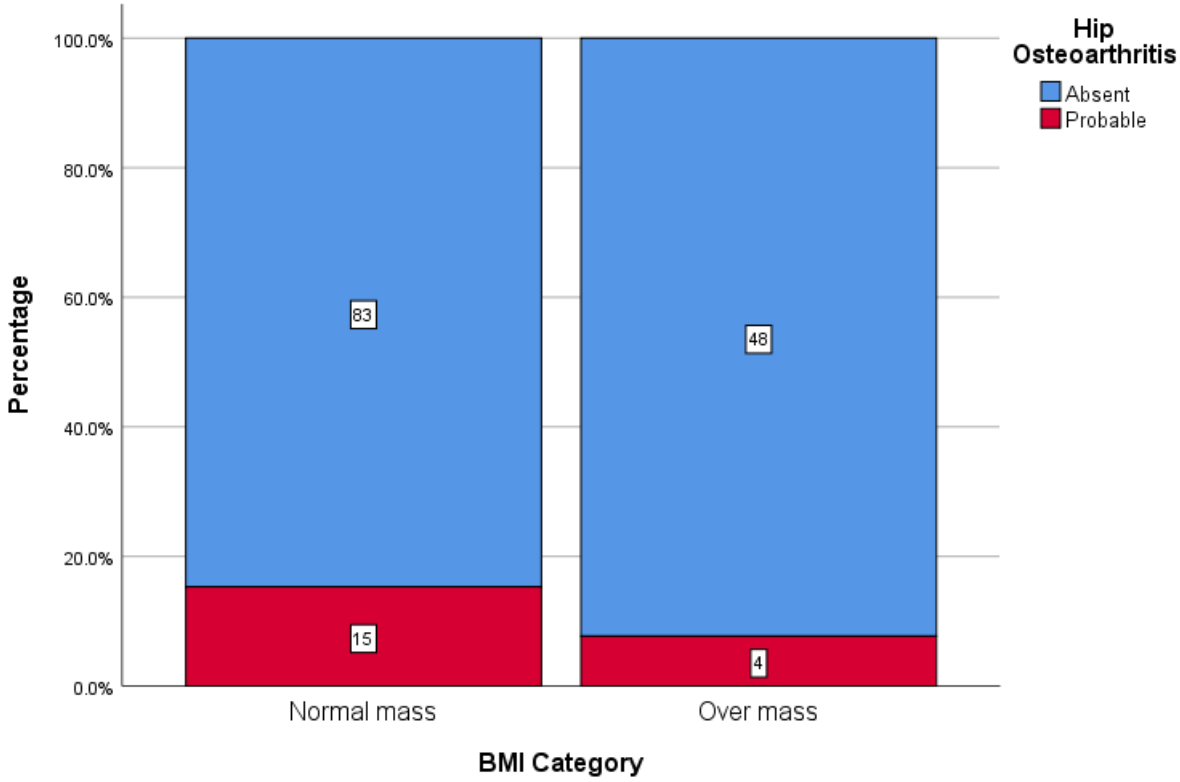

Figure 7.52 Distribution of diagnoses for hip osteoarthritis by BMI category. Count is indicated within the box.

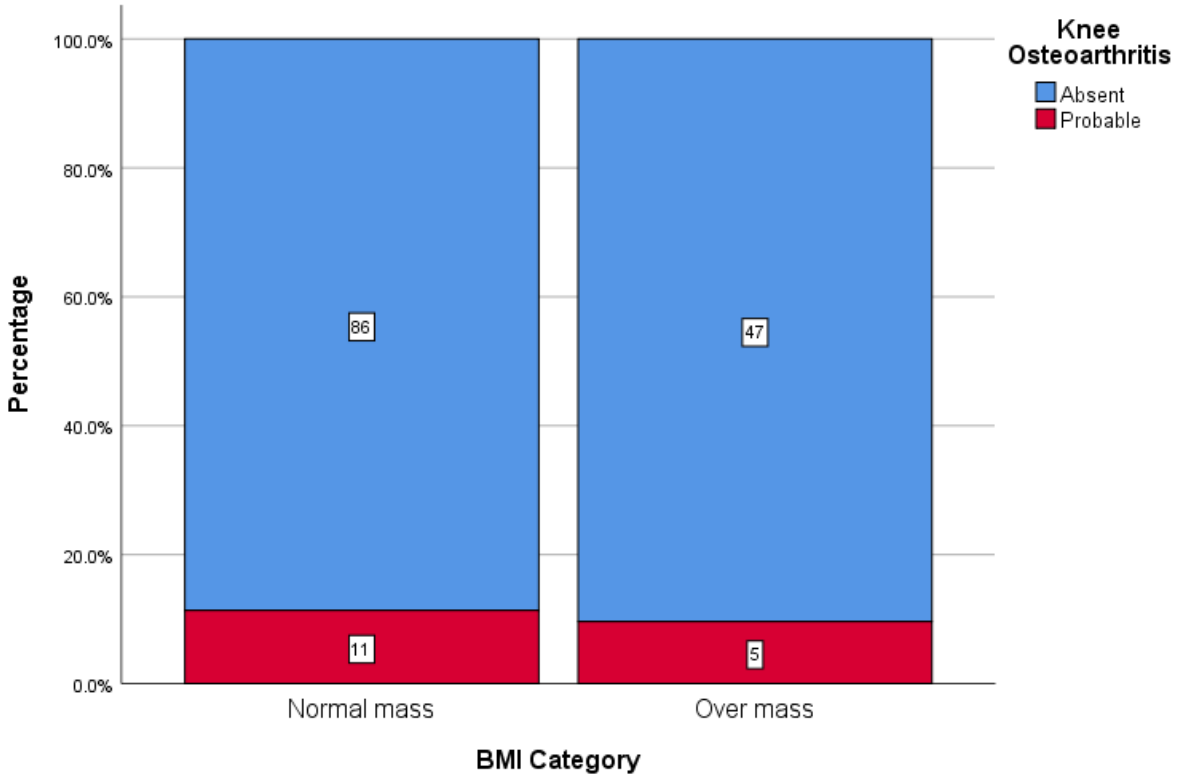

Figure 7.53 Distribution of diagnoses for knee osteoarthritis by BMI category. Count is indicated within the box.

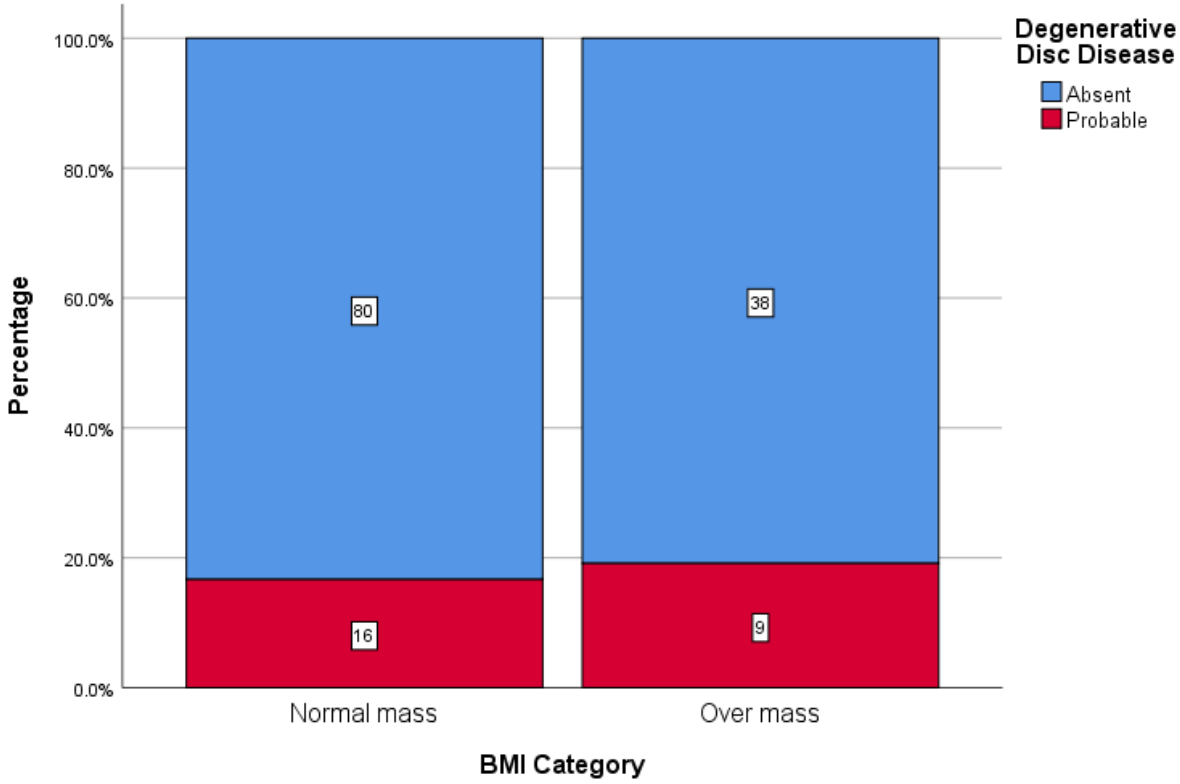

Figure 7.54 Distribution of diagnoses for degenerative disc disease by BMI category. Count is indicated within the box.

The distribution of the joint conditions by body mass category can be seen in Table 7.58. These percentages were equated using the diagnoses at each categorical level (ex. Early normal mass urban males) and used the total number of individuals that fell within said category from Table 7.56. For the urban males, the early adults showed no prevalence higher than 22.2% (OA: n = 2) for normal mass and 0.0% for over mass. The middle adults displayed the highest prevalence being 50.0% (DDD: n =2) for normal mass and 20.0% (n = 1) for all conditions, except for SOA, for over mass. The late adults had a range of prevalences from 50.0% (OA: n = 7) – 14.3% (KOA: n = 2) for normal mass and 50.0% (OA/HOA: n = 4) - 12.5% (SOA/KOA: n = 1) for over mass. For the urban females, the early adults displayed a high of 42.9% (OA: n = 3) for normal mass and 20.0% (OA/SOA/HOA: n = 1) for over mass. The middle adults all displayed a prevalence for 16.7 (n = 1) for normal mass, except for HOA, and a high of 50.0% (OA/SOA/HOA: n = 1) for over mass. The late adults displayed a range of 33.3% (OA/DDD: n = 2) – 16.7% (SOA/HOA/KOA: n = 1) for normal mass and only DDD had a prevalence (66.7%, n = 2) for over mass.

The early adult rural males had high of 60.0% (OA: n = 3) for normal mass and only DDD had a prevalence (33.3%, n = 1) for over mass. The middle adults had a high of 25.0% (OA/SOA: n = 2) for normal mass and all conditions, with the exception of KOA, had a prevalence of 33.3% (n = 1) for over mass. The late adults displayed a large range of 71.4% (OA: n = 10) – 7.1% (KOA: n = 1) for normal mass and 100.0% (OA/HOA: n = 4) – 25.0% (KOA: n = 1), with DDD having 0.0%. For rural females, the early adults had a high of 33.3% (DDD: n = 3) for normal mass and only DDD had a prevalence (25.0%, n = 1) for over mass. No middle adults had a prevalence within the normal mass category and all conditions, except for DDD, had 100.0% (n = 1) for over mass. The late adults had a range of 70.0% (OA: n = 7) - 10.0% (HOA/DDD: n = 1) for normal mass and 100.0% (OA/HOA: n = 4) – 25.0% (KOA: n = 1), with DDD having no prevalences.

Table 7.58 Multivariate distribution of probable diagnoses and body mass with inclusion of the site category, biological sex, and age at death variables.

			OSTEOARTHRITIS				SPINAL OSTEOARTHRITIS				HIP OSTEOARTHRITIS				KNEE OSTEOARTHRITIS				DEG.DISC DISEASE			
			NORMAL		OVER		NORMAL		OVER		NORMAL		OVER		NORMAL		OVER		NORMAL		OVER	
			N	%	N	%	N	%	N	%	N	%	N	%	N	%	N	%	N	%	N	%
URBAN	MALE	AGE AT DEATH EARLY	2	22.2	0	0.0	1	11.1	0	0.0	1	11.1	0	0.0	0	0.0	0	0.0	1	11.1	0	0.0
		AGE AT DEATH MID	1	25.0	1	20.0	1	25.0	0	0.0	0	0.0	1	20.0	0	0.0	1	20.0	2	50.0	1	20.0
		AGE AT DEATH LATE	7	50.0	4	50.0	6	42.9	1	12.5	4	28.6	4	50.0	2	14.3	1	12.5	4	28.6	2	25.0
	FEMALE		N	%	N	%	N	%	N	%	N	%	N	%	N	%	N	%	N	%	N	%
		AGE AT DEATH EARLY	3	42.9	1	20.0	1	14.3%	1	20.0	1	14.3	1	20.0	2	28.6	0	0.0	0	0.0	0	0.0
		AGE AT DEATH MID	1	16.7	1	50.0	1	16.7%	1	50.0	0	0.0	1	50.0	1	16.7	0	0.0	1	16.7	0	0.0
		AGE AT DEATH LATE	2	33.3	0	0.0	1	16.7%	0	0.0	1	16.7	0	0.0	1	16.7	0	0.0	2	33.3	2	66.7
RURAL	MALE		N	%	N	%	N	%	N	%	N	%	N	%	N	%	N	%	N	%	N	%
		AGE AT DEATH EARLY	3	60.0	0	0.0	2	40.0%	0	0.0	1	20.0	0	0.0	1	20.0	0	0.0	0	0.0	1	33.3
		AGE AT DEATH MID	2	25.0	1	33.3	2	25.0%	1	33.3	1	12.5	1	33.3	0	0.0	0	0.0	0	0.0	1	33.3
		AGE AT DEATH LATE	10	71.4	3	30.0	8	57.1%	1	10.0	5	35.7	3	30.0	1	7.1	1	10.0	3	21.4	1	10.0
	FEMALE		N	%	N	%	N	%	N	%	N	%	N	%	N	%	N	%	N	%	N	%
		AGE AT DEATH EARLY	1	11.1	0	0.0	1	11.1%	0	0.0	0	0.0	0	0.0	0	0.0	0	0.0	3	33.3	1	25.0
		AGE AT DEATH MID	0	0.0	1	100.0	0	0.0%	1	100.0	0	0.0	1	100.0	0	0.0	1	100.0	0	0.0	0	0.0
		AGE AT DEATH LATE	7	70.0	4	100.0	6	60.0%	3	75.0	1	10.0	4	100.0	3	30.0	1	25.0	1	10.0	0	0.0

Percentages are based on the age at death categories within the site category and biological sex groupings of each condition (ex. osteoarthritis: early adult urban male). These figures are based on the normal and over mass figures listed in Table 7.56.

In addition to the assignment of individuals to pre-determined body mass categories based on modern data, all individuals were also assigned to one of four categories created using interquartile range figures (see Table 7.59). These groupings reflected the relative mass of each individual with respect to the archaeological sample as a whole. Note that the values represent point data from the skeletal samples. Therefore, some samples may have the same value, but are split evenly between two quartiles. These IQR values have been used for the statistical testing.

Table 7.59 The point value ranges within each quartile.

	Q1 <25%	Q2 25-50%	Q3 50-75%	Q4 >75%
BMI	19.76-22.95	22.97-24.04	24.1-25.51	25.51-31.08

The duplicate 25.51 represents different individuals that have by been sorted into the different categories to ensure the quartiles have been evenly distributed. Q = quartile

In summation, distribution of body mass index categories was uniform between the urban and rural population with roughly one third of the entire sample population being over mass. Biological sex displayed a similar uniformity to the site category, with males having a slightly larger over mass population. Normal mass individuals had a similar age distribution, with middle adult having the lowest percentages amongst the females and late adult having the larger numbers within the males. Male over massed individuals largely fell within the late adult category, while females fell into the early adult category. Probable diagnoses were roughly similar between body mass index categories for osteoarthritis, knee osteoarthritis and degenerative disc disease, while spinal and knee osteoarthritis had a differently of roughly 10% between the body mass index categories.

7.4.2 Entheseal Changes (EC)

Entheseal changes were scored and allocated into one of three categories based on the criteria set forth in the methods section (see Section 6.1.4). Individuals with overall moderate entheseal change were the largest proportion at 50.9% (n = 89) (see Table 7.60/ Figure 7.55), with gracile changes being present in 40.0% (n = 70) and robust changes present in 9.14% (n = 16). The rural population follows a similar trend to the sample as a whole with individuals with moderate entheseal change representing the larger proportion (60.0%, n = 51), followed by gracile changes (28.2%, n = 24) and robust changes (11.8%, n = 10). The urban population

displayed a different trend with individuals with gracile entheseal changes representing the larger proportion (51.1%, n = 46), followed by those with moderate changes (42.2%, n = 38) and the robust changes (6.7%, n = 6). Thus, the urban population were more gracile overall than rural population.

Table 7.60 Distribution of entheseal change grades by site category.

		URBAN		RURAL		TOTAL	
		N	%	N	%	N	%
ENTHESEAL CHANGES	GRACILE	46	51.1%	24	28.2%	70	40.0%
	MODERATE	38	42.2%	51	60.0%	89	50.9%
	ROBUST	6	6.7%	10	11.8%	16	9.1%

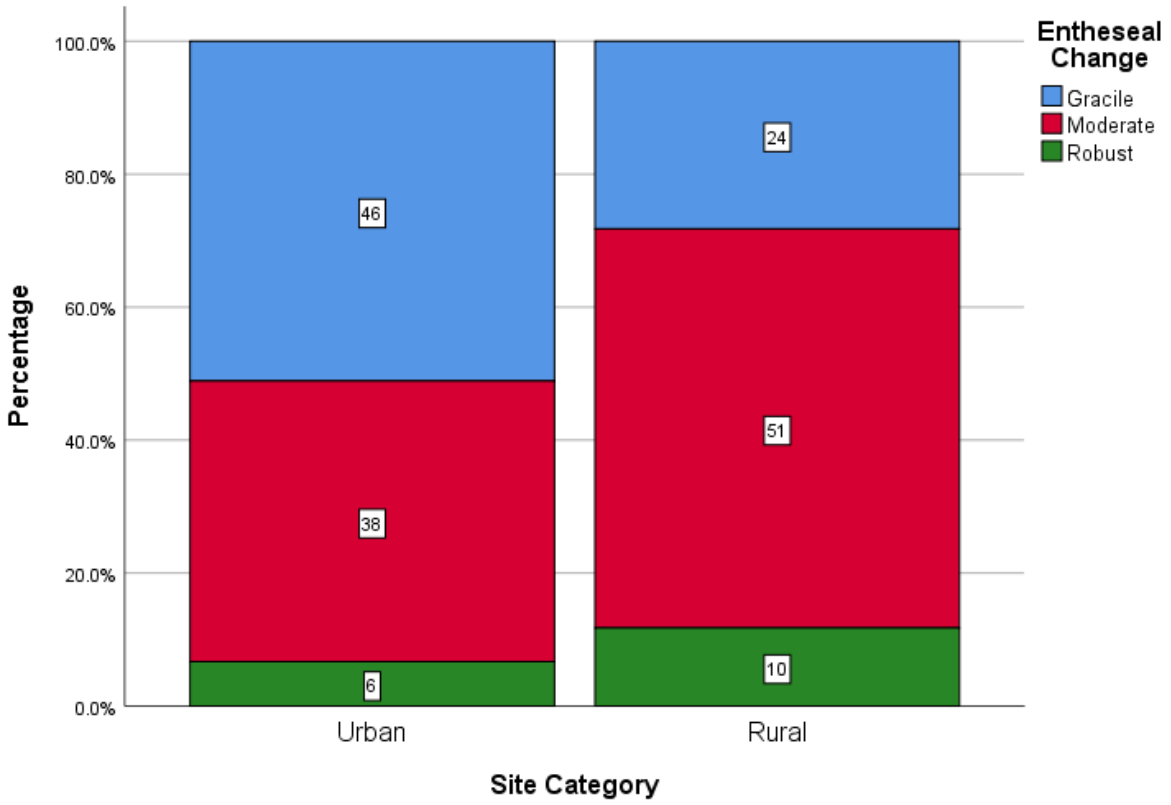

Figure 7.55 Distribution of entheseal change grades by site category. Count is displayed in the box of contained within each grouping.

Moderate entheseal changes continued to have the higher proportion when divided between males and females with 52.4% (n = 54) and 48.6% (n = 35) respectively (see Table 7.61/ Figure 7.56). For males, gracile changes were observed in 35.9% (n = 37) and robust

changes in 11.7% (n = 12). A greater proportion of females also had gracile changes (45.8%, n = 33) than robust changes (5.6%, n = 4), but females were also, overall, more gracile than males.

Table 7.61 Distribution of entheseal change grades by biological sex.

		MALE		FEMALE	
		N	%	N	%
ENTHESEAL CHANGES	GRACILE	37	35.9%	33	45.8%
	MODERATE	54	52.4%	35	48.6%
	ROBUST	12	11.7%	4	5.6%

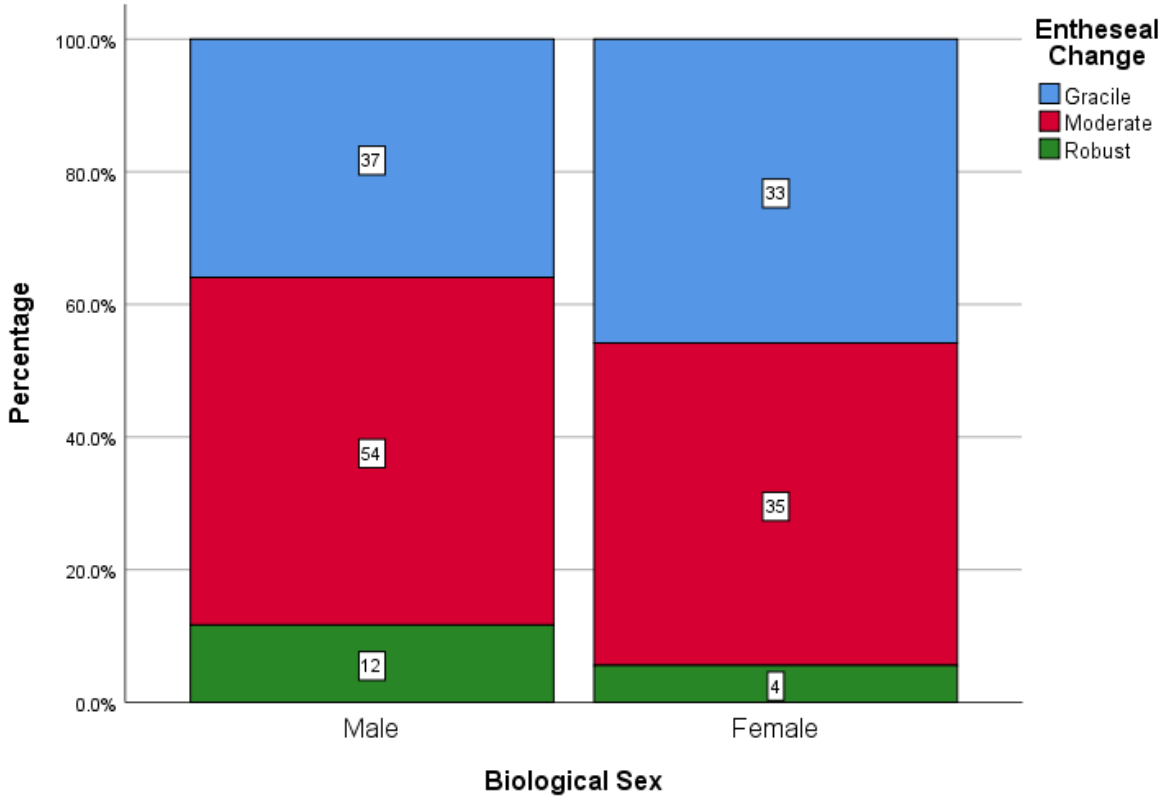

Figure 7.56 Proportional distribution of entheseal grades by biological sex and age at death. Count is indicated within the bars.

Further dividing the categories and comparing entheseal changes with age at death categories within the site categories displayed the same trend towards the majority of individuals presenting moderate EC, however, the urban site category displayed some deviation from this pattern (see Table 7.62/ Figure 7.57). The urban male percentages show a trend of increasing robusticity to the EC as the age category increases. The urban early adult population

displayed the largest percentage of gracile EC with this population at 59.4% (n = 19), and then had 37.5% (n = 12) moderate EC and 3.1% (n = 2) robust EC. The urban middle adult population had a percentage of 42.9% (n = 9) gracile EC, the largest percentage of moderate EC within this population at 52.4% (n = 11) and 4.8% (n = 1) robust EC. The urban late adult population had a percentage of 48.6% (n = 18) gracile EC, 40.5% (n = 15) moderate EC, and the displayed the largest percentage of robust EC within this population at 10.8% (n = 4). The rural males show an increasing robusticity of EC as the ages increase, however the early adults did show a higher level of individuals with moderate changes than the urban population. The early adult rural population displayed the largest percentage of gracile EC within this population at 33.3% (n = 8), then 58.3% (n = 14) moderate EC and 8.3% (n = 2) robust EC. The rural middle adult population had a percentage of 31.6% (n = 6) gracile entheseal changes, then displayed the largest percentage of moderate EC within this population at 63.2% (n = 12), and 5.3% (n = 1) of robust EC. The rural late adult population had a percentage of 23.8% (n = 10), then 59.5% (n = 25), and displayed the highest percentage of robust EC within this population at 16.7% (n = 7).

Table 7.62 Distribution of entheseal change grades by age at death category and site category.

		AGE AT DEATH CATEGORIES					
		EARLY		MIDDLE		LATE	
		N	%	N	%	N	%
URBAN	GRACILE	19	59.4	9	42.9	18	48.6
	MODERATE	12	37.5	11	52.4	15	40.5
	ROBUST	1	3.1	1	4.8	4	10.8
RURAL	GRACILE	8	33.3	6	31.	10	23.8
	MODERATE	14	58.3	12	63.2	25	59.5
	ROBUST	2	8.3	1	5.3	7	16.7

Percentages are by age at death and site categories.

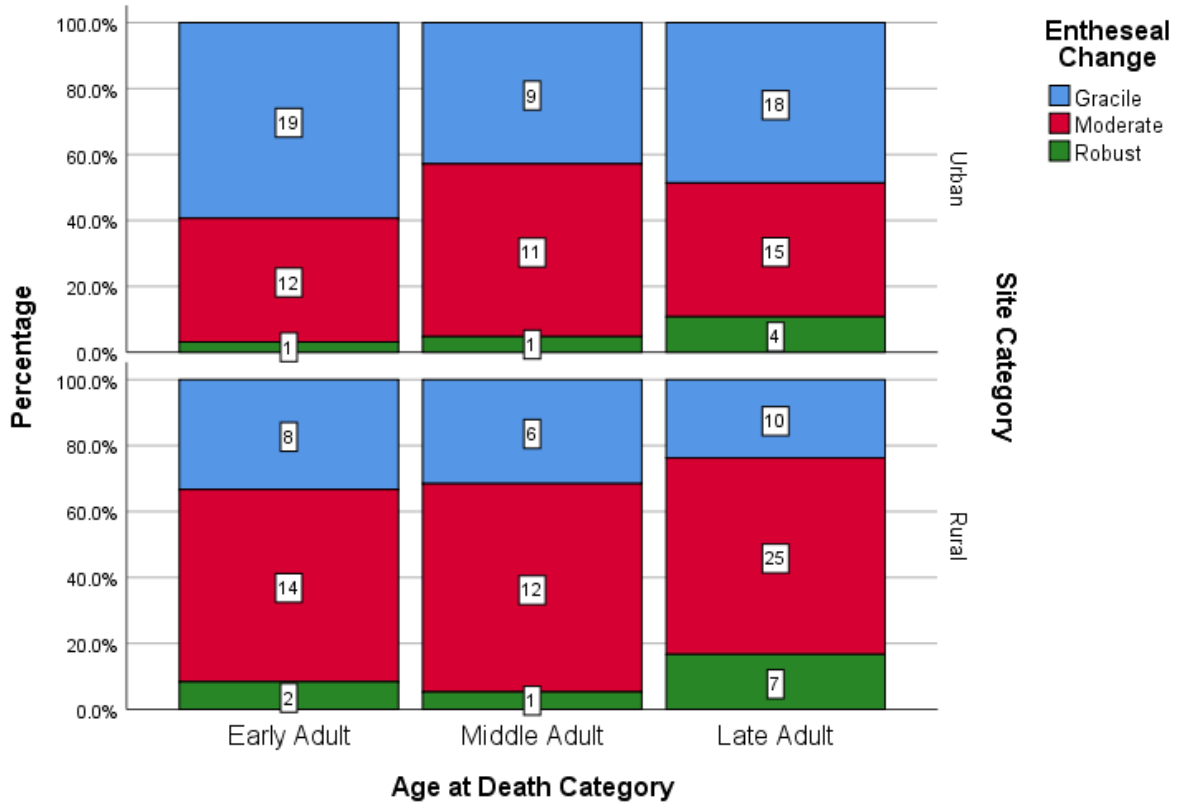

Figure 7.57 Proportional distribution of entheseal change grades by site category and age at death. Count is indicated within the bars.

Further breakdown shows that females are overrepresented with gracile EC in the early adult category and males are overrepresented with robust EC in the late adult category. A trend has emerged and continued to show that EC increases as age increases (see Table 7.63/ Figure 7.58 and Figure 7.59). Early adult males had percentages of 39.3% (n = 11) gracile EC, 53.6% (n = 15) moderate EC and 7.1% (n = 2) robust EC. Middle adult males had percentages of 36.4% (n = 8) gracile EC, 59.1% (n = 13) moderate EC and 4.5% (n = 1) robust EC. Late adult males had percentages of 34.0% (n = 18) gracile EC, 49.1% (n = 26) moderate EC and 17.0% (n = 9) robust EC. Early adult females had percentages of 57.1% (n = 16) gracile EC, 39.3% (n = 11) moderate EC and 3.6% (n = 1) robust EC. Middle adult females had percentages of 38.9% (n = 7) gracile EC, 55.6% (n = 10) moderate EC and 5.6% (n = 1) robust EC. Late adult females had percentages of 38.5% (n = 10) gracile EC, 53.8% (n = 14) moderate EC and 7.7% (n = 2) robust changes.

Table 7.63 Distribution of entheseal change grades by age category and biological sex.

		AGE AT DEATH CATEGORIES					
		EARLY		MIDDLE		LATE	
		N	%	N	%	N	%
MALES	GRACILE	11	39.3	8	36.4	18	34.0
	MODERATE	15	53.6	13	59.1	26	49.1
	ROBUST	2	7.1	1	4.5	9	17.0
FEMALES	GRACILE	16	57.1	7	38.9	10	38.5
	MODERATE	11	39.3	10	55.6	14	53.8
	ROBUST	1	3.6	1	5.6	2	7.7

Percentages are by biological sex and age at death.

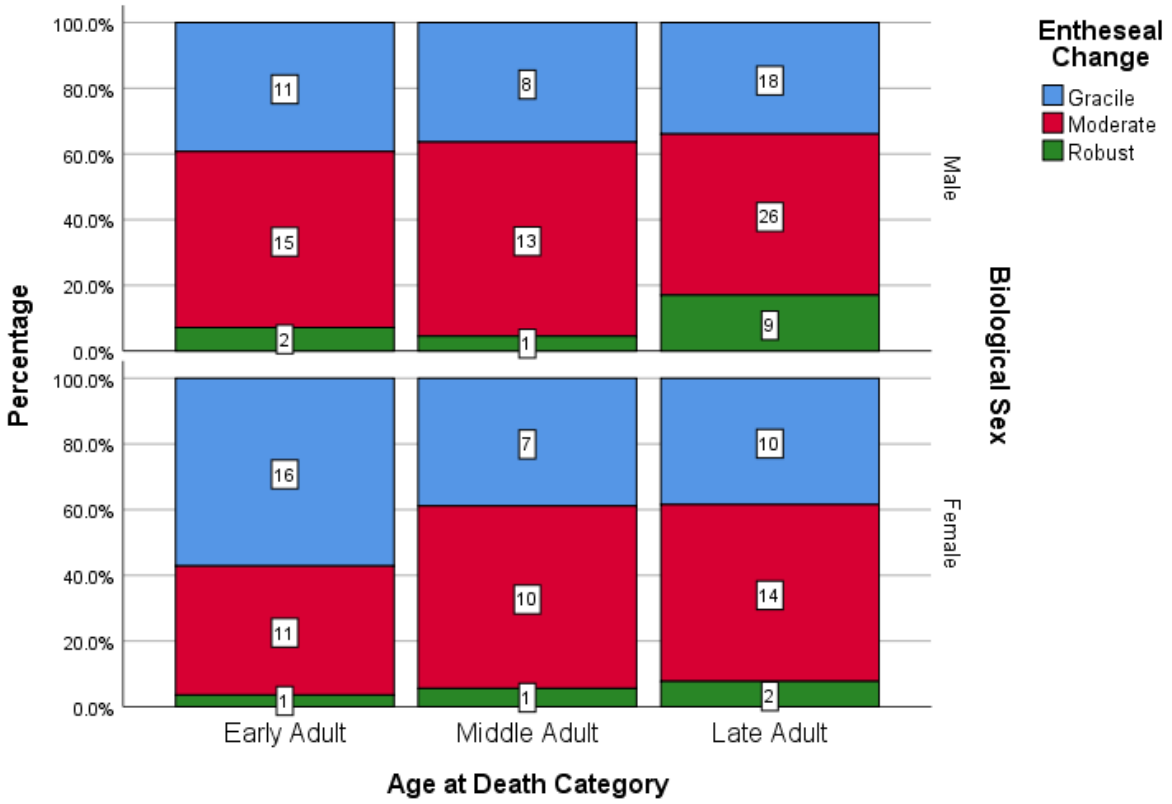

Figure 7.58 Proportional distribution of entheseal change grades by biological sex and age at death. Count is indicated within the bars.

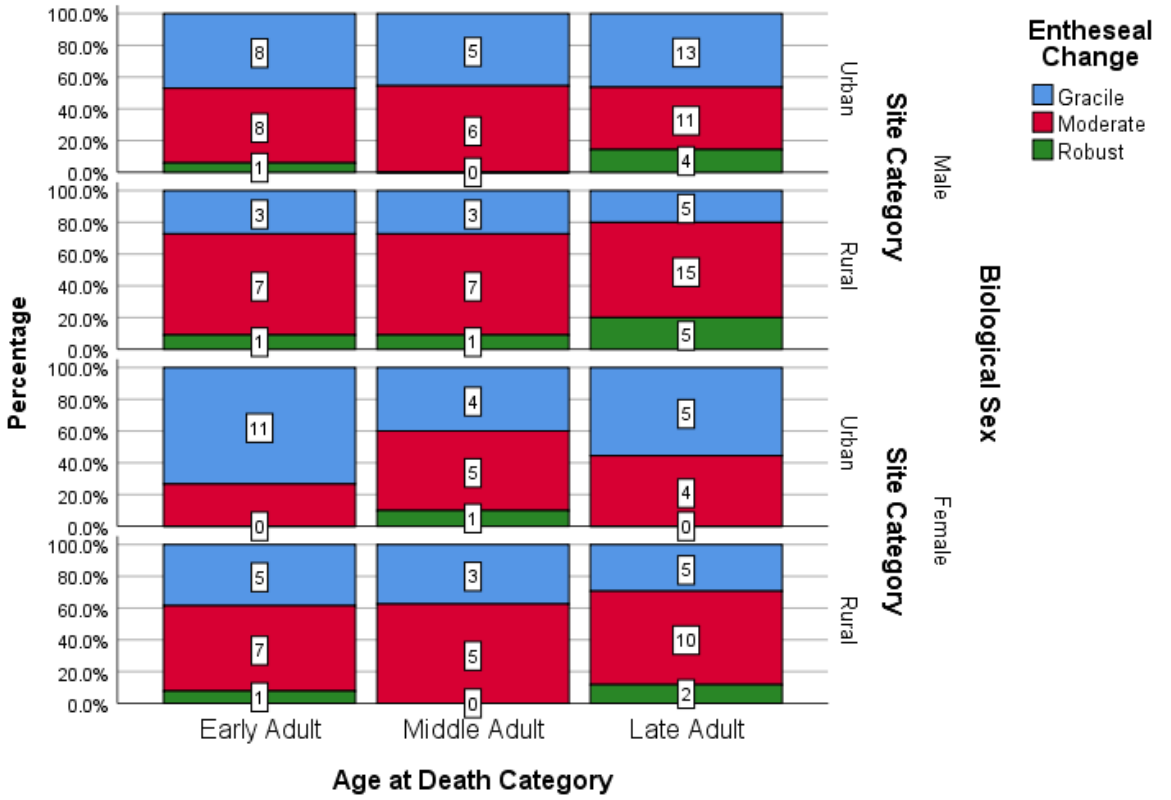

Figure 7.59 Proportional distribution of entheseal change grades by site category, biological sex, and age at death. Count is indicated within the bars.

A comparison of data concerning entheseal changes with prevalence of joint condition suggests that, in all cases, individuals with each form of joint condition tend to have moderate entheseal changes and those with robust changes the lowest prevalence (see Table 7.63/ Figure 7.60). The individuals with probable diagnoses for the joint conditions, with the exception of degenerative disc disease, had more robust changes in general, and a higher proportion of robust changes in particular, than the absent diagnoses. Cases of hip osteoarthritis had the higher proportion of individuals with robust entheseal changes at 22.73% (n = 5) and knee osteoarthritis had the highest proportion of individuals with gracile entheseal changes at 35.29% (n = 6).

Table 7.64 Distribution of entheseal change grades by joint conditions.

		OA		SOA		HOA		KOA		DDD	
		N	%	N	%	N	%	N	%	N	%
ENTHESEAL CHANGES	GRACILE	16	26.67	10	24.39	6	27.27	6	35.29	10	34.48
	MODERATE	34	56.67	24	58.54	11	50.00	9	52.94	18	62.07
	ROBUST	10	16.67	7	17.07	5	22.73	2	11.76	1	3.45

Percentages are by joint condition.

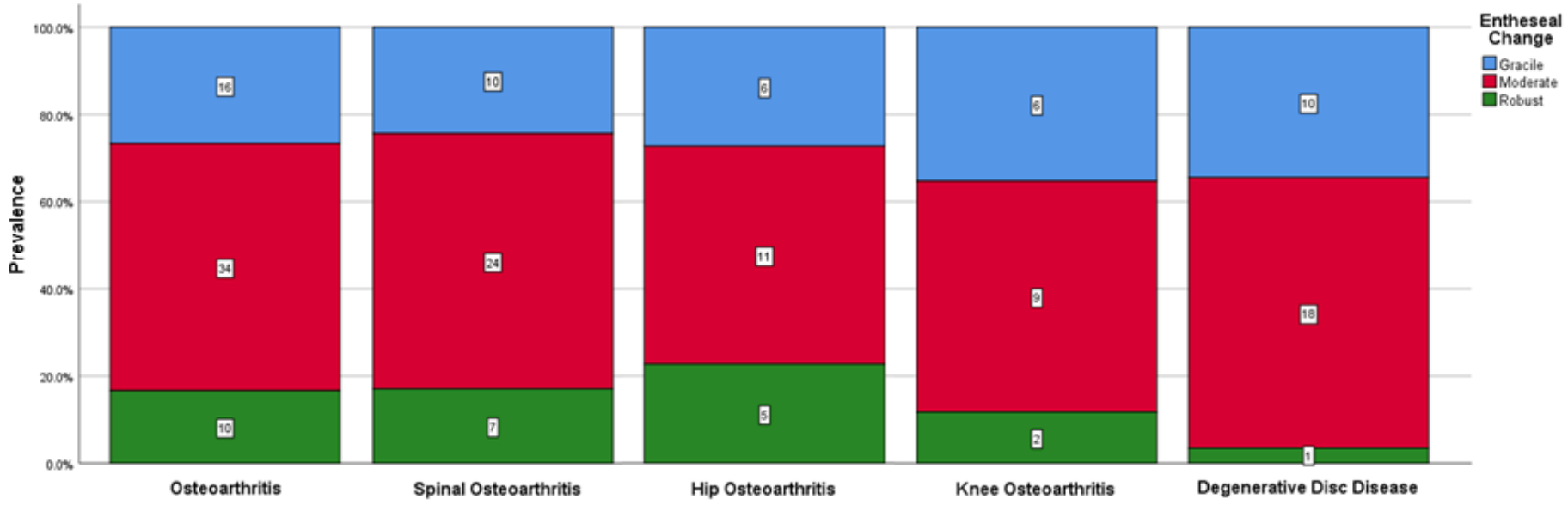

Figure 7.60 Proportional distribution of entheseal change grades by joint conditions. The bars represent only the probable diagnoses and count is indicated within the bar.

In summation, moderate entheseal changes were the most common grade found within this sample population. The urban population had the higher percentage of EC as moderate, while the rural population had the higher percentage of EC as gracile. Males had a higher percentage of EC as moderate, but the females displayed a more uniform percentage between gracile and moderate EC. While still rare, robust EC displayed a trend of increasing with age. Gracile changes also increased in the late adult category within both site categories, but the urban saw a larger proportion of gracile changes. Males and females displayed uniform distribution patterns, with exception of early adult females, who had a greater proportion of gracile changes and late adult males, who presented more robust changes. Individuals with joint condition were, overall, more robust than those without. The group with probable cases of hip osteoarthritis had the largest proportion of robust changes. In contrast, across the joint conditions the proportional distribution between gracile and moderate changes remained relatively similar.

7.4.3 Cross-sectional Indices (CSG)

The cross-sectional indices focus on two skeletal locations, femoral subtrochanteric and midshaft regions, and comprise of four variables: shape, area, robusticity and polar second moment of area. Comparison of the cross-sectional indices was completed using point estimates, as well as the same data placed into groups based on interquartile ranges. Note that, as with the body mass index interquartile ranges, the values represent point data from the skeletal samples.

The cross-sectional indices showed little deviation between the urban and rural site categories for the femoral subtrochanteric variables (see Table 7.64). The femoral midshaft variables showed greater variation between the site categories with the urban population typically have a larger mean, but smaller range.

Table 7.65 Mean, range and standard deviations for the cross-sectional variables by site category.

		MEAN	RANGE		STD DEV
			MIN	MAX	
URBAN	FSS	.87	.70	1.08	.07
	FSR	67.73	57.74	80.40	4.34
	FSJ	241.92	126.20	345.94	42.99
	FSA	1146.25	859.16	1574.37	143.14
	FMS	1.03	.86	1.35	.09
	FMR	61.01	52.69	76.53	3.92
	FMJ	163.65	71.54	296.95	32.46
	FMA	932.18	704.86	1533.77	123.03
RURAL	FSS	.87	.68	1.06	.08
	FSR	67.73	56.76	76.36	3.80
	FSJ	238.13	88.61	317.08	41.77
	FSA	1143.52	728.29	1468.46	136.18
	FMS	1.02	.76	1.29	.09
	FMR	62.06	52.63	79.11	4.37
	FMJ	173.49	84.95	281.98	37.42
	FMA	963.14	602.24	1368.47	144.61

Table 7.66 Mean, range and standard deviation for the cross-sectional variables by biological sex.

		MEAN	RANGE		STD DEV
			MIN	MAX	
MALES	FSS	.88	.71	1.08	.37
	FSR	67.01	57.74	77.54	3.61
	FSJ	248.21	153.66	341.11	32.81
	FSA	1181.27	865.98	1574.37	127.39
	FMS	1.04	.85	1.35	.09
	FMR	61.12	52.69	76.53	3.72
	FMJ	178.33	104.78	296.95	29.37
	FMA	984.24	721.30	1533.77	120.99
FEMALES	FSS	.86	.71	1.08	.37
	FSR	68.73	56.76	80.4	4.49
	FSJ	228.91	88.61	345.94	50.83
	FSA	1094.55	728.29	1457.51	140.57
	FMS	1.01	.76	1.20	.09
	FMR	62.03	52.63	79.11	4.69
	FMJ	153.92	71.54	281.98	39.05
	FMA	893.17	602.24	1368.24	134.65

There is no apparent variation in cross-sectional indices between the biological sex or age at death categories (see Figure 7.61 and Figure 7.62). Males and females have similar means for the individual cross-sectional variable, with only minor variations in skew, range, and outliers. For instance, femoral midshaft polar SMA illustrates how the female data range grows with the increasing age, while the males have relatively smaller and more similar ranges.

The following two example scatterplots demonstrate how the indices are spread by biological sex across the age at death spectrum (see Figure 7.61 and Figure 7.62). Across the cross-sectional indices, the R^2 linear values deviate and display differing strength relationships. The correlations and relationships between these variables will be explored in more depth in the following section. The scatterplots display parallel groupings of individuals of both sexes consistently throughout the indices, with no relationship between the comparisons. For the further comprehensive break down of the cross-sectional variables by biological sex and age at death see the appendix Section Cross-Sectional Indices.

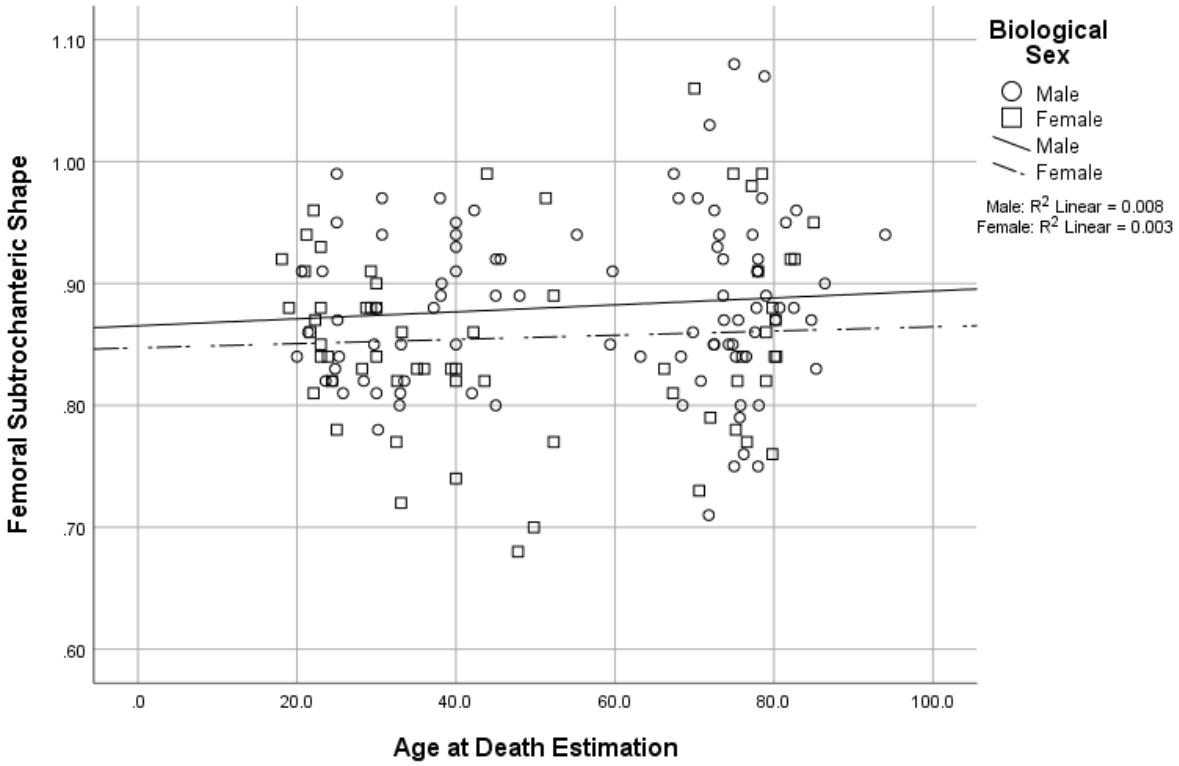

Figure 7.61 Distribution of the sample population by age at death and femoral subtrochanteric shape. The samples have further been identified by biological sex with R^2 values.

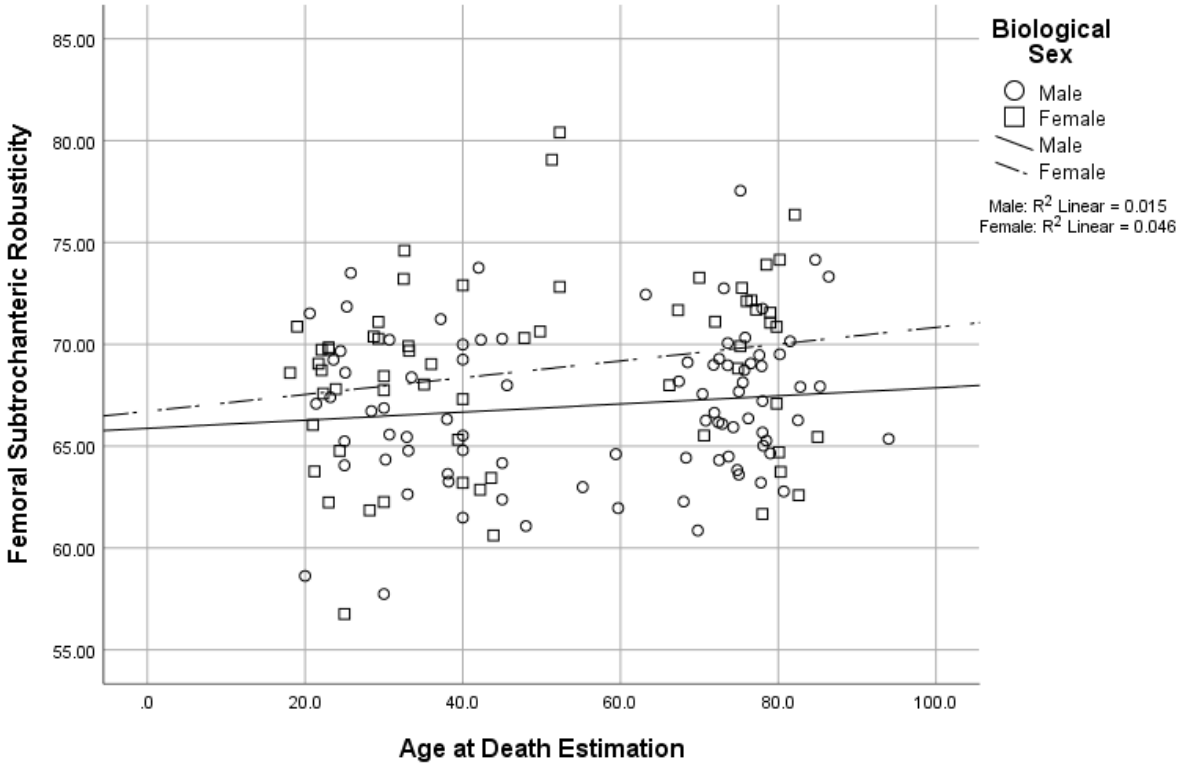

Figure 7.62 Distribution of the sample population by age at death and femoral subtrochanteric robusticity. The samples have further been identified by biological sex with R^2 values.

A comparison of the cross-sectional variables and the joint conditions demonstrates that the means and ranges are similar between individuals with and without the joint conditions, with the probable cases falling within the ranges of the absent categories (see Table 7.67 - Table 7.71/ Figure 7.63 - Figure 7.70). The whiskers representing Q1/Q4 are stay uniform within each variable graph for absent diagnoses, however regarding the probable diagnoses these whiskers fluctuate in size and direction by joint condition. The outliers within the absent diagnoses remain consistent, and the outliers for the probable diagnoses are more variable with only a few individuals appearing in multiple comparisons. The initial examination of the data comparing the diagnoses with the cross-sectional indices does not indicate any significant differences between them. This can be further explored in the following sections (see Section 7.4.4.2), in which statistical testing is used to compare the variables with the joint conditions.

Table 7.67 Mean, range and standard deviation of the cross-sectional indices by osteoarthritis diagnosis.

			MEAN	RANGE		STD DEV
				MIN	MAX	
OSTEOARTHRITIS	ABSENT	FSS	.89	.73	1.08	.09
		FSR	67.33	56.76	80.40	4.23
		FSJ	236.18	88.61	345.94	47.04
		FSA	1129.55	728.29	1574.37	148.59
		FMS	1.02	.76	1.35	.10
		FMR	61.19	52.63	79.11	4.12
		FMJ	165.85	71.54	296.95	35.02
		FMA	935.78	602.24	1533.77	135.29
	PROBABLE	FSS	.86	.68	.99	.06
		FSR	68.48	60.62	76.36	3.71
		FSJ	246.67	155.60	317.08	32.08
		FSA	1173.36	857.82	1420.13	116.60
		FMS	1.03	.86	1.27	.09
		FMR	62.08	53.03	75.43	4.20
		FMJ	172.70	91.51	281.98	35.27
		FMA	966.77	721.24	1356.25	130.82

Table 7.68 Mean, range and standard deviation of the cross-sectional indices by spinal osteoarthritis diagnosis.

			MEAN	RANGE		STD DEV
				MIN	MAX	
SPINAL OSTEOARTHRITIS	ABSENT	FSS	.86	0.68	.99	.06
		FSR	67.55	56.76	80.40	4.16
		FSJ	237.91	88.61	345.94	44.70
		FSA	1137.43	728.29	1574.37	146.13
		FMS	1.02	.76	1.35	.10
		FMR	61,39	52.63	79.11	4.29
		FMJ	166.90	71.54	296.95	34.77
		FMA	942.24	602.24	1533.77	137.10
	PROBABLE	FSS	.91	.73	1.08	.09
		FSR	68.59	60.62	76.36	3.74
		FSJ	247.91	155.60	317.08	33.62
		FSA	1172.69	857.82	1389.80	113.23
		FMS	1.03	.89	1.27	.09
		FMR	62.01	54.44	75.43	4.03
		FMJ	172.17	91.51	281.98	37.87
		FMA	962.02	721.24	1356.25	133.71

Table 7.69 Mean, range and standard deviation of the cross-sectional indices by hip osteoarthritis diagnosis.

			MEAN	RANGE		STD DEV
				MIN	MAX	
HIP OSTEOARTHRITIS	ABSENT	FSS	.87	.68	1.08	.07
		FSR	67.68	56.76	80.40	4.17
		FSJ	238.92	88.61	345.94	43.69
		FSA	1138.77	728.29	1574.27	140.87
		FMS	1.02	.76	1.35	.09
		FMR	61.43	52.63	79.11	4.21
		FMJ	167.24	71.54	296.95	36.32
		FMA	941.17	602.24	1533.77	136.27
	PROBABLE	FSS	.85	.76	.97	.06
		FSR	68.15	62.28	73.50	3.36
		FSJ	248.48	193.11	298.85	29.98
		FSA	1192.50	10002.38	1420.13	120.54
		FMS	1.03	.86	1.21	.10
		FMR	62.09	54.31	68.69	3.77
		FMJ	176.61	140.57	226.91	24.03
		FMA	989.66	800.29	1233.58	110.41

Table 7.70 Mean, range and standard deviation of the cross-sectional indices by knee osteoarthritis diagnosis.

			MEAN	RANGE		STD DEV
				MIN	MAX	
KNEE OSTEOARTHRITIS	ABSENT	FSS	.87	.68	1.08	.07
		FSR	67.11	56.76	80.40	4.14
		FSJ	240.32	88.61	345.95	43.42
		FSA	1146.01	728.29	1574.37	142.63
		FMS	1.02	.76	1.35	.09
		FMR	61.39	52.63	79.11	4.07
		FMJ	168.49	71.54	296.95	35.33
		FMA	945.77	602.24	1533.77	135.88
	PROBABLE	FSS	.88	.80	.99	.06
		FSR	68.11	60.62	72.82	4.02
		FSJ	237.17	155.60	292.74	34.25
		FSA	1138.86	857.82	1333.25	122.56
		FMS	1.06	.91	1.27	.09
		FMR	61.88	53.03	69.30	4.83
		FMJ	167.43	109.73	248.21	35.94
		FMA	940.72	721.73	1232.80	129.61

Table 7.71 Mean, range and standard deviation of the cross-sectional indices by degenerative disc disease diagnosis.

			MEAN	RANGE		STD DEV
				MIN	MAX	
DEGENERATIVE DISC DISEASE	ABSENT	FSS	.87	.68	1.08	.07
		FSR	68.07	57.74	80.40	3.99
		FSJ	240.83	10.388	345.94	42.70
		FSA	1154.24	857.82	1574.37	140.21
		FMS	1.02	.76	1.35	.09
		FMR	61.75	52.63	79.11	4.24
		FMJ	166.90	71.54	296.95	34.77
		FMA	952.86	602.24	1533.77	141.51
	PROBABLE	FSS	.87	.72	1.07	.08
		FSR	66.87	56.76	72.90	4.39
		FSJ	240.25	88.61	294.16	41.38
		FSA	1114.56	728.29	1388.27	134.60
		FMS	1.03	.88	1.29	.10
		FMR	60.79	52.94	68.11	4.22
		FMJ	172.17	91.51	281.98	37.87
		FMA	923.64	729.03	1126.52	111.71

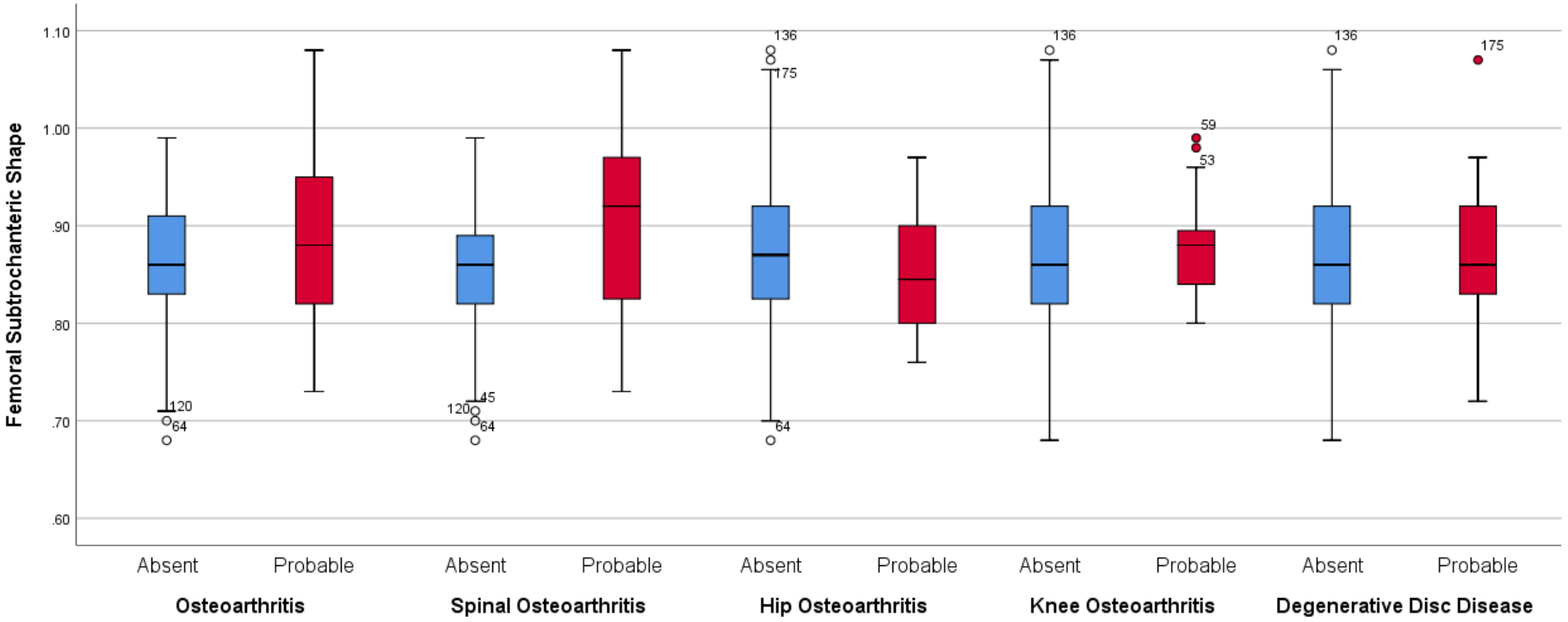

Figure 7.63 Boxplot of femoral subtrochanteric shape across the joint conditions. Note the relatively standard mean and uniform skew.

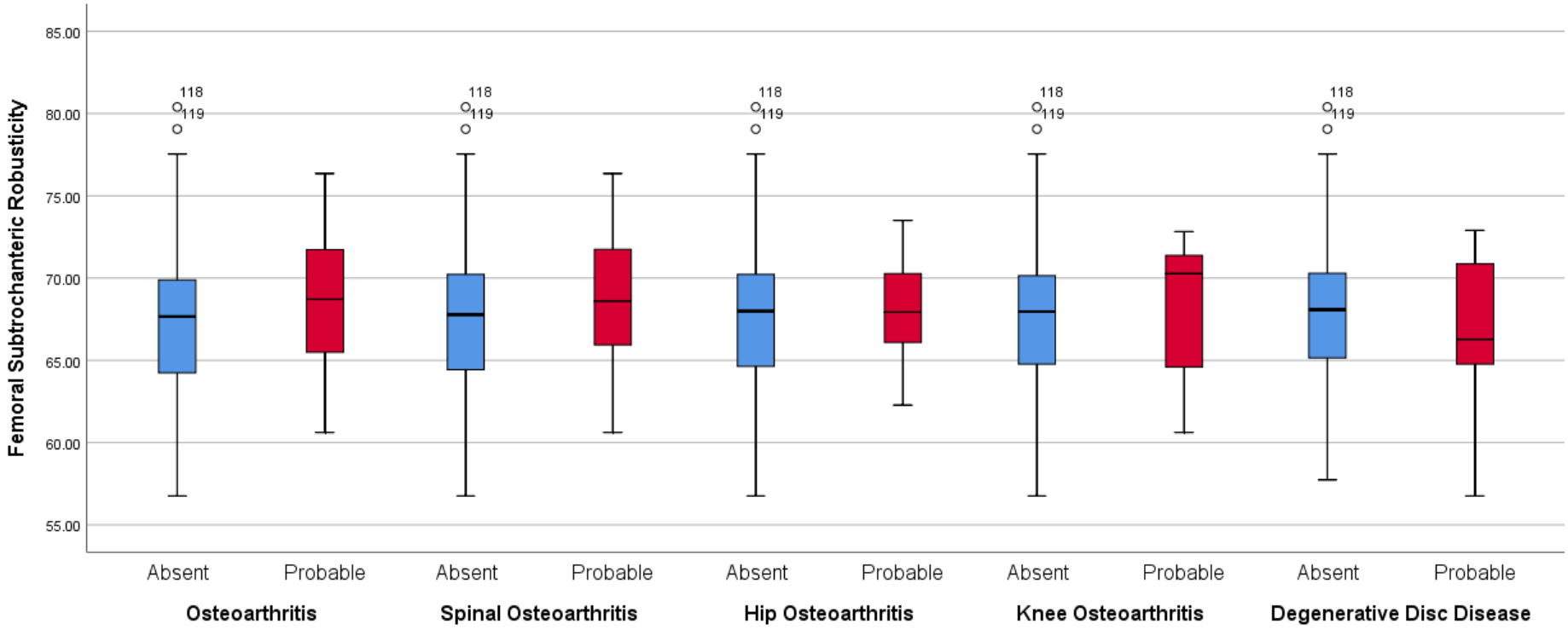

Figure 7.64 Boxplot of femoral subtrochanteric robusticity across the joint conditions. Note the relatively standard mean and uniform skew.

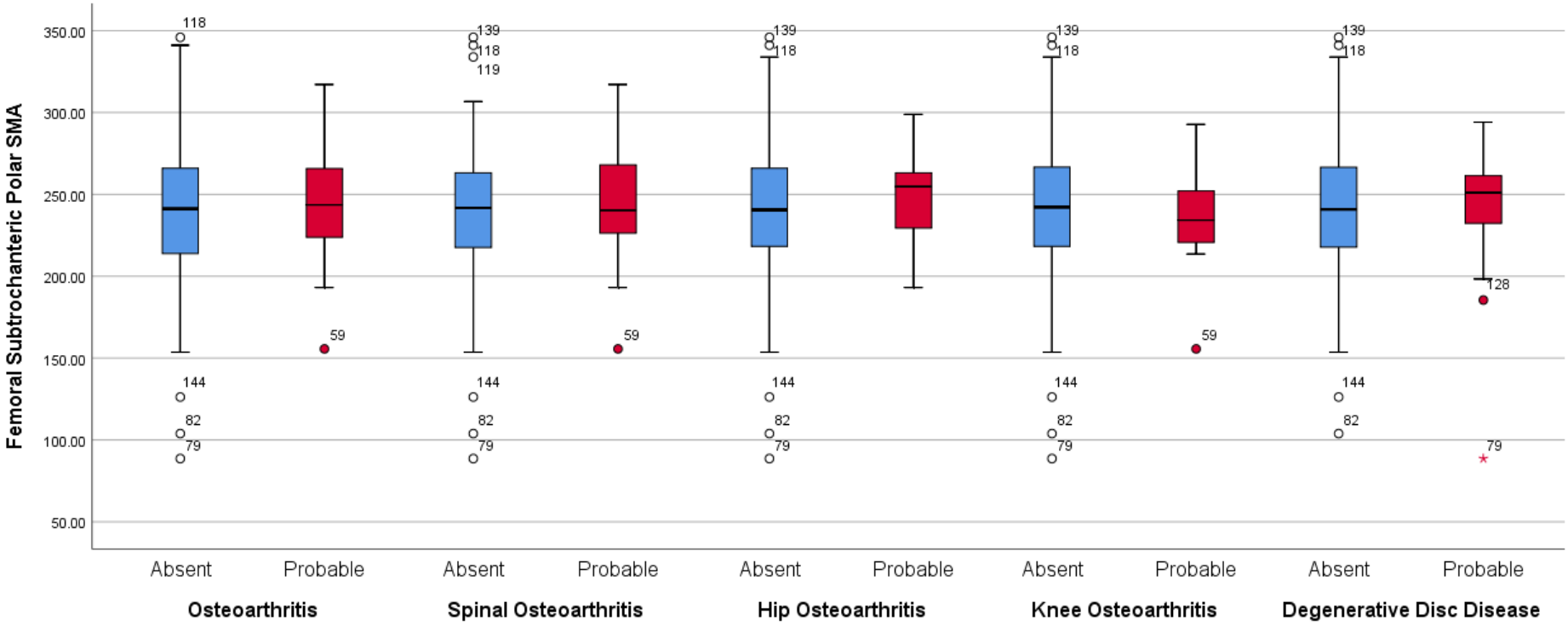

Figure 7.65 Boxplot of femoral subtrochanteric polar SMA across the joint conditions. Note the relatively standard mean and uniform skew.

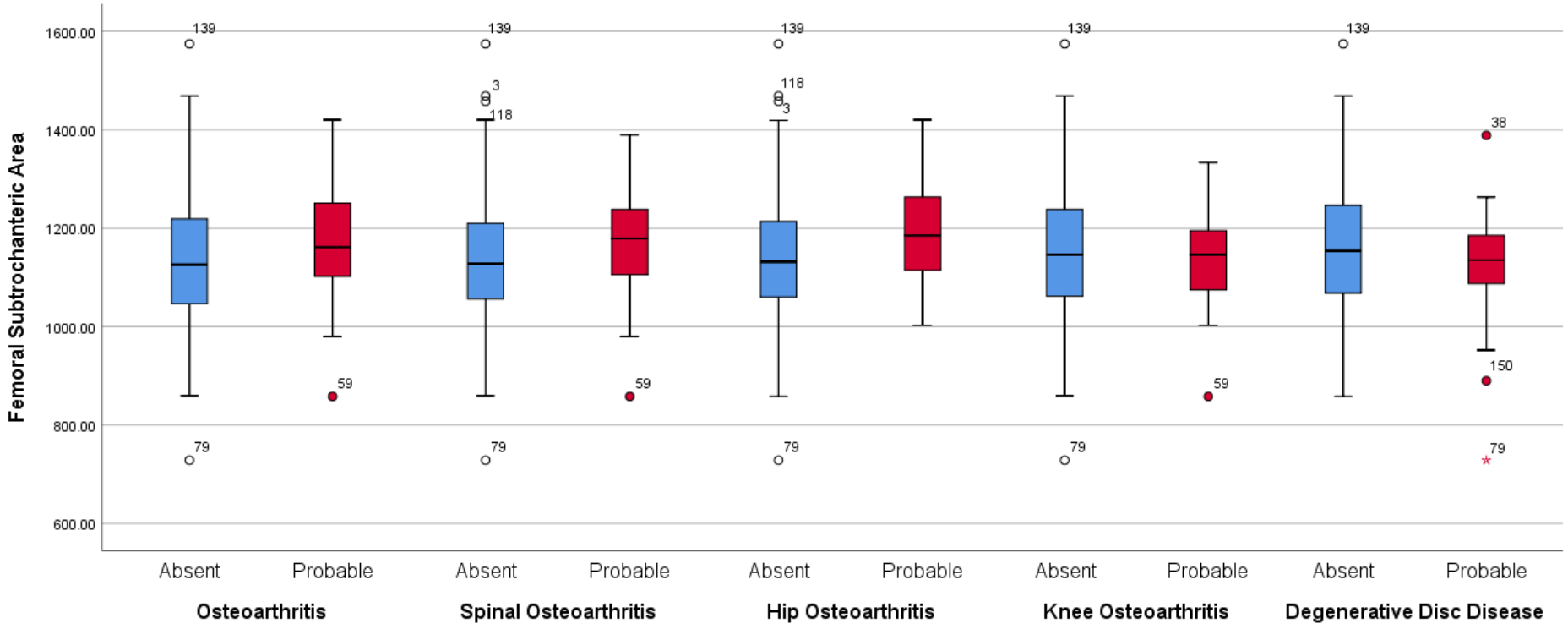

Figure 7.66 Boxplot of femoral subtrochanteric area across the joint conditions. Note the relatively standard mean and uniform skew.

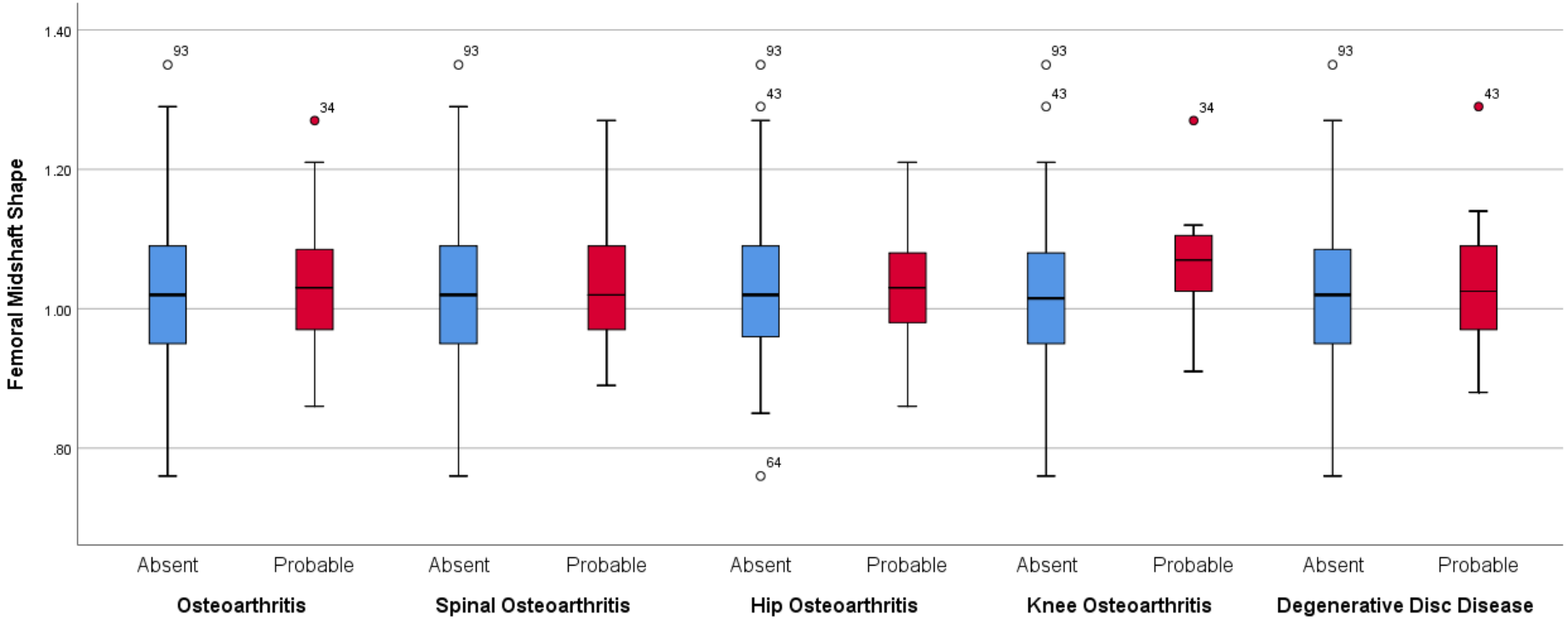

Figure 7.67 Boxplot of femoral midshaft shape across the joint conditions. Note the relatively standard mean and uniform skew.

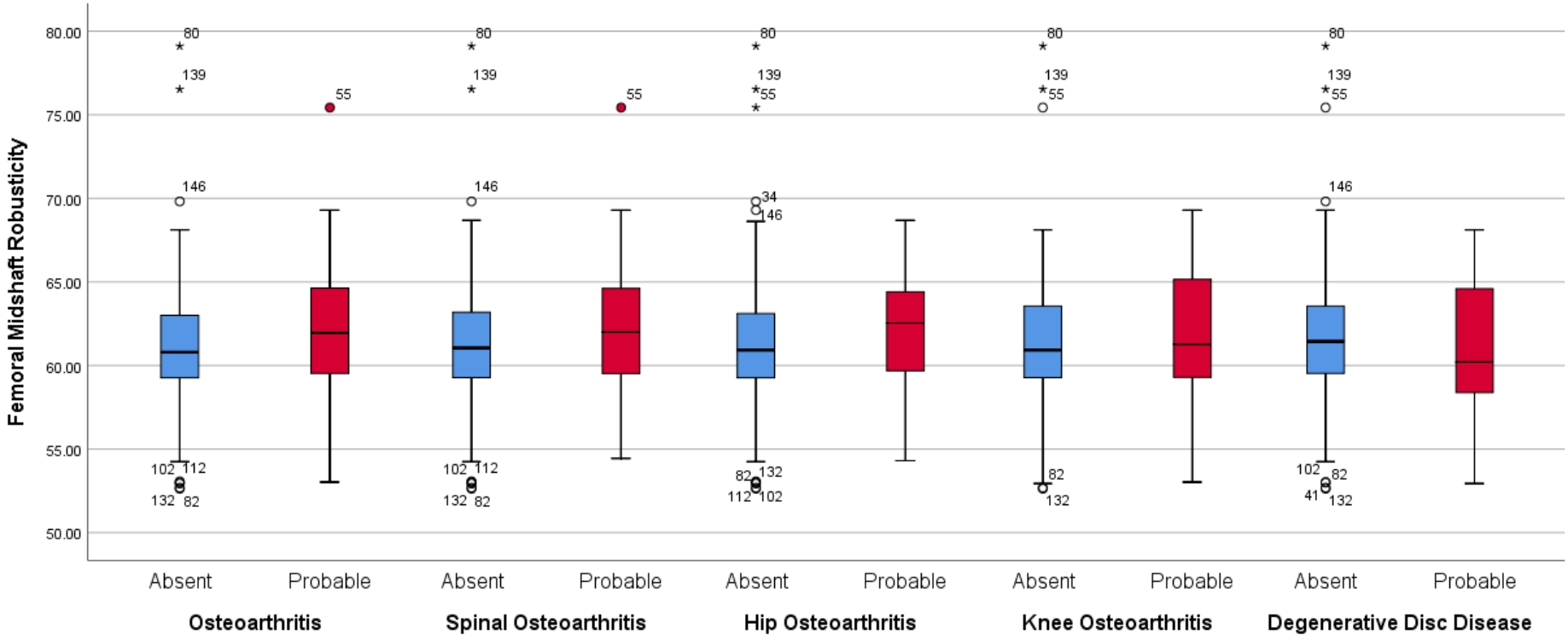

Figure 7.68 Boxplot of femoral midshaft robusticity across the joint conditions. Note the relatively standard mean and uniform skew.

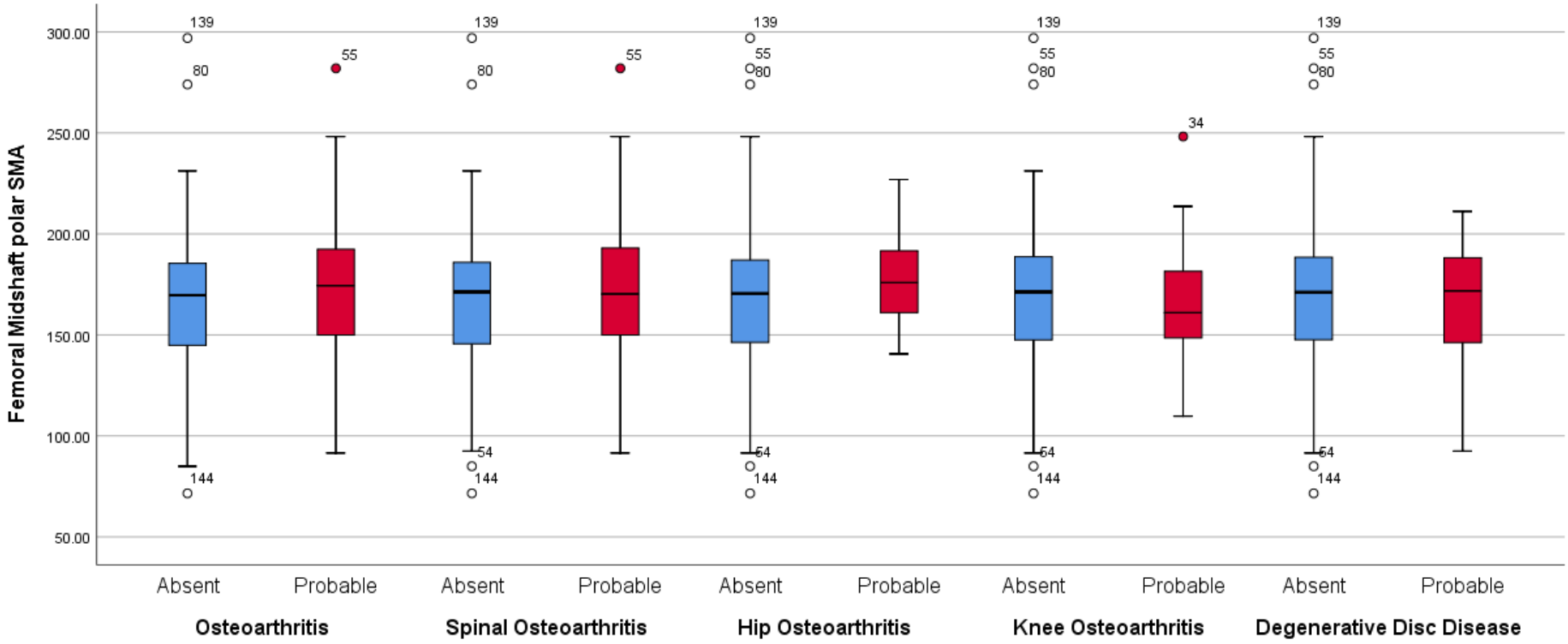

Figure 7.69 Boxplot of femoral midshaft polar SMA across the joint conditions. Note the relatively standard mean and uniform skew.

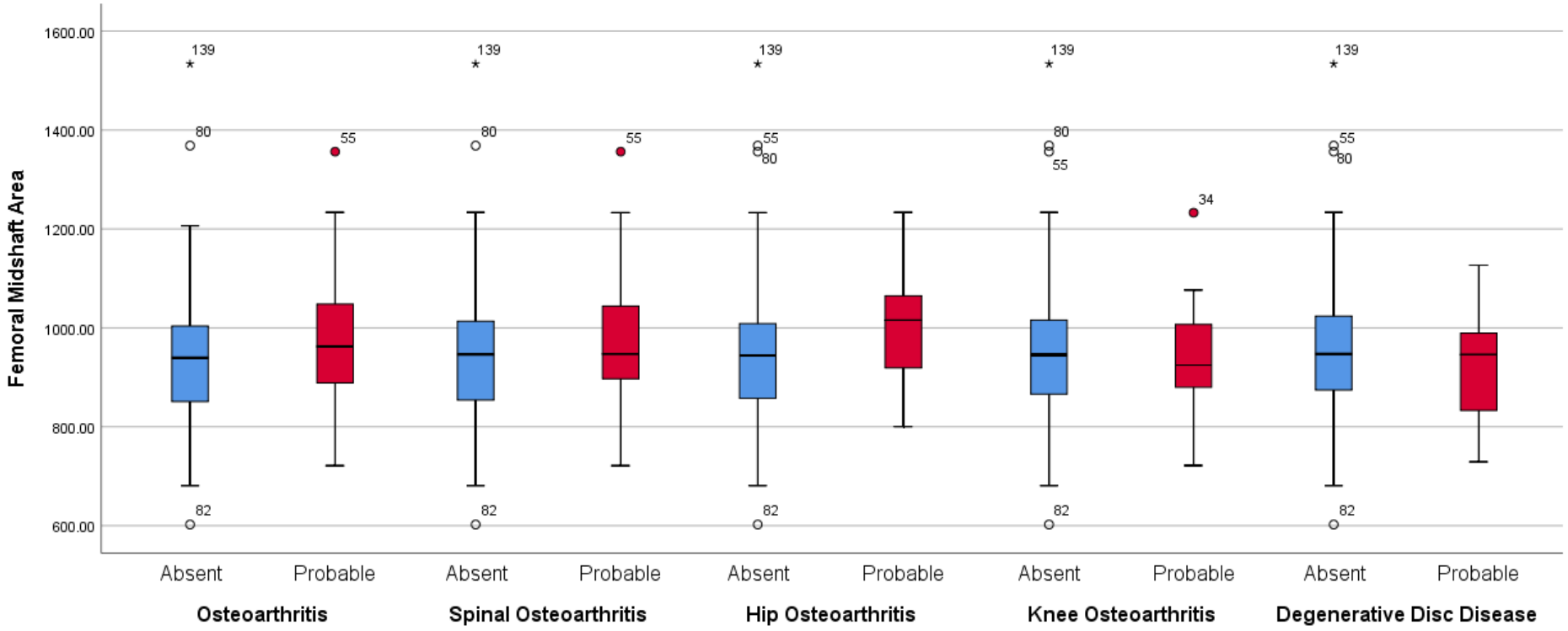

Figure 7.70 Boxplot of femoral midshaft area across the joint conditions. Note the relatively standard mean and uniform skew.

The interquartile ranges were used for testing concerning categorical data primarily for using chi-squared (see Table 7.72). This type of categorization allowed for the testing within the regression analyses to show the spread of data.

Table 7.72 Interquartile ranges for the cross-sectional variables.

	Q1 <25%	Q2 25-50%	Q3 50-75%	Q4 >75%
FSS	0.68-0.82	0.82-0.86	0.87-0.91	0.92-1.08
FSR	56.76-64.49	64.60-67.58	67.68-71.75	71.85-80.40
FSJ	103.88-216.60	217.59-240.00	240.26-263.12	264.84-345.94
FSA	800.93-1059.15	1060.35-1142.33	1144.76-1227.61	1229.23-1574.37
FMS	0.72-0.92	0.93-1.02	1.02-1.09	1.09-1.35
FMR	52.30-59.15	59.21-60.83	60.86-63.88	64.09-79.11
FMJ	71.54-144.86	145.54-168.48	168.82-186.52	187.08-298.55
FMA	602.24-851.03	855.90-939.39	941.44-1010.01	1011.45-1533.77

The ranges listed represent actual point data of skeletal samples. Q = quartile.

7.4.4 Inter-variable Correlations/Associations

The initial examinations of the data displayed surface patterns and basic demographic profiles but is insufficient for the purposes of determining the significance of the relationships between the variables and the joint conditions. The following sections are separated to contextualize the patterns between the variables themselves and then between the variables and the joint conditions. Correlation and chi-squared tests were used to show the relationships between the categorical data and then ANOVA and T-Tests were used to show the relationships and directionality amongst the continuous data. The data is a mix of different types of data (ordinal, nominal, and scale), and for tests of correlations, the final figures produced by differing tests were similar, and the choice was made to use a standardized test for the mixed data analyses. Testing for correlation and association help to determine if a potential relationship between variables exists and the directionality of the relationship. For instance, the theory that increased body mass should relate to higher numbers of joint conditions. However, correlation/association does not even equal causation and so these values should not be considered singly, but within the greater context of the analyses.

7.4.4.1 Statistical Testing of Variables

Entheseal changes displayed a significant, weak positive correlation and significant association with the site category. When compared with the early data in the previous sections, the rural population then has significantly higher levels of EC than the urban population. FMA also displayed a significant association with site category, however, the correlation was not significant. Biological sex displayed multiple significant, weak positive correlations as well as significant associations. FSR, FSJ, FSA, FMJ and FMA displayed significant correlations and FSR, FSA, FSJ and FMA displayed significant associations. Age at death showed no significant correlations or associations with the dependent variables.

Table 7.73 Correlations of independent risk factor variables and dependent risk factor variables using Kendall's tau-b test.

		BMI	EC	FSS	FSR	FSJ	FSA	FMS	FMR	FMJ	FMA
SITE CATEGORY	**r**	-0.017	0.223	0.025	0.026	-0.035	-0.017	-0.034	0.116	0.134	0.088
	Sig.	0.822	0.002	0.731	0.732	0.647	0.817	0.650	0.119	0.077	0.247
BIOLOGICAL SEX	**r**	-0.084	-0.116	-0.124	0.228	-0.197	-0.281	-0.108	0.051	-0.332	-0.350
	SIG	0.261	0.112	0.094	0.002	0.011	0.000	0.148	0.502	0.000	0.000
AGE AT DEATH	**r**	0.045	0.128	0.101	0.034	0.082	0.138	0.114	0.106	0.148	0.178
	SIG	0.526	0.064	0.150	0.633	0.265	0.053	0.104	0.140	0.042	0.013

Green boxes indicate a significant relationship. No significant relationships higher than weak were found. Bonferroni correction: α altered = .05/10 = .005.

Table 7.74 Associations of independent risk factor variables and dependent risk factor variables using Chi-square test.

		BMI	EC	FSS	FSR	FSJ	FSA	FMS	FMR	FMJ	FMA
SITE CATEGORY	**X^2**	1.367	9.678	0.510	6.180	1.050	2.360	0.874	2.701	8.703	18.189
	p	0.713	0.008	0.917	0.103	0.789	0.501	0.832	0.440	0.034	0.000
BIOLOGICAL SEX	**X^2**	0.755	2.884	3.627	13.086	7.808	16.069	4.210	3.379	20.265	23.791
	p	0.385	0.236	0.305	0.004	0.050	0.001	0.240	0.337	0.000	0.000
AGE AT DEATH	**X^2**	0.237	5.585	4.052	7.678	7.890	11.200	15.025	3.776	0.265	14.840
	p	0.888	0.232	0.670	0.263	0.246	0.082	0.020	0.707	0.124	0.022

Green boxes indicated a significant association. Bonferroni correction: α altered = .05/10 = .005.

Testing of the dependent variables with continuous data was conducted using Independent Samples T-Tests and One-way ANOVA tests (see Table 7.75 and Table 7.76). After correction, only three variables compared against biological sex were found to be significant using the T-Tests (FSA, FMJ and FMA) and those variables indicated that the males

have the larger values. The One-way ANOVA Tests only had two significant tests found (FMJ: E/L and FMA: E/L) and in both cases indicated the values were higher as age increased.

Table 7.75 Independent T-Tests for the dependent variables with continuous data and the independent variables with binary categories.

		LEVENE'S TEST		T-TEST						
		F	SIG.	t	df	SIG. (2-TAILED)	MEAN DIFF.	STD. ERR. DIFF.	95% CONFIDENCE LOWER	95% CONFIDENCE UPPER
SITE CATEGORY	BMI	2.063	.153	.391	148	.697	0.124	0.319	-0.505	0.754
	FSS	.434	.511	-.531	151	.596	-0.006	0.012	-0.030	0.017
	FSR	.560	.456	.011	146	.991	0.007	0.673	-1.323	1.337
	FSJ	.235	.629	.529	138	.598	3.792	7.171	-10.386	17.971
	FSA	.070	.791	.119	146	.905	2.738	23.010	-42.737	48.213
	FMS	.073	.788	.210	150	.834	0.003	0.015	-0.027	0.033
	FMR	.265	.607	-1.531	144	.128	-1.050	0.686	-2.406	0.306
	FMJ	.589	.444	-1.677	140	.096	-9.836	5.866	-21.433	1.762
	FMA	2.109	.149	-1.397	144	.164	-30.960	22.156	-74.754	12.833
BIOLOGICAL SEX	BMI	1.225	.270	1.207	148	.306	0.331	0.323	-0.306	0.969
	FSS	.169	.682	2.206	151	.029	0.026	0.012	0.003	0.049
	FSR	2.131	.147	-2.572	146	.011	-1.714	0.667	-3.032	-0.397
	FSJ	6.228	.014*	2.55	92.375	.012	19.300	7.555	4.297	34.303
	FSA	.251	.617	3.912	146	.000	86.711	22.168	42.901	130.522
	FMS	.002	.965	1.802	150	.074	0.027	0.015	-0.003	0.058
	FMR	2.889	.091	-1.280	144	.203	-0.893	0.698	-2.272	0.486
	FMJ	5.000	.027*	4.074	97.240	.000	24.403	5.989	12.516	36.290
	FMA	.325	.569	4.271	144	.000	21.324	21.324	48.927	133.223

* - indicates assumption of unknown variances.

Table 7.76 One way ANOVA test using Bonferroni's test for the dependent variables with continuous data and age at death categories.

VAR	AGE CATEGORY		MEAN DIFFERENCE (I - J)	STD. ERR.	SIG.	95% CONFIDENCE INTERVAL		VAR	AGE CATEGORY		MEAN DIFFERENCE (I - J)	STD. ERR	SIG.	95% CONFIDENCE INTERVAL	
	I	J				LOWER	UPPER		I	J				LOWER	UPPER
BMI	E	M	-0.267	0.438	1.000	-1.329	0.794	FMS	E	M	0.014	0.021	1.000	-0.037	0.065
		L	-0.29	0.372	1.000	-1.191	0.611			L	-0.011	0.017	1.000	-0.053	0.031
	M	E	0.267	0.438	1.000	-0.794	1.329		M	E	-0.014	0.021	1.000	-0.065	0.037
		L	-0.023	0.406	1.000	-1.005	0.959			L	-0.025	0.02	.591	-0.073	0.022
	L	E	0.29	0.372	1.000	-0.611	1.191		L	E	0.011	0.017	1.000	-0.031	0.053
		M	0.023	0.406	1.000	-0.959	1.005			M	0.025	0.02	.591	-0.022	0.073
FSS	E	M	-0.008	0.016	1.000	-0.047	0.032	FMR	E	M	-0.044	0.95	1.000	-2.346	2.259
		L	-0.019	0.014	.476	-0.052	0.014			L	-1.382	0.788	.245	-3.291	0.528
	M	E	0.008	0.016	1.000	-0.032	0.047		M	E	0.044	0.95	1.000	-2.259	2.346
		L	-0.012	0.015	1.000	-0.048	0.025			L	-1.338	0.885	.399	-3.482	0.806
	L	E	0.019	0.014	.476	-0.014	0.052		L	E	1.382	0.788	.245	-0.528	3.291
		M	0.012	0.015	1.000	-0.025	0.048			M	1.338	0.885	.399	-0.806	3.482
FSR	E	M	-0.179	0.923	1.000	-2.415	2.057	FMJ	E	M	-9.115	8.043	.777	-28.606	10.376
		L	-0.911	0.779	.733	-2.798	0.977			L	-19.802	6.656	.010	-35.931	-3.673
	M	E	0.179	0.923	1.000	-2.057	2.415		M	E	9.115	8.043	.777	-10.376	28.606
		L	-0.732	0.857	1.000	-2.809	1.345			L	-10.687	7.451	.461	-28.742	7.369
	L	E	0.911	0.779	.733	-0.977	2.798		L	E	19.802	6.656	.010	3.673	35.931
		M	0.732	0.857	1.000	-1.345	2.809			M	10.687	7.451	.461	-7.369	28.742

Table 7.76 Continued

VAR	AGE CATEGORY		MEAN DIFFERENCE (I - J)	STD. ERR.	SIG.	95% CONFIDENCE INTERVAL		VAR	AGE CATEGORY		MEAN DIFFERENCE (I - J)	STD. ERR	SIG.	95% CONFIDENCE INTERVAL	
	I	J				LOWER	UPPER		I	J				LOWER	UPPER
FSJ	E	M	-3.349	9.831	1.000	-27.178	20.479	FMA	E	M	-21.214	30.238	1.000	-94.466	52.037
		L	-16.542	8.270	.142	-36.586	3.503			L	-68.809	25.078	.021	-129.559	-8.060
	M	E	3.349	9.831	1.000	-20.479	27.178		M	E	21.214	30.238	1.000	-52.037	94.466
		L	-13.193	9.025	.438	-35.067	8.682			L	-47.595	28.160	.280	-115.812	20.622
	L	E	16.542	8.270	.142	-3.503	36.586		L	E	68.809	25.078	.021	8.060	129.559
		M	13.193	9.025	.438	-8.682	35.067			M	47.595	28.160	.280	-20.622	115.812
FSA	E	M	-30.545	31.089	.982	-105.846	44.756	VAR- VARIABLE; E- EARLY ADULT; M- MIDDLE ADULT; L- LATE ADULT							
		L	-64.473	26.243	.046	-128.035	-0.912								
	M	E	30.545	31.089	.982	-44.756	105.846								
		L	-33.928	28.873	.726	-103.861	36.005								
	L	E	64.473	26.243	.046	0.912	128.035								
		M	33.928	28.873	.726	-36.005	103.861								

Next, the scale data for the cross-sectional indices were correlated with each other using the Pearson correlation coefficient, to explore any interrelationships. The Bonferroni corrected p-value for the following tests was α altered = .007. The strongest correlation was FMJ/FMA, with a positive relationship r >.800 (see Table 7.77). Strong correlations existed between FSR/FSJ, FSR/FSA, FMR/FMJ and FMR/FMA, which had positive relationships r>.600. Moderate correlations existed between FSR/FMR, FSJ/FMJ, FSJ/FMA, FSA/FMR, FSA/FMJ and FSA/FMA, which displayed a positive relationship r>.400. Weak correlations existed between FSR/FMJ and FSR/FMA, which displayed a positive relationship r>.200. A very weak correlation existed between FSS/FMS, which displayed a positive relationship r>.200. Femoral subtrochanteric shape or midshaft shape did not exhibit any significant correlations with any of other cross-sectional variables. This illustrates the interrelations within the cross-sectional indices, with obvious exception to shape. Figure 7.71 and Figure 7.72 display examples of the relationships between the individual variables of the cross-sectional indices, with individuals marked by biological sex. The remaining scatterplots between all of the variables can be found within the appendix Section Cross-Sectional Indices.

Table 7.77 Correlations between the cross-sectional variables using Pearson correlation coefficient.

		FSS	FSR	FSJ	FSA	FMS	FMR	FMJ	FMA
FSS	**r**		-0.079	-0.087	-0.014	0.145	-0.172	-0.073	-0.154
	SIG		0.250	0.216	0.837	0.035	0.014	0.302	0.027
FSR	**r**			0.618	0.600	-0.042	0.421	0.272	0.292
	SIG			0.000	0.000	0.546	0.000	0.000	0.000
FSJ	**r**				0.799	-0.081	0.382	0.468	0.433
	SIG				0.000	0.251	0.000	0.000	0.000
FSA	**r**					-0.011	0.449	0.528	0.536
	SIG					0.875	0.000	0.000	0.000
FMS	**r**						0.034	0.054	0.082
	SIG						0.628	0.444	0.234
FMR	**r**							0.632	0.663
	SIG							0.000	0.000
FMJ	**r**								0.814
	SIG								0.000

Color coded relationships: Blue - Weak. Green - Moderate. Orange - Strong. Red - Very strong. Black area is shade to prevent overlapping figures. Bonferroni correction: α altered = .05/7 = .007.

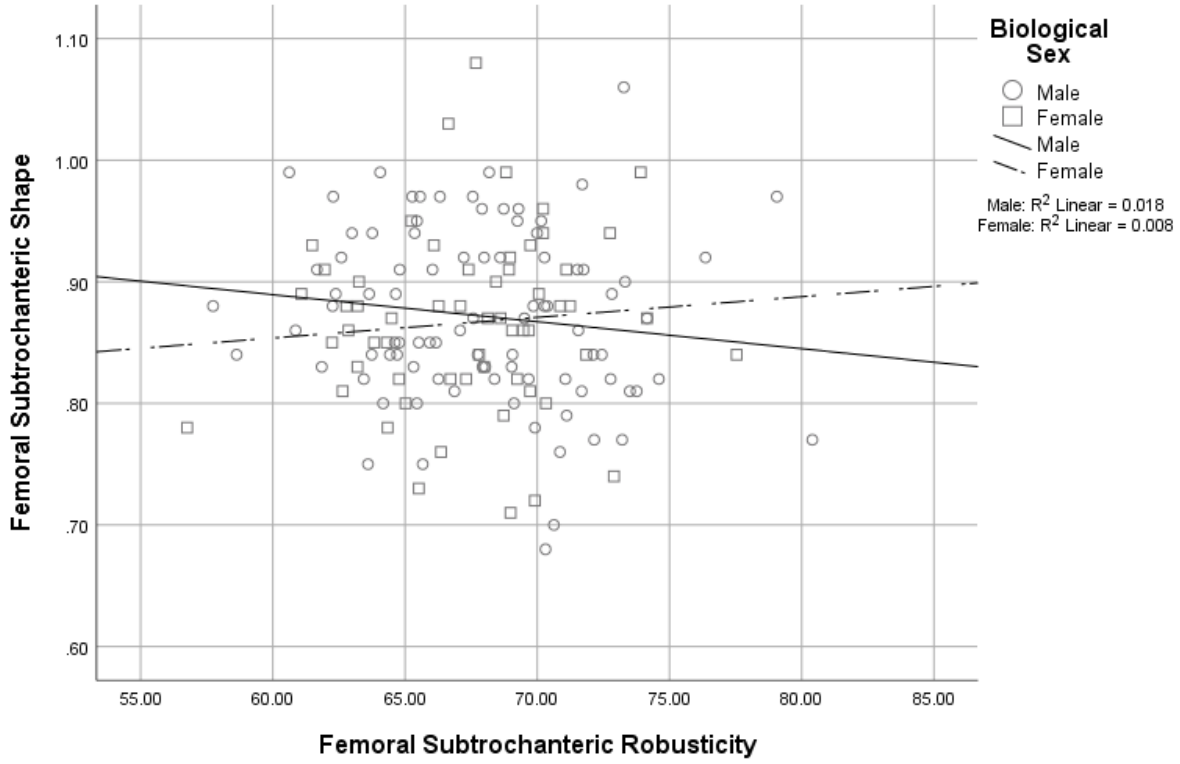

Figure 7.71 Distribution of the sample population by femoral subtrochanteric shape and femoral subtrochanteric robusticity. The samples have further been identified by biological sex with R^2 values for each.

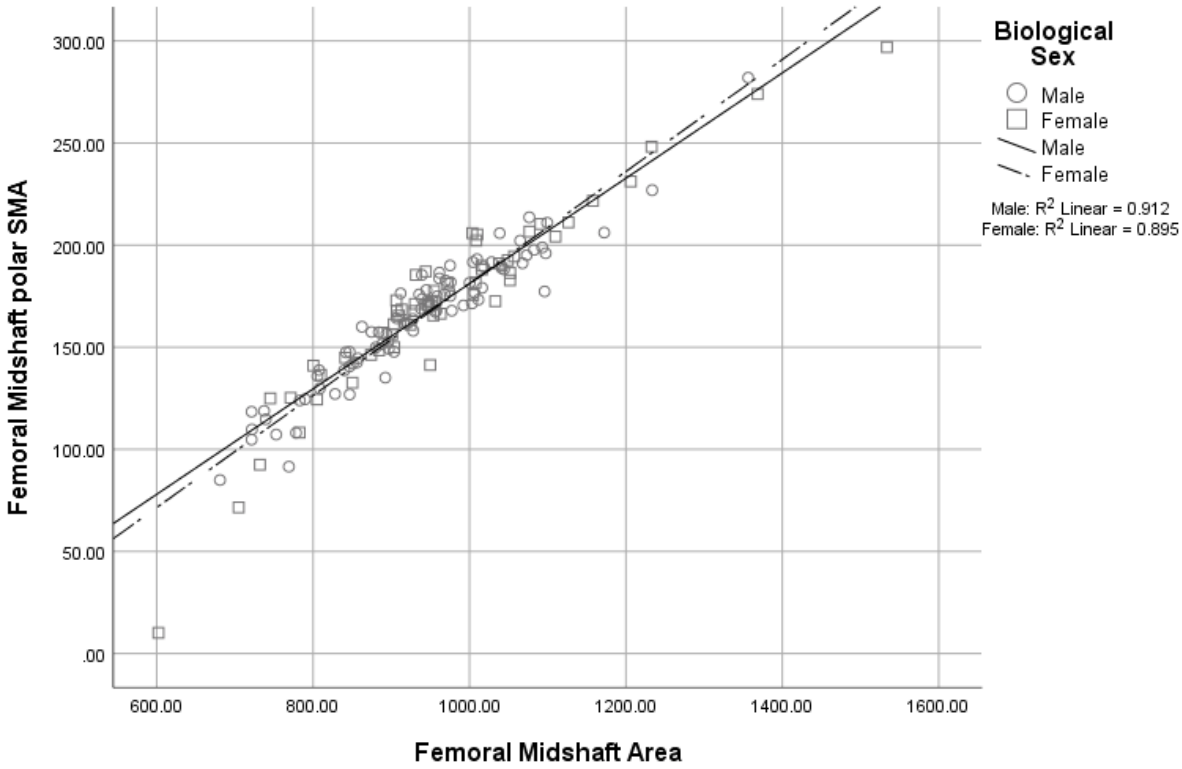

Figure 7.72 Distribution of the sample population by femoral midshaft polar SMA and femoral midshaft area. The samples have further been identified by biological sex with R^2 values.

7.4.4.2 Statistical Testing of the Joint conditions

The joint conditions can now be compared with the risk factors to identify any significant bivariate correlations or associations. The Bonferroni corrected p-value for the following tests was α altered = .004. Only two significant bivariate correlations were found between the joint conditions and risk factor variables r >.300 (Age/OA: r = .313; FSS/SOA: r = .342) (see Table 7.78). Age at death and entheseal changes were the only risk factor variables to display a significant relationship with joint conditions, which were positive relationships. General and spinal osteoarthritis were the only joint conditions to display a significant relationship with the risk factor variables, which were positive relationships. Age at death displayed the highest number of significant associations (OA/ SOA) with the joint conditions (see Table 7.79). Femoral subtrochanteric shape was the only other variable to display significant associations with any of the joint conditions.

Table 7.78 Correlations between the joint conditions and risk factor variables using phi correlation coefficient.

		OA	SOA	HOA	KOA	DDD
SITE CATEGORY	r	.093	.118	.011	-.016	-.147
	SIG	.219	.126	.886	.838	.056
BIOLOGICAL SEX	r	-.066	-.030	-.142	.078	-.067
	SIG	.385	.693	.060	.304	.388
AGE AT DEATH CATEGORY	r	.313	.216	.212	.135	.129
	SIG	.000	.020	.020	.208	.248
BODY MASS INDEX IQR	r	.129	.171	.165	.089	.107
	SIG	.479	.236	.254	.036	.116
ENTHESEAL CHANGES	r	.243	.216	.187	.896	.325
	SIG	.006	.020	.047	.896	.325
FEM SUBTROCH SHAPE IQR	r	.208	.342	.123	.160	.076
	SIG	.084	.001	.509	.280	.841
FEM SUBTROCH ROBUSTICITY IQR	r	.236	.210	.119	.245	.095
	SIG	.042	.099	.552	.033	.736
FEM SUBTROCH POLAR SMA IQR	r	.225	.244	.092	.184	.147
	SIG	.069	.045	.756	.196	.410
FEM SUBTROCH AREA IQR	r	.200	.153	.130	.085	.164
	SIG	.117	.343	.478	.788	.283
FEM MIDSHAFT SHAPE IQR	r	.085	.103	.166	.211	.096
	SIG	.776	.668	.240	.083	.719
FEM MIDSHAFT ROBUSTICITY IQR	r	.192	.149	.099	.043	.179
	SIG	.147	.373	.699	.966	.215
FEM MIDSHAFT POLAR SMA IQR	r	.149	.217	.122	.076	.030
	SIG	.368	.092	.552	.845	.989
FEM MIDSHAFTAREA IQR	r	.142	.134	.144	.032	.087
	SIG	.399	.467	.385	.985	.790

Green shows significant correlations. Bonferroni correction: α adjusted = .05/13 = .004.

Table 7.79 Associations between the joint conditions and risk factor variables using Chi-square.

		OA	SOA	HOA	KOA	DDD
SITE CATEGORY	X^2	1.511	2.338	0.021	0.042	3.640
	P VALUE	0.219	0.126	0.886	0.838	0.056
BIOLOGICAL SEX	X^2	0.755	-0.156	3.524	0.078	0.744
	P VALUE	0.385	0.693	0.060	0.304	0.388
AGE AT DEATH CATEGORY	X^2	17.17	13.09	7.829	3.142	2.787
	P VALUE	0.000	0.001	0.020	0.208	0.248
BODY MASS INDEX IQR	X^2	10.320	7.865	6.102	0.220	2.245
	P VALUE	0.006	0.020	0.047	0.896	0.325
ENTHESEAL CHANGES	X^2	2.478	4.246	4.073	1.182	1.642
	P VALUE	0.479	0.236	0.254	0.757	0.65
FEM SUBTROCH SHAPE IQR	X^2	6.645	17.260	2.316	3.830	0.835
	P VALUE	0.084	0.001	0.509	0.280	0.841
FEM SUBTROCH ROBUSTICITY IQR	X^2	8.216	6.284	2.101	8.706	1.271
	P VALUE	0.042	0.099	0.552	0.033	0.736
FEM SUBTROCH POLAR SMA IQR	X^2	7.101	8.045	1.189	4.690	2.885
	P VALUE	0.069	0.045	0.756	0.196	0.410
FEM SUBTROCH AREA IQR	X^2	5.897	3.334	2.488	1.055	3.806
	P VALUE	0.117	0.343	0.478	0.788	0.283
FEM MIDSHAFT SHAPE IQR	X^2	1.104	1.561	4.207	6.665	1.341
	P VALUE	0.776	0.668	0.240	0.083	0.719
FEM MIDSHAFT ROBUSTICITY IQR	X^2	5.365	3.122	1.429	0.270	4.473
	P VALUE	0.147	0.373	0.699	0.966	0.215
FEM MIDSHAFT POLAR SMA IQR	X^2	3.155	6.444	2.100	0.817	0.123
	P VALUE	0.368	0.092	0.552	0.845	0.989
FEM MIDSHAFTAREA IQR	X^2	2.952	2.547	3.042	0.148	1.048
	P VALUE	0.399	0.467	0.385	0.985	0.790

Green shows significant associations. Bonferroni correction: α adjusted = .05/13 = .004.

Independent Sample T-Tests of the joint conditions against the variables with continuous data sets were completed to test the differences in the data between absent and probable diagnoses (see Table 7.80 - Table 7.84). KOA and DDD displayed no significant tests with the variables. OA, SOA and HOA displayed significance with age at death with increased age appearing to affect absent diagnoses over probable diagnoses. SOA also displayed significance with FSS, whereas increased FSS values appear to affect probable diagnoses over absent diagnoses.

Table 7.80 Independent Samples T-Tests for osteoarthritis against the variables with continuous data sets.

		LEVENE'S TEST		T-TEST						
		F	SIG.	t	df	SIG. (2-TAILED)	MEAN DIFFERENCE	STD. ERR. DIFFERENCE	95% CONFIDENCE LOWER	95% CONFIDENCE UPPER
	AGE	1.127	.290	-4.304	173	.000	-14.831	3.446	-21.632	-8.030
	BMI	0.369	.544	1.295	148	.197	0.426	0.329	-0.224	1.076
	FSS	10.732	.001*	-2.101	87.076	.039	-0.028	0.013	-0.054	-0.001
	FSR	0.193	.661	-1.648	146	.101	-1.150	0.698	-2.529	0.229
	FSJ	3.953	.049*	-1.565	135.088	.120	-10.493	6.704	-23.752	2.765
	FSA	2.602	.109	-1.841	146	.068	-43.819	23.806	-90.868	3.230
	FMS	0.308	.580	-0.094	150	.925	-0.001	0.016	-0.033	0.030
	FMR	0.709	.401	-1.245	144	.215	-0.893	0.717	-2.310	0.525
	FMJ	0.107	.744	-1.121	140	.264	-6.854	6.116	-18.946	5.238
OA	FMA	0.215	.644	-1.341	144	.182	-30.990	23.112	-76.671	14.692

* - indicates assumption of unknown variances. Green = significant values. Bonferroni correction: α adjusted = .05/10 = .005.

Table 7.81 Independent Samples T-Tests for spinal osteoarthritis against the variables with continuous data sets.

		LEVENE'S TEST		T-TEST						
		F	SIG.	t	df	SIG. (2-TAILED)	MEAN DIFFERENCE	STD. ERR. DIFFERENCE	95% CONFIDENCE LOWER	95% CONFIDENCE UPPER
	AGE	4.100	.044*	-3.767	73.956	.000	-14.194	3.768	-21.702	-6.686
	BMI	0.609	.436	1.047	143	.297	0.380	0.363	-0.337	1.096
	FSS	16.370	.000*	-3.087	50.225	.003	-0.049	0.016	-0.081	-0.017
	FSR	0.275	.601	-1.332	141	.185	-1.032	0.775	-2.564	0.500
	FSJ	1.371	.244	-1.257	133	.211	-10.184	8.102	-26.210	5.842
	FSA	2.256	.135	-1.333	141	.185	-35.250	26.441	-87.523	17.023
	FMS	0.026	.872	-0.188	145	.852	-0.003	0.018	-0.038	0.032
	FMR	0.021	.885	-0.770	139	.443	-0.622	0.808	-2.220	0.976
	FMJ	0.476	.492	-0.769	135	.443	-5.272	6.855	-18.829	8.285
SOA	FMA	0.014	0.905	-0.759	139	.449	-19.783	26.077	-71.343	31.776

* - indicates assumption of unknown variances. Green = significant values. Bonferroni correction: α adjusted = .05/10 = .005.

Table 7.82 Independent Samples T-Tests for hip osteoarthritis against the variables with continuous data sets.

		LEVENE'S TEST		T-TEST						
		F	SIG.	t	df	SIG. (2-TAILED)	MEAN DIFFERENCE	STD. ERR. DIFFERENCE	95% CONFIDENCE LOWER	95% CONFIDENCE UPPER
	AGE	6.227	.014*	-3.097	29.380	.004	-14.283	4.612	-23.710	-4.857
	BMI	0.001	.978	1.882	148	.062	0.891	0.474	-0.045	1.827
	FSS	0.847	.359	1.229	151	.221	0.022	0.018	-0.014	0.058
	FSR	0.580	.448	-0.453	146	.652	-0.477	1.054	-2.559	1.606
	FSJ	1.255	.265	-0.874	138	.384	-9.569	10.953	-31.226	12.089
	FSA	0.361	.549	-1.502	146	.135	-53.727	35.778	-124.437	16.983
	FMS	0.026	.872	-0.311	150	.756	-0.007	0.023	-0.053	0.039
	FMR	0.000	.997	-0.617	144	.538	-0.663	1.075	-2.788	1.461
	FMJ	1.649	.201	-1.032	140	.304	-9.375	9.083	-27.332	8.582
HOA	FMA	0.149	.700	-1.406	144	.162	-48.489	34.483	-116.648	19.670

* - indicates assumption of unknown variances. Green = significant values. Bonferroni correction: α adjusted = .05/10 = .005.

Table 7.83 Independent Samples T-Tests for knee osteoarthritis against the variables with continuous data sets.

		LEVENE'S TEST		T-TEST						
		F	SIG.	t	df	SIG. (2-TAILED)	MEAN DIFFERENCE	STD. ERR. DIFFERENCE	95% CONFIDENCE LOWER	95% CONFIDENCE UPPER
	AGE	1.112	.293	-1.898	170	.059	-10.888	5.738	-22.214	0.439
	BMI	0.149	.700	0.519	147	.604	0.268	0.517	-0.753	1.290
	FSS	2.119	.148	-0.584	148	.560	-0.012	0.020	-0.051	0.028
	FSR	0.113	.737	-0.370	143	.712	-0.416	1.125	-2.640	1.808
	FSJ	1.158	.284	0.270	137	.787	3.146	11.639	-19.869	26.161
	FSA	0.584	.446	0.186	143	.853	7.150	38.393	-68.741	83.041
	FMS	0.476	.491	-1.790	147	.076	-0.045	0.025	-0.095	0.005
	FMR	1.267	.262	-0.426	141	.671	-0.482	1.133	-2.723	1.758
	FMJ	0.048	.827	0.110	139	.913	1.060	9.667	-18.054	20.173
KOA	FMA	0.004	.950	0.137	141	.891	5.053	36.917	-67.929	78.035

Bonferroni correction: α adjusted = .05/10 = .005.

Table 7.84 Independent Samples T-Tests for degenerative disc disease against the variables with continuous data sets.

		LEVENE'S TEST		T-TEST						
		F	SIG.	t	df	SIG. (2-TAILED)	MEAN DIFFERENCE	STD. ERR. DIFFERENCE	95% CONFIDENCE LOWER	95% CONFIDENCE UPPER
	AGE	0.008	.928	-1.485	166	.139	-6.8919	4.6398	-16.0526	2.2688
	BMI	0.245	.622	-1.373	141	.172	-0.58389	0.42516	-1.42440	0.25661
	FSS	0.073	.787	0.112	144	.911	0.00177	0.01574	-0.02935	0.03289
	FSR	0.678	.412	1.338	139	.183	1.19904	0.89621	-0.57293	2.97101
	FSJ	0.894	.346	0.060	131	.952	0.58194	9.74070	-18.68749	19.85137
	FSA	0.553	.458	1.292	139	.198	39.67468	30.70603	-21.03659	100.38595
	FMS	0.013	.908	-0.385	143	.701	-0.00786	0.02043	-0.04825	0.03252
	FMR	0.467	.496	1.008	137	.315	0.95868	0.95114	-0.92212	2.83949
	FMJ	0.333	.565	0.141	133	.888	1.16370	8.23026	-15.11543	17.44283
DDD	FMA	0.577	0.449	0.951	137	.344	29.21638	30.73672	-31.56337	89.99614

Bonferroni correction: α adjusted = .05/10 = .005.

7.4.4.3 Summary of Bivariate Testing

The independent variables (site category, biological sex, and age at death) were compared against the dependent variables (body mass index, entheseal changes, and cross-sectional indices using a 2-tailed Kendall's tau-b test. For the testing, all of the variables were converted into their categorical form. Using Bonferroni correction (α altered = .05/10), resulted in a significance threshold for these tests of α altered = .005. A significant, positive correlation was obtained for 6 of the 30 comparisons (2 positive and 4 negative), but all were weak ($r \leq 0.350$). Significant associations were obtained in 5 of the 30 variables, with half of these associations relating to biological sex.

In summation, the bivariate assessments of the risk factors assisted with determining the relationships, if any, amongst the risk factors, and then between the risk factors and the joint conditions displayed few relationships. Biological sex was the independent risk factor that displayed the most significant relationships between the dependent variables, however, the relationships were weak, and except for one, were all negative correlations. Age at death was the only risk factor that displayed both positive significant correlations and associations with the joint conditions and entheseal change presented significant correlations with the joint conditions. However, these relationships do not denote whether the relationships indicate a direct connection or causality. Further testing using multi-variate analyses will help to indicate how these relationships change when additional factors are present.

7.5 Risk Ratios

Risk ratios display possible rates at which an individual is likely to exhibit a condition based on a certain risk factor, with a risk ratio of 1 indicating no change in risk between the two categories. A risk ratio that is <1 or >1 but close to 1 indicates minimal risk. A risk ratio of >1 indicates that the risk increases as the risk factor variable increases (i.e., chance of presenting joint condition increases with increasing age or robusticity) and a risk ratio of <1 indicates that the risk is increased as the variable decreases. Analyzing the risk ratios displays how the differing variables are associated with prevalence of the joint conditions and is directly complementary to the multivariate analysis above which has indicated the power of the risk factors for predicting joint condition.

Risk ratios for the joint condition and risk factors studies in this study are presented in Table 7.84. For the comprehensive tables of each variable with the risk ratios for each sub-grouping, see the appendix Section Risk Ratios. Age at death (OA, SOA, HOA and KOA) and EC (OA, SOA, HOA, DDD) made the most contributions to risk of joint conditions. For EC, as the variable increases the risk increased for SOA and HOA by more than 3 times. Age at death displayed an increased risk of SOA, by 3.48 times, as age increased. Body mass displayed low risk, however, the risk appeared to be greater when mass decreases, with the exceptions of DDD which increases and KOA that has no risk.

Hip OA was the only joint condition to display a noticeable change in risk for biological sex, which showed an increase to risk for males. SOA displayed no change in risk for any of the cross-sectional indices. KOA displayed the most risk with the cross-sectional variables, with greater risk for the variables at the midshaft, as the variables decreased. The greatest risk found was with FMA and KOA, whereas a greater risk was present (4.76 times) as the area decreases. DDD is distinct from the OAs in that less EC resulted in higher risk and lower FSS values results in increased risk. FSS was found to affect the risk of no other joint condition in this study.

Table 7.85 The risk ratios for the joint conditions by risk factor variables.

		JOINT CONDITIONS				
		OA	SOA	HOA	KOA	DDD
RISK FACTOR VARIABLE	BIOLOGICAL SEX	1.21	1.12	2.38*	0.62	1.36
	AGE AT DEATH	2.58*	3.48**	2.84*	2.62*	1.81
	ENTHESEAL CHANGE	2.73*	3.13**	3.65**	1.42	0.45*
	BODY MASS INDEX	0.68	0.48*	0.40*	1.00	1.49
	FS SHAPE	1.08	0.91	1.00	1.62	0.35*
	FS ROBUSTICITY	1.21	1.25	1.35	0.46*	0.94
	FS POLAR SMA	0.81	0.69	0.69	1.09	1.29
	FS AREA	0.69	0.69	0.69	0.36*	1.29
	FM SHAPE	0.86	1.14	0.38*	0.24**	1.87
	FM ROBUSTICITY	0.96	1.29	1.03	0.43*	0.82
	FM POLAR SMA	0.49*	1.04	0.43*	0.41*	1.27
	FM AREA	0.49*	0.71	0.63	0.21***	1.44

Biological sex is a binary variable and >1 indicates a greater risk for males and <1 for females. Light blue – Risk increases as the variable decreases. Light orange – ratios with no relationship. Unshaded – risk increases as the variables increases. FS – femoral subtrochanteric. FM – femoral midshaft. An * denotes a difference by scale of 2,3,4. * - 2x. ** - 3x. ***- 4x.

7.6 Multi-variate Analysis

The previous sections dealt with prevalence relating to proportional data, which offers valid descriptive information, enabling the identification of surface trends and bivariate statistical relationship. However, the clinical evidence presented earlier in this thesis suggests a potentially more complex interaction between multiple risk factors in the formation of joint conditions. The analysis thus far will not help to identify more complex relationships or trends in a manner that is statistically reliable. Bivariate assessments display direct relationships between two variables, while multivariate statistical testing enables the interactions of three or more variables to be assessed via statistical modelling. The latter can also help to establish whether a relationship is affected as one more new variable is added to the model. In this way, a variable that was not statistically significant on its own, may display evidence of significance when combined with other variables. The main method of multivariate analysis used for this research was binary logistic regression.

Binary logistic regression facilitates the prediction of a binary outcome from multiple input variables and was used here to explore the contribution of all the variables discussed above to the prediction of whether joint condition was present or absent (Campbell *et al.,* 2007; Field, 2013; Wagner, 2015). This form of regression analysis is also flexible regarding the types of data included in the model; thus, the regression equations were able to include all the variables, in the following categorical formats:

Nominal: joint conditions, biological sex

Ordinal: age at death, body mass index interquartile values, entheseal changes, and cross-sectional index interquartile values

For the data that is categorical, the entire variable may not be found to be significant, but a single sub-category may be significant (e.g., age at death: early, middle or adult) and can show up in the final stage (i.e., Age at Death (1) indicating middle adults). The IQR values were used to help determine any significant changes that may occur between each sub-grouping that might indicate any patterns for further analyses. Site category was dropped from the regression variables as it features as a descriptive or sorting variable, and while independent, the factors of mass and activity that may occur at each site were decided to be the more important variables for determining the likelihood of joint conditions.

The equations used a backwards, stepwise method to decrease the number of variables until only those that made a significant contribution ($\alpha < .05$) or those that approached significance ($\alpha < .07$) remained within the model. Each additional step means that a variable was removed from the equation and its predictive power once more tested, until the conditions mentioned above are met. With a backwards step regression model, the final variables should all display a significant contribution to the model.

With each successive step, the significance of the tests improved with the p values of all joint conditions steadily approaching <.001 (see Table 7.86). Osteoarthritis and spinal osteoarthritis tests had a final significance of <.001, hip osteoarthritis had a significance of .009, and knee osteoarthritis had a significance of .002. Degenerative disc disease is the only remaining joint condition that approaches significance ($p = .053$) in the final but is not significant ($\alpha<.05$). This predictive model shows that for each joint condition, the predictive factors are better than the null model, even if the variables are not significant. The level of explanation at the final step is low for all the joint conditions with DDD having the lowest at 15.0% explained and KOA and HOA having the highest (40.3%/ 38.1).

The Hosmer and Lemeshow Goodness-of-Fit assesses whether the observed rates match the predicted rates through the changes of the chi-square values and significance, which in turn demonstrates the fitness of the data in each step. In other statistical tests, $p < 0.05$ denotes significance, but with this test $p > 0.05$ indicates that the data fits the model. Each of the joint conditions was found to be significant ($p > .05$) demonstrating that the variables in each step fit the model, even if the variables are not significant in the step.

Table 7.86 The values for each successive test of the binary logit regression for each joint condition.

			JOINT CONDITIONS				
			OA	SOA	HOA	KOA	DDD
OMNIBUS	STEP	CHI²	-5.028	-4.364	-4.359	-2.481	-4.005
		DF	3	2	3	2	2
		SIG	.170	.113	.225	.289	.135
	MODEL	CHI²	26.515	28.654	30.791	30.647	12.429
		DF	7	8	15	12	6
		SIG	.000	.000	.009	.002	.053
MODEL SUMMARY		-2 LL	155.379	127.961	71.957	63.772	108.911
		C&S R^2	0.176	0.195	0.201	0.202	0.091
		N R^2	0.239	0.251	0.381	0.403	0.150
H&L GOODNESS OF FIT		CHI²	9.697	8.737	9.238	3.558	2.622
		DF	7	8	8	7	8
		SIG	.206	.365	.323	.829	.956

The complete tables with each step can be found in the appendix Section Multivariate Analyses.

The classification tables offer the predictive percentages for each joint disease. The cut value for each joint condition was .500, meaning that if case fell above that line, it was classified as probable and if it fell below, it was absent. The overall percentage classification displays the percentage of cases correctly classed between the observed and predicted characteristics. The probable classification displays the sensitivity for the percentage accurately classing cases compared between the observed and predicted characteristics. Absent classification displays the specificity for the percentage of having a true negative between the observed and predicted characteristics.

The final step variable tables display the changes of significance between the individual variables, as they are sorted by their category groupings. As the variables are in their ordinal format, as opposed to the category the test has included values for each step. A variable might not have significance, but one of the categories within may be significant. The variables listed within the tables are not duplicates but represent each category within that variable. For instance, age at death has three rows of values, with the first row representing early adults, the second is middle adults and the final is late adults.

The final step for the regression testing concerning OA had an accuracy of 50.0% for probable and 73.7% overall and displayed three variables as significant: age at death, entheseal change and femoral subtrochanteric robusticity (see Table 7.87 and Table 7.88). Age at death was significant overall ($p = .036$) and demonstrated that increased age was a factor (Age at Death [3]: $p = .036$; Exp[B] = 2.676). EC was significant overall ($p = .020$) and demonstrated

that increased EC of OA (EC [2]: p = .008; Exp[B] = 7.573). FSR was significant overall (p = .031) and increased FSR values were a factor in the development of OA (FSS [3]: p = .038; Exp[B] = 3.053).

Table 7.87 The classification table showing the predictive power of the final stage regression test for OA.

CLASSIFICATION	ABSENT	88.2%
	PROBABLE	50.0%
	OVERALL	73.7%

Table 7.88 The final variables of the binomial logistic regression for general osteoarthritis with B and expected B values, as well as Wald scores and significance.

		VARIABLES IN THE EQUATION							
		B	S.E.	WALD	DF	SIG	EXP(B)	95% CI FOR EXP(B) LOWER	UPPER
STEP 11	AGE AT DEATH			6.648	2	0.036			
	AGE AT DEATH (1)	-0.075	0.580	0.017	1	0.898	0.298	0.298	2.891
	AGE AT DEATH (2)	0.984	0.470	4.387	1	0.036	2.676	1.065	6.721
	ENTHESEAL CHANGE			7.777	2	0.020			
	ENTHESEAL CHANGE (1)	0.770	0.428	3.228	1	0.072	2.159	0.932	4.998
	ENTHESEAL CHANGE (2)	2.025	0.764	7.016	1	0.008	7.573	1.693	33.871
	FS ROBUSTICITY			8.857	3	0.031			
	FS ROBUSTICITY (1)	0.775	0.561	1.906	1	0.167	2.170	0.722	6.520
	FS ROBUSTICITY (2)	-0.427	0.603	0.502	1	0.479	0.652	2.000	2.126
	FS ROBUSTICITY (3)	1.116	0.539	4.297	1	0.038	3.053	1.063	8.773
	CONSTANT	-2.007	0.611	10.778	1	0.001	0.134		

Green = Overall significance of the variable. Yellow = Significance between levels within a variable's sub-categories.

The final step for the regression testing concerning SOA had a predictive value of 35.1% for probable diagnoses and 75.8% overall and showed that three variables were significant: entheseal change, femoral subtrochanteric shape and femoral subtrochanteric polar SMA (see Table 7.89 and Table 7.90). EC was significant overall (p = .039) and increased EC was found to be a factor in the development of SOA (EC [2]: p = .012; Exp[B] = 7.581). FSS was significant overall (p = .008), however as FSS increased the significance increased but remained p > .05. Therefore, definitive significant concerning directionality was not found. FSJ was significant overall (p = .026) and while the lower FSJ values were found to be a factor for the development of SOA it was not significant in a decreasing order. This suggests while the lower values of FSJ are significant IQR 2 was more significant than IQR 1 (FSJ [1]: p = 011; Exp[B] = 5.789).

Table 7.89 The classification table showing the predictive power of the final stage regression test for SOA.

CLASSIFICATION	ABSENT	91.6%
	PROBABLE	35.1%
	OVERALL	75.8%

Table 7.90 The final variables of the binomial logistic regression for spinal osteoarthritis with B and expected B values, as well as Wald scores and significance.

		VARIABLES IN THE EQUATION							
		B	S.E.	WALD	DF	SIG	EXP(B)	95% CI FOR EXP(B) LOWER	UPPER
STEP 11	ENTHESEAL CHANGE			6.512	2	0.039			
	ENTHESEAL CHANGE (1)	0.771	0.523	2.173	1	0.140	2.163	0.775	6.034
	ENTHESEAL CHANGE (2)	2.026	0.805	6.337	1	0.012	7.581	1.566	36.702
	FS SHAPE			11.965	3	0.008			
	FS SHAPE (1)	-0.533	0.668	0.637	1	0.425	0.587	0.158	2.173
	FS SHAPE (2)	-1.015	0.677	2.245	1	0.134	0.363	0.096	1.367
	FS SHAPE (3)	1.033	0.602	2.945	1	0.086	2.810	0.863	9.147
	FS POLAR SMA			9.241	3	0.026			
	FS POLAR SMA (1)	1.756	0.687	6.533	1	0.011	5.789	1.506	22.255
	FS POLAR SMA (2)	0.150	0.732	0.042	1	0.837	1.162	0.277	4.881
	FS POLAR SMA (3)	1.184	0.683	3.011	1	0.083	3.269	0.858	12.458
	CONSTANT	-2.384	0.762	9.801	1	0.002	0.092		

Green = Overall significance of the variable. Yellow = Significance between levels within a variable's sub-categories.

The final step of the regression testing concerning HOA had a predictive value of 23.5% for probable diagnoses and 89.8% overall and displayed two variables as significant: age at death, biological sex (see Table 7.91 and Table 7.92). Age at death was significant overall (p = .013) and increased age was found to be a factor in the development of HOA (Age at Death [2]: p = .010; Exp[B]: 15.803). Biological sex was found to be a significant factor (p = .010) and females had the higher risk (Exp[B] = .080). Four additional variables remained in the equation, and while not significant overall, each displayed significance when compared to a differing category level. BMI, FSS, FMJ and FMA were not found to be significant overall, but each variable was found to display significance when compared against a different category level of the same variable (e.g., FMJ IQR 1 and IQR 2). Except for FSJ, the variables displayed significance as the values decreased.

Table 7.91 The classification table showing the predictive power of the final stage regression test for HOA.

CLASSIFICATION	ABSENT	99.2%
	PROBABLE	23.5%
	OVERALL	89.8%

Table 7.92 The final variables of the binomial logistic regression for hip osteoarthritis with B and expected B values, as well as Wald scores and significance.

		VARIABLES IN THE EQUATION							
		B	S.E.	WALD	DF	SIG	EXP(B)	95% CI FOR EXP(B) LOWER	UPPER
STEP 8	AGE AT DEATH			8.636	2	0.013			
	AGE AT DEATH (1)	-0.103	1.352	0.006	1	0.939	0.902	0.054	12.765
	AGE AT DEATH (2)	2.760	1.073	6.619	1	0.010	15.803	1.930	129.408
	BIOLOGICAL SEX (1)	-2.525	0.974	6.725	1	0.010	0.080	0.012	0.540
	BODY MASS INDEX			7.014	3	0.071			
	BODY MASS INDEX (1)	-1.882	1.066	3.119	1	0.077	0.152	0.019	1.229
	BODY MASS INDEX (2)	-2.416	1.090	4.907	1	0.027	0.089	0.011	0.757
	BODY MASS INDEX (3)	-3.448	1.403	6.042	1	0.014	0.032	0.002	0.497
	FS SHAPE			5.352	3	0.148			
	FS SHAPE (1)	-1.010	0.977	1.070	1	0.301	0.364	0.054	2.469
	FS SHAPE (2)	-0.733	0.863	0.722	1	0.396	0.481	0.089	2.606
	FS SHAPE (3)	-2.637	1.143	5.319	1	0.021	0.072	0.008	0.673
	FM POLAR SMA			6.028	3	0.110			
	FM POLAR SMA (1)	4.687	1.918	5.974	1	0.015	108.514	2.531	4652.670
	FM POLAR SMA (2)	5.312	2.398	4.904	1	0.027	202.688	1.842	22306.401
	FM POLAR SMA (3)	5.493	2.591	4.498	1	0.034	243.105	1.514	39023.603
	FM AREA			7.756	3	0.051			
	FM AREA (1)	-5.155	2.065	6.234	1	0.013	0.006	0.000	0.330
	FM AREA (2)	-6.869	2.535	7.344	1	0.007	0.001	0.000	0.149
	FM AREA (3)	-6.469	2.798	5.347	1	0.021	0.002	0.000	0.373
	CONSTANT	0.313	1.303	0.058	1	0.810	1.368		

Green = Overall significance of the variable. Yellow = Significance between levels within a variable's sub-categories.

The final step of the regression testing concerning KOA had a predictive value of 20.0% for probable diagnoses and 89.7% overall and displayed no variables with an overall significance (see Table 7.93 and Table 7.94), however, BMI and FMS displayed significance between its categories. BMI reached significance as mass increased (BMI [3]: p = .035; Exp[B] = 19.511). FMS was found in the final step and the overall variable was not significant but was found to be significant at the category level displaying significance as the values increased (FMS [3]: p = .044; Exp[B] = 9.484).

Table 7.93 The classification table showing the predictive power of the final stage regression test for KOA.

CLASSIFICATION	ABSENT	98.3%
	PROBABLE	20.0%
	OVERALL	89.7%

Table 7.94 The final variables of the binomial logistic regression for knee osteoarthritis with B and expected B values, as well as Wald scores and significance.

		VARIABLES IN THE EQUATION							
		B	S.E.	WALD	DF	SIG	EXP(B)	95% CI FOR EXP(B) LOWER	UPPER
STEP 10	BODY MASS INDEX			6.126	3	0.106			
	BODY MASS INDEX (1)	0.663	1.172	0.320	1	0.572	1.940	0.198	19.289
	BODY MASS INDEX (2)	1.327	1.243	1.400	1	0.286	3.770	0.330	43.090
	BODY MASS INDEX (3)	2.971	1.409	4.445	1	0.035	19.511	1.233	308.844
	FS ROBUSTICITY			5.775	3	0.123			
	FS ROBUSTICITY (1)	-0.102	1.053	0.009	1	0.923	0.903	0.115	7.112
	FS ROBUSTICITY (2)	-17.872	6143.036	0.000	1	0.998	0.000	0.000	
	FS ROBUSTICITY (3)	2.899	1.498	3.746	1	0.053	18.151	0.964	341.814
	FS POLAR SMA			7.601	3	0.055			
	FS POLAR SMA (1)	1.952	1.179	2.742	1	0.098	7.043	0.699	71.002
	FS POLAR SMA (2)	-0.576	1.539	0.140	1	0.708	0.562	0.028	11.471
	FS POLAR SMA (3)	-1.201	1.539	0.569	1	0.451	0.301	0.013	6.826
	FM SHAPE			6.856	3	0.077			
	FM SHAPE (1)	-0.979	1.384	0.501	1	0.479	0.376	0.025	5.659
	FM SHAPE (2)	1.431	1.043	1.882	1	0.170	4.181	0.542	32.285
	FM SHAPE (3)	2.250	1.116	4.062	1	0.044	9.484	1.064	84.540
	CONSTANT	-5.751	1.870	9.460	1	0.002	0.003		

Green = Overall significance of the variable. Yellow = Significance between levels within a variable's sub-categories.

The final step of the regression testing concerning DDD had a predictive value of 0.0% for probable diagnoses and 82.3% overall and displayed no variables with any level of significance for DDD (see Table 7.95 and Table 7.96). FSA and FMR, while not significant, were in the final step of the testing. This indicates that these two variables are better predictors of DDD than the null model.

Table 7.95 The classification table showing the predictive power of the final stage regression test for DDD.

CLASSIFICATION	ABSENT	100.0%
	PROBABLE	0.0%
	OVERALL	82.3%

Table 7.96 The final variables of the binomial logistic regression for degenerative disc disease with B and expected B values, as well as Wald scores and significance.

		VARIABLES IN THE EQUATION							
		B	S.E.	WALD	DF	SIG	EXP(B)	95% CI FOR EXP(B) LOWER	UPPER
STEP 12	FS AREA			6.504	3	0.089			
	FS AREA (1)	0.724	0.723	1.002	1	0.317	2.062	0.500	8.503
	FS AREA (2)	1.090	0.757	2.073	1	0.150	2.974	0.675	13.114
	FS AREA (3)	-0.758	0.943	0.645	1	0.422	0.469	0.074	2.979
	FM ROBUSTICITY			6.042	3	0.110			
	FM ROBUSTICITY (1)	-0.559	0.678	0.680	1	0.409	0.572	0.152	2.158
	FM ROBUSTICITY (2)	-1.768	0.901	3.847	1	0.505	0.171	0.029	0.999
	FM ROBUSTICITY (3)	0.295	0.741	0.159	1	0.690	1.344	0.315	5.739
	CONSTANT	-1.552	0.587	6.989	1	0.008	0.212		

The omnibus tests of model coefficients appear satisfactory because each successive step the p value approaches =.000, making each successive test more significant than the prior one. However, the model summary shows that even though the successive steps remove insignificant variables, thereby reducing statistical noise, and the model becomes more significant, less is being explained as the R^2 values decrease and the -2-log likelihood increases with each step. The Hosmer and Lemeshow Goodness-of-Fit tests would seem to verify that each step is explaining something, though the low Chi-square values can be explained by low sample sizes. The classification table was too indiscriminate to offer any explanation or assurance of a truly predictive model. Therefore, as each successive step model becomes more significant, reducing the noise in the data, less variance at each step is being explained.

DDD was the only joint disease to not have a single significant correlation with the variables used. General osteoarthritis showed significant relationships with age at death, EC and FSR as the variables increased in value. SOA showed significant relationships with EC as the categories increased, FSJ only as the lower values were increased as well as a general relationship with FSS. HOA showed significant relationships with age at death as it increases and biological sex with a lean towards the females. BMI and FMJ showed significant relationships across the categories as the values increase and FMA was significant with the values increasing but was slighter than the others. KOA showed significant relationships with BMI and FMS as the values increased.

7.6 Summary

<u>Key Findings:</u>

Demographics:

- Rural v urban split evenly
- Males had a slightly higher presence than females (3:2)
- Percentage for age at death showed higher late and early than middle adults

Joint Conditions:

- OA and SOA had largest prevalences

- OA, SOA and DDD only JC to display large difference between site categories
- Late adults tended to have larger prevalences for the JC
- AS and SI had too low of a sample size for viable analyses

Aetiological Factors:

- Age at death did not display significance with the dependent variables
- Biological sex displayed weak positive significance with
- The CSG were positively and strongly intercorrelated (exception of shape)
- Age at death (OA) and FSS (SOA) were only non-weak significant variables

Statistical Analyses:

- Biological sex, age at death and EC displayed risk for OA as they increased
- Regression testing displayed DDD to be only JC that did not show significance with variables
- Final variables in testing
    - OA: age at death, EC, FSR
    - SOA: EC, FSS, FSJ
    - HOA: age at death, BMI, FSS, FMJ, FMA
    - KOA: BMI, FSR, FSJ, FMJ
    - Biological sex not found in final step for any JC

The final variables were not uniform, and each joint condition had different variables and a different number of them remaining. While age at death was significant during the bivariate testing of the joint conditions, it was only found in the final step of two: general and hip osteoarthritis. Therefore, no single variable proved to be found more commonly to be a stronger predictor of the conditions than any other, which could implicate underlying factors that were not visible during the testing and analysis of the data within this body of research. While the statistical assessment helps to show some of the deeper meaning and relationships, it is still important to understand and evaluate the surface patterns and trends which will be discussed in the following chapter.

Chapter 8 Discussion

In the following discussion, the key patterns illumined in the preceding results section are identified, elaborated, and explored, with a view to explaining their contribution to understanding the relationship between skeletal manifestation of joint conditions and the key risk factors implicated for these diseases in the clinical literature, assessed here through their skeletal proxies. To contextualize the data gathered in the previous chapter, they are compared to two bodies of collated comparative data. The first concerns post-medieval cemetery populations from England, thereby enabling the data from this study to be compared with other populations of similar time period and broadly comparable lifestyle. The second concerns clinical data from modern populations, which benefits from levels of scope and detail not possible for the archaeological data.

The discussion begins by exploring the relationship between the joint conditions with respect to site category, and the demographic risk factors biological sex and age at death, concluding with a comparison of prevalence rates for the joint conditions between archaeological sites in England spanning the last millennium. The second section goes on to focus on BMI, entheseal change and femoral cross-sectional geometry as potential risk factors in joint condition. The section ends with a reflection on the study's approach to the study of ankylosing spondylitis and sacroiliitis, whose prevalence was too low to be able to pursue the desired course of research.

8.1 Joint Conditions: Prevalence by Site Type, Sex and Age

In the preceding results section, prevalence of osteoarthritis of the spine, hip and knee and degenerative disc disease, was compared with site category and demographic factors. The conditions were measured against each factor individually and then further measured by adding an additional independent factor. In the following section, the findings of these analyses are interpreted, discussed, and contextualized by comparing the results of this study against similar research. These comparisons of prevalence rates of spinal, hip, and knee osteoarthritis will occur using clinical data and, where possible, of the archaeological data of the English sites of the early medieval, medieval, post-medieval and modern periods.

8.1.1 Urban vs Rural Sites

The samples were split between urban and rural population types, which facilitated the exploration of differences in joint condition prevalence based on geographical and occupational factors (See Materials Section 5.4) (Croft *et al.,* 1992; Baetsen *et al.,* 1997; Meinzer *et al.,* 2019). Hip and knee osteoarthritis displayed a prevalence similarly distributed between the urban and rural populations (12.2%/ 12.9% and 10.3%/ 9.4% respectively), although with HOA, there was a trend of higher prevalence at younger ages within the rural male population (see Section 7.3.8). General osteoarthritis and spinal osteoarthritis were more prevalent among the rural population (38.8%/ 30.0% and 29.4%/ 19.3%) and degenerative disc disease was more common in the urban population (22.9%/ 11.8%). Although these differences did not attain statistical significance, the trends in prevalence between site types would suggest that differences between the population types do influence both the extent of joint condition and the development of different kinds of joint condition and, with a larger sample size, this could be further tested (Van Saase *et al.*, 1989; Meinzer *et al.,* 2019; Williams *et al.,* 2019).

Prevalence of the joint conditions at four of the seven geographical site locations used within this research were compared (South Shields, Wolverhampton, Kingston-upon-Thames, and Barton-upon-Humber). The remaining two (Sheffield and Bristol) had sample sizes too low to be compared on their own. General and spinal osteoarthritis was found to have the lowest prevalence rates in Wolverhampton; however, this could be confounded by the relatively low sample size of the site. Wolverhampton, Kingston, and Barton followed the historical trend of hip osteoarthritis having higher prevalence rates than knee osteoarthritis (5.0%/ 0.0%, 11.1%/ 6.3% and 12.9%/ 9.4%). Therefore, while the urban and rural split displays a higher degree variation between the two categories for general and spinal osteoarthritis, the variation appears less pronounced for knee osteoarthritis and degenerative disc disease, and non-existent for hip osteoarthritis. For more information on potential explanations for the differences between the sites or site categories, see the following Discussion Section 8.2 Risk Factors. South Shields did not fall into the traditional trend of hip osteoarthritis having a larger prevalence than knee osteoarthritis, as the prevalence rates were similar (Rogers and Dieppe, 1994). A 1998 national survey conducted by the French National Survey on Health Impairment and Disability (Rossignol *et al.,* 2002), sorted the prevalence of osteoarthritis symptoms for 10,412 patients

by site category (urban/rural), age, biological sex, and overarching occupation type (white collar, blue collar, agriculture, commercial and mixed). The mean age of the individuals in the survey was 66.2 with a mean duration of osteoarthritis being 9.3 years and the average age of onset being 57.0 years of age. The diagnoses were mixed with 91.2% being radiographic. Prevalence of hip osteoarthritis was 15.7%, knee osteoarthritis was 35.6%, and 37.1% of the individuals surveyed was diagnosed with osteoarthritis at multiple locations. While the survey found different ratios between the occupation groups of the males and females, the interesting observation made was when the individuals began to develop signs and symptoms of the conditions. More than 80% of the population developed the conditions after or around the time they ceased the activities or retired. However, the agricultural grouping developed signs before the other groups, indicating that the physical stressors of performing this type of activities with exposure to the activity for a long length of time, might be the cause. This could explain why there was no significant difference between urban and rural populations for the data within this research as the profession or activity was not the risk factor, but the length of time the activity was performed was. For further assessment with age, see Section 8.1.3.

Combining archaeological data from multiple sites across England dating to the post-medieval period shows the rural category to have the higher prevalence of osteoarthritis by joint location (see Table 8.1). The rations shows that hip has the highest disparity between the 2 population types by a factor of 2.2, with general osteoarthritis having 1.9, knee osteoarthritis having 1.7 and spinal osteoarthritis having 1.5. This difference bears further assessment and will be explored further in the following section with emphasis on including biological sex as a factor.

Table 8.1 Prevalence of osteoarthritis by skeletal location and site category for data from the post-medieval period.

		SITE CATEGORY		
		URBAN	RURAL	RATIO (R:U))
JOINT LOCATION	GENERAL	20.6	38.8	1.9
	SPINAL	19.6	29.4	1.5
	HIP	5.8	12.9	2.2
	KNEE	5.4	9.4	1.7

Further breakdown of the individual sites can be seen in Table 8.2 (Baetsen *et al.,* 1997; WORD database, 2019).

8.1.2 Biological Sex

Variation of the prevalence of the joint conditions varied in a more clearly defined manner with biological sex than site category. There was a trend towards males having a higher prevalence. The ratio of males to females with joint conditions was as follows: general osteoarthritis (1.72), spinal osteoarthritis (1.56), hip osteoarthritis (3.4), and degenerative disc disease (1.9). Knee osteoarthritis was the only condition to be more common among females (1.23).

A higher prevalence rate in females for knee osteoarthritis is comparable with modern clinical studies of individuals in Western Europe (females: 4.5%/ males: 2.5%) (see Figure 8.1 and Figure 8.2) (Cross *et al.,* 2014), however both the rates, and the difference between males and females, were greater within this research (females: 12.7%/ males: 7.9%). The prevalence of hip osteoarthritis between the sexes is ambiguous in modern studies as women tend to have a higher symptomatic prevalence, while men have a higher radiographic prevalence (Cross *et al.*, 2014; Allen and Golightly, 2015). It might be inferred that the skeletal evidence would mirror the higher rate seen in males when assessed radiographically, as in this case the physical changes to the skeleton are being assessed, but there is a wider interpretive issue with the clinical literature raised here. This discrepancy between symptomatic and radiographic prevalence rates, and their implications for the integration of clinical and osteological data in studies of joint conditions are discussed further in section 8.1.3.

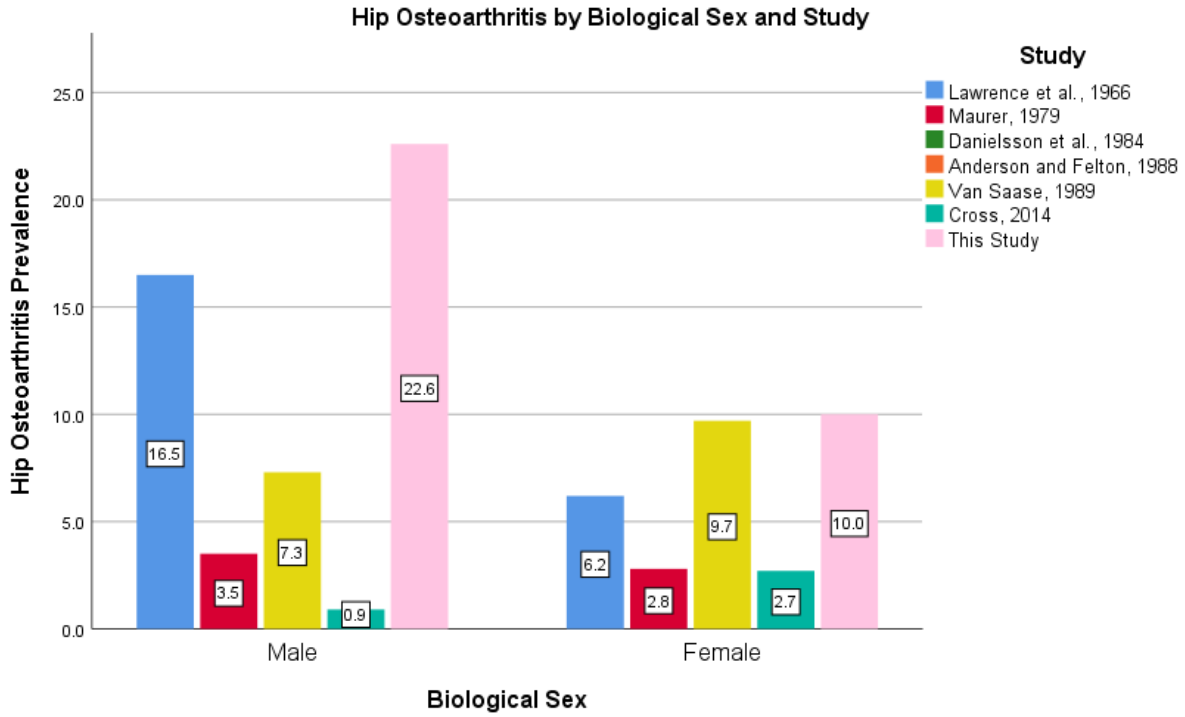

Figure 8.1 Prevalence rates of hip osteoarthritis of the modern clinical studies, sorted by study and biological sex, with the post-medieval data from this study included. The data have been taken from Table 8.4 and where multiple age groups existed, a mean rate was created for comparison. Percentage is in the box.

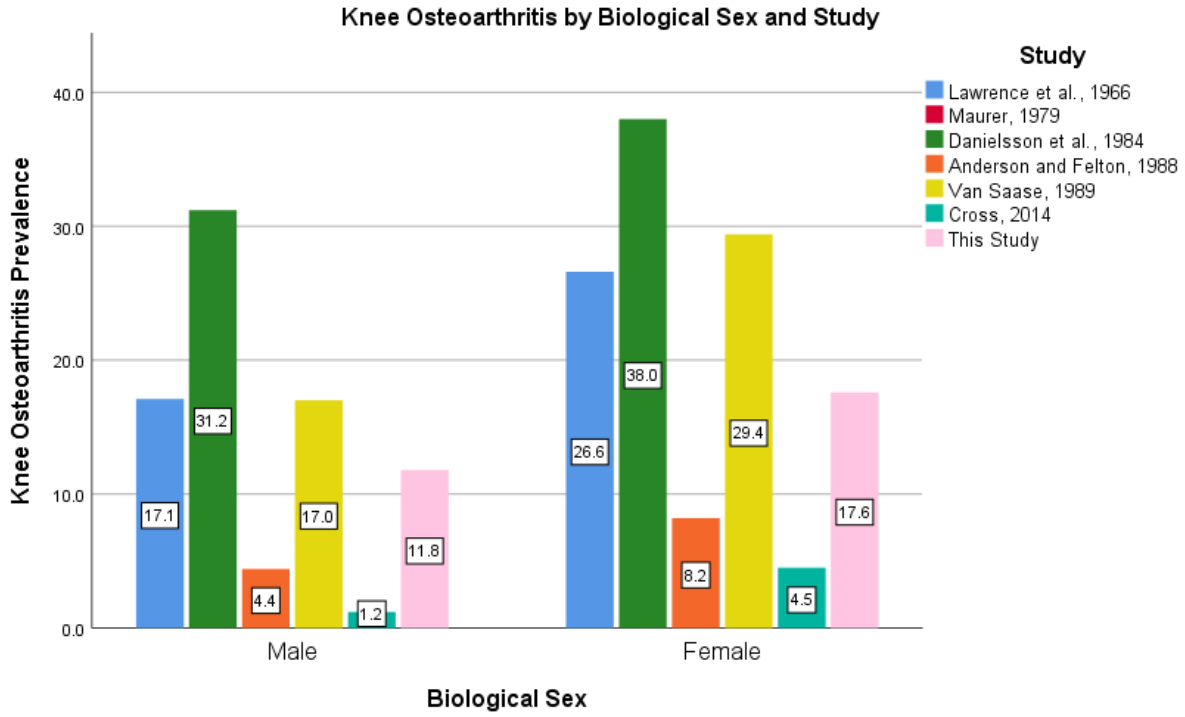

Figure 8.2 Prevalence rates of knee osteoarthritis of the modern studies, sorted by study and biological sex, with the post-medieval data from this study included. The data has been taken from Table 8.4 and where multiple age groups existed, a mean rate was created for comparison. Percentage is in the box.

The post-medieval data sets demonstrate that the prevalence rates of the different joint conditions between the sexes varied by site location (see Table 8.2 and Figure 8.3- Figure 8.5). This data was created from differing sites and is currently held within the Wellcome Osteological Research Database. St Benet Sherehog and St Bride's Lower have similar general osteoarthritis prevalence rates and are far lower than Barton-upon-Humber, but the spinal osteoarthritis rates display no similarity between the former two sites. Instead, Barton-upon-Humber and St Bride's Lower have more similar prevalence rates than the St Benet Sherehog population. The sex with the higher prevalence rate for general osteoarthritis varied by site. There were higher prevalence rates of osteoarthritis in the spine than the hip or knee across all geographical sites, however, as with general osteoarthritis, no single biological sex had a consistently higher prevalence with a total ratio of 1.1 (male: female). Hip osteoarthritis was more prevalent among the males with an overall ratio 1.8, with the exception of individual cases such as Kingston-upon-Thames and St Marylebone. Females had a higher prevalence of knee arthritis than the males at all sites by a factor of 2.2, once more mirroring the clinical study trends. The trend observed earlier for an overall lower prevalence for knee osteoarthritis than hip osteoarthritis did not clearly manifest within the eight sites with comparable data. The rates were narrowly divided with four having a higher prevalence in the knee, three having a higher prevalence in the hip and a single site having similar rates. Therefore, while sites may have common prevalence rates for one joint location, there is no standard set of prevalence rates found across England during the post-medieval period. This would suggest that each site had a unique set of underlying risk factors, that would have impacted on the onset and development of joint conditions for different joint location.

Table 8.2 Distribution of prevalence rates for each joint condition by site location and biological sex.

		POST-MEDIEVAL SITES WITH PERCENTAGE OF OSTEOARTHRITIS											
		SOUTH SHIELDS[3]			WOLVERHAMPTON[3]			KINGSTON-UPON-THAMES[3]			BARTON-UPON-HUMBER[3]		
		TOTAL	MALE	FEMALE	TOTAL	MALE	FEMALE	TOTAL	MALE	FEMALE	TOTAL	MALE	FEMALE
JOINT LOCATION	GENERAL	34.8	40.7	26.3	10.0	8.3	12.5	38.9	36.4	42.9	38.8	42.6	34.2
	SPINAL	20.0	23.1	15.8	11.8	10.0	14.3	26.7	33.3	16.7	29.4	29.8	28.9
	HIP	15.2	18.5	10.5	5.0	8.3	-	11.1	9.1	14.3	12.9	19.1	5.3
	KNEE	15.2	14.8	18.8	-	-	-	6.3	-	16.7	9.4	6.4	13.2
		CHURCH OF ST LAWRENCE*[1]			ST BENET SHEREHOG[2]			ST BRIDE'S LOWER[2]			BROADGATE[2]		
		TOTAL	MALE	FEMALE	TOTAL	MALE	FEMALE	TOTAL	MALE	FEMALE	TOTAL	MALE	FEMALE
JOINT LOCATION	GENERAL	-	-	-	17.6	24.1	27.8	17.5	24.2	28.0	-	-	-
	SPINAL	-	-	-	16.9	15.1	22.2	31.7	34.0	32.8	12.0	15.0	10.0
	HIP	9.2	-	-	4.7	6.2	2.2	2.8	3.2	2.6	-	-	-
	KNEE	6.4	-	-	2.4	1.2	4.3	3.8	3.6	4.0	-	-	-
		CHELSEA OLD CHURCH[2]			ST MARYLEBONE[2]			ST THOMAS' HOSPITAL[2]			[1]- Baetsen *et al.,* 1997		
		TOTAL	MALE	FEMALE	TOTAL	MALE	FEMALE	TOTAL	MALE	FEMALE	[2]- WORD database, 2019		
JOINT LOCATION	GENERAL	23.2	28.2	25.7	18.4	19.8	20.9	4.1	5.2	3.4	[3]- Data from McAfee research		
	SPINAL	28.8	31.4	22.4	24.6	23.4	28.0	4.3	4.2	6.5			
	HIP	2.0	2.6	1.4	0.9	0.6	1.3	1.1	1.7	-			
	KNEE	6.6	5.1	8.1	1.6	-	0.7	1.1	-	3.4			

* - Location is outside of England at Alkmaar, Netherlands.

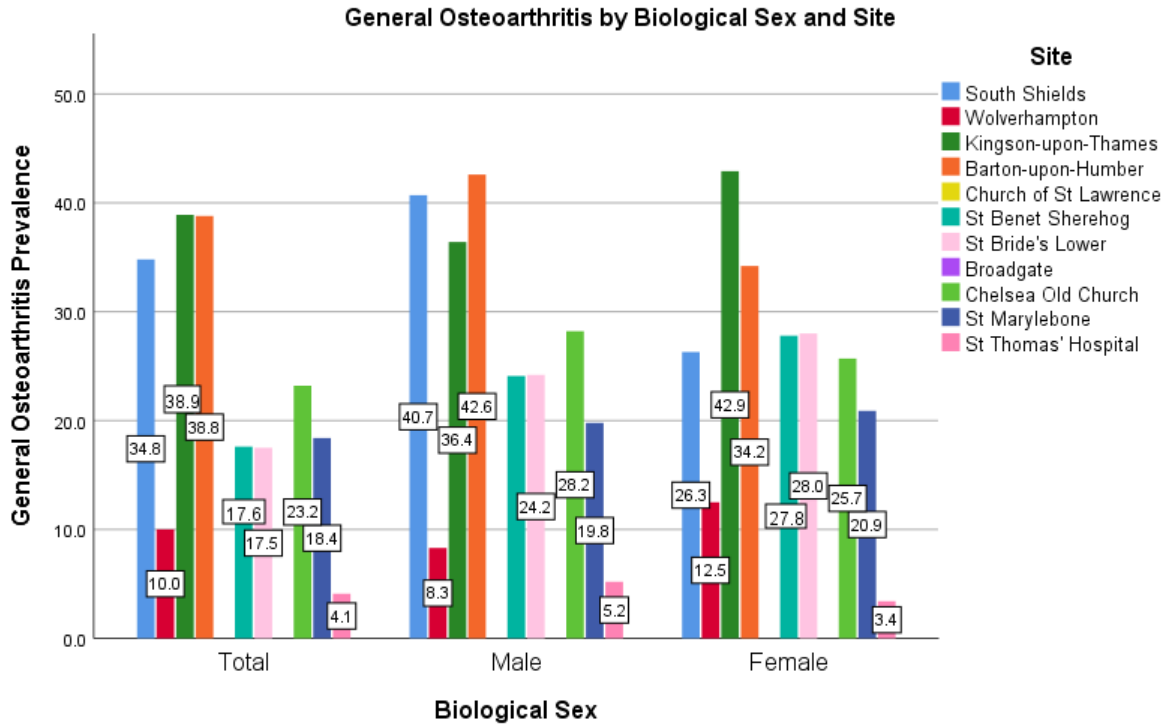

Figure 8.3 Prevalence rates of general osteoarthritis by post-medieval site location and sorted by biological sex. Percentage is in the box.

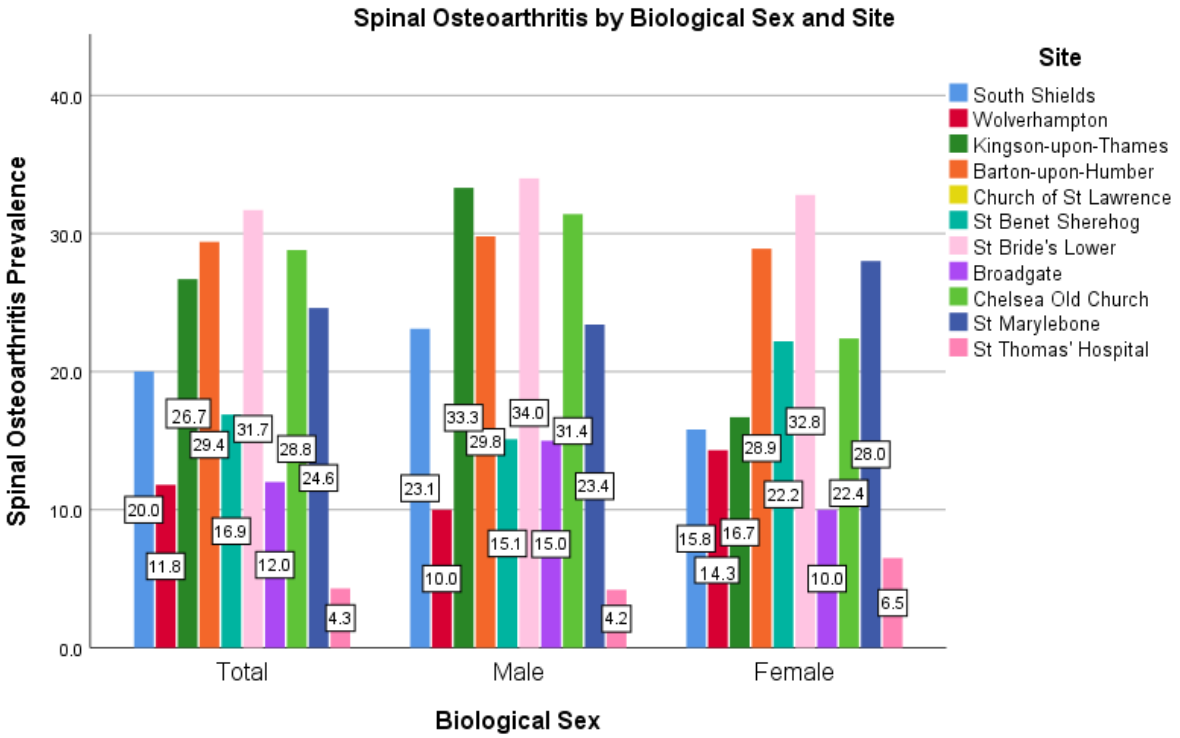

Figure 8.4 Prevalence rates of spinal osteoarthritis by post-medieval site location and sorted by biological sex. Percentage is in the box.

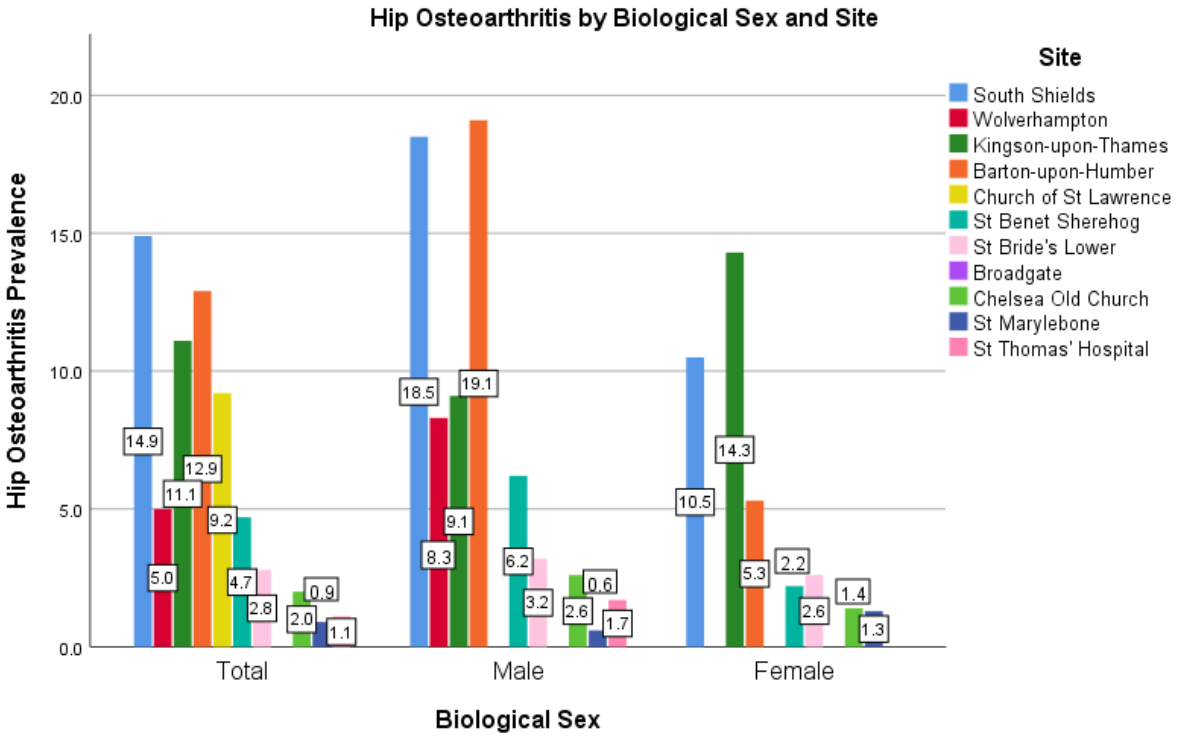

Figure 8.5 Prevalence rates of hip osteoarthritis by post-medieval site location and sorted by biological sex. Percentage is in the box.

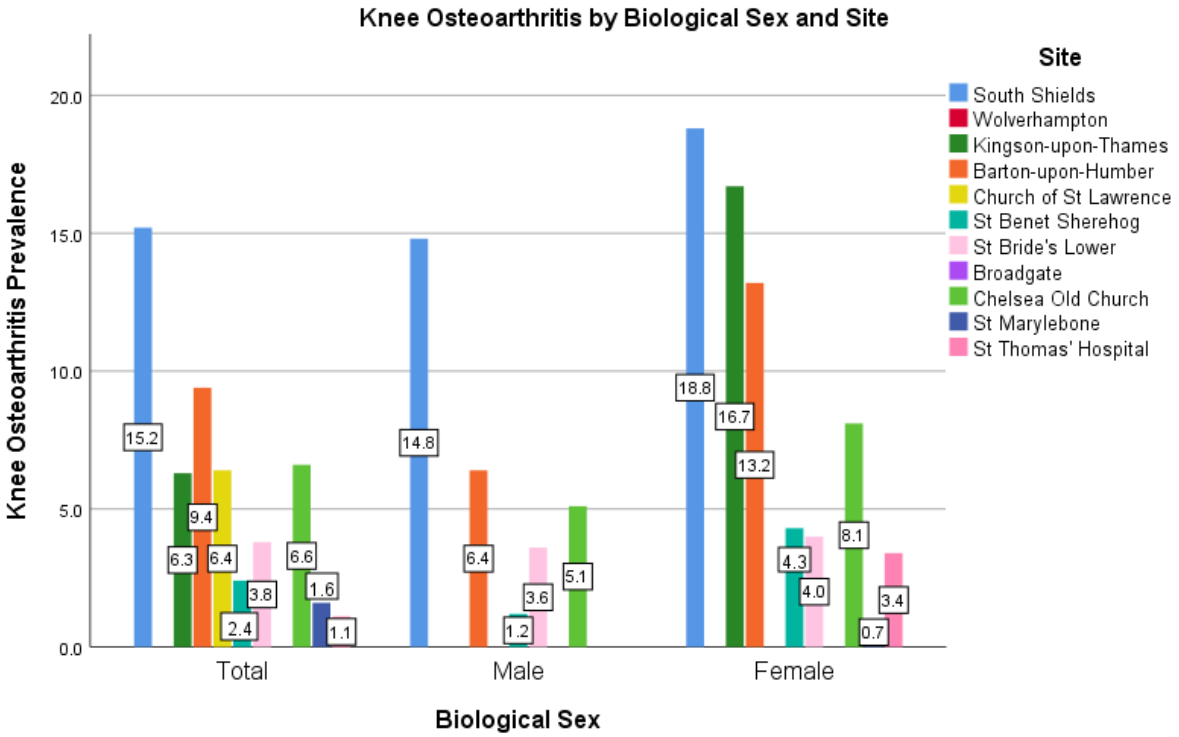

Figure 8.6 Prevalence rates of knee osteoarthritis by post-medieval site location and sorted by biological sex. Percentage is in the box.

Table 8.3 Ratios of osteoarthritis by joint location and site.

		SOUTH SHIELDS[3]	WOLVERHAMPTON[3]	KINGSTON-UPON-THAMES[3]	BARTON-UPON-HUMBER[3]
		RATIO	RATIO	RATIO	RATIO
JOINT LOCATION	GENERAL	1.5	0.8	0.8	1.2
	SPINAL	1.5	2.0	2.0	1.0
	HIP	1.8	0.6	0.6	3.6
	KNEE	0.8	-	-	0.5
		CHURCH OF ST LAWRENCE[1]	ST BENET SHEREHOG[2]	ST BRIDE'S LOWER[2]	BROADGATE[2]
		RATIO	RATIO	RATIO	RATIO
JOINT LOCATION	GENERAL	-	0.9	0.9	-
	SPINAL	-	1	1	1.5
	HIP	-	1.2	1.2	-
	KNEE	-	0.9	0.9	-
		CHELSEA OLD CHURCH[2]	ST MARYLEBONE[2]	ST THOMAS' HOSPITAL[2]	TOTAL
		RATIO	RATIO	RATIO	RATIO
JOINT LOCATION	GENERAL	1.1	1.5	1.5	1.0
	SPINAL	1.4	0.6	0.6	1.1
	HIP	1.9	-	-	1.8
	KNEE	0.6	-	-	0.4

Ratios are Male: Female. Values based on Table 8.2. [1]- Baetsen *et al.,* 1997. [2]- WORD database, 2019. [3]- Data from McAfee research.

8.1.3 Age

Prevalence of joint condition increased with age at death, with the early adult category typically having the lowest prevalence and late adults having the highest prevalence. The exception was hip osteoarthritis, which had a slightly higher prevalence within early adults than in middle adults resulting in a decline between the first two age categories. However, age at death presented the most defined relationship with prevalence rates compared the previous two factors, site, and sex. This would fit with most established research regarding joint conditions, especially for variants of OA, that the prevalence increases as age increases (Rogers and Waldron, 1995; Weiss, 2006). This appears to be a constant, at least within humans and can be seen in a prehistoric Californian Amerind population, in which the variants of OA were found to be positively correlated with increased age (SOA: r = .507, HOA: r = .507, KOA: r = .528) (Weiss, 2006).

The prevalence of joint conditions with age at death does appear to fit with clinical studies (see Table 8.4), with the higher rates of the condition appearing among individuals in older age categories, and prevalence increasing with age. However, while age may be a risk factor for joint conditions in and of itself, the researcher must take into account that increased age also increases the chance for an individual to be affected by other risk factors (Felson *et al.*, 1989).

Table 8.4 Prevalence rates of osteoarthritis by joint location for modern clinical studies conducted over the last 60 years and sorted by the age categories used in study.

HIP PREVALENCE				KNEE PREVALENCE			
STUDY	AGE GROUPING	MALE	FEMALE	STUDY	AGE GROUPING	MALE	FEMALE
LAWRENCE *et al.*, 1966[1]	<55	16.5	6.2	LAWRENCE *et al.*, 1966[1]	35-44	5.6	4.0
MAURER, 1979[1]	55-74	3.5	2.8		45-54	8.2	13.1
DANIELSSON *et al.*, 1984[1]	40-44	-	0.4		55-64	28.2	40.0
	45-49	0.4	0.4		65+	26.4	49.1
	50-54	0.8	0.4	FELSON, 1987[1]	<70	30.4	25.1
	55-59	1.2	1.2		70-79	30.7	36.2
	60-64	1.6	1.6		≥80	32.6	52.6
	65-69	2.8	0.8	ANDERSON & FELTON, 1988[1]	35-44	1.2	1.2
	70-74	2.4	5.2		45-54	2.2	3.6
	75-79	6.4	4.7		55-54	5.1	7.5
	80-84	11.5	5.0		65-74	9.0	20.3
	85-89	5.6	10.0	VAN SAASE, 1989[1]	45-49	7.7	12.7
VAN SAASE, 1989[1]	45-49	2.8	2.6		50-54	11.2	16.1
	50-54	2.2	2.0		55-59	11.8	14.0
	55-59	5.9	2.6		60-64	23.0	24.2
	60-64	10.1	3.8		65-69	13.1	33.3
	65-69	11.2	10.9		70-74	24.7	40.2
	70-74	4.7	14.8		75-79	22.0	40.2
	75-79	10.2	14.5		≥80	22.2	54.6
	≥80	11.1	26.0	CROSS *et al.*, 2014	-	2.7	4.5
CROSS *et al.*, 2014	-	0.9	1.2	THIS THESIS	<45	4.9	8.1
THIS THESIS	<45	7.3	2.6		45-55	0.0	14.3
	45-55	20.0	0.0		56-65	0.0	0.0
	56-65	0.0	100.0		>66	11.8	19.2
	>66	24.5	11.5				

The data from this study has been modified to fit similar age categories to show a comparison. [1]- Radiographic diagnoses. [2]- Symptomatic and radiographic diagnoses.

There is an inherent limitation when assessing the association of skeletal evidence for joint conditions and age. The researcher does not know the age of onset for the condition or, if the individual died at an older age, how long the condition with skeletal manifestations was present. Severity of the skeletal manifestations could lead to assumptions for how long an individual had the condition, however, there are many factors that could influence the speed of progression that would be unknown, therefore, the assumption may not be reliable or accurate. As age at death did not appear to have a statistically significant relationship with the conditions, at any joint location, the explanation above could justify this statistical irregularity when

comparing archaeological with modern populations at younger ages. This flaw demonstrates the need for further research into the skeletal development of the conditions to help determine if it is possible to assess how long an individual could have been affected. The issue becomes determining and tracing the varying facets that can affect the development and onset of the skeletal manifestations of osteoarthritis such as: (1) How long was the cartilage and joint capsule degraded or destroyed? (2) How long was the individual utilizing the joint before and after the degraded or destroyed cartilage/joint capsule? (3) What was the intensity of the activity on the affected joint before and after the cartilage/joint capsule degraded or destroyed? Therefore, while the onset would not be 'fast' it may be relatively quicker in others due to those factors (see Chapter 3.1) (Fergusson, 1987; Guilak, 2011).

In modern England, 10.9% of individuals over the age of 45 have reported symptoms of hip osteoarthritis (Versus Arthritis, 2012), while this study showed that 14.3% of individuals over the age of 45 display signs of skeletal hip osteoarthritis (see Table 8.5). While these values are relatively similar, the modern grouping is based on symptoms and does not include radiographic evidence that could cause the prevalence to be higher. Underreporting of symptoms is believed to be common as individuals experiencing pain have easy access to analgesics and may have little access to medical facilities (Versus Arthritis, 2012; Cross *et al.,* 2014*).* Another study showed that 50.0% of modern American individuals over the age of 65 had radiographical evidence of knee osteoarthritis (Anderson and Loeser, 2010), while the present study showed only 14.1% of the individuals over the age of 65 displaying skeletal evidence of knee osteoarthritis (see Table 8.6). This finding would support the theory that tibiofemoral osteoarthritis is a more modern condition, increasing the overall prevalence of knee osteoarthritis in modern populations (Rogers and Dieppe, 1994). The potential role of obesity in this argument will be discussed in the following section.

Table 8.5 Distribution of prevalence rates from this study by biological sex using an age cut-off of 45 years.

		GENERAL OA		SPINAL OA		HIP OA		KNEE OA	
		N	%	N	%	N	%	N	%
TOTAL	<45	17	21.5	11	14.3	4	5.1	5	6.4
	>45	43	44.8	30	33.0	18	18.8	12	12.8
MALE	<45	9	22.0	5	12.8	3	7.3	2	4.9
	>45	29	46.8	20	33.9	14	22.6	6	10.0
FEMALE	<45	8	21.1	6	15.8	1	2.6	3	8.1
	>45	14	41.2	10	31.3	4	11.8	6	17.6

Note that degenerative disk disease has been removed, as the comparative studies do not include this condition.

Table 8.6 Distribution of prevalence rates between refined age groups for comparison with different studies.

		GENERAL OA		SPINAL OA		HIP OA		KNEE OA	
		N	%	N	%	N	%	N	%
TOTAL	<45	17	21.5	11	14.3	4	5.1	5	6.4
	45-55	1	8.3	1	8.3	1	8.3	1	8.3
	56-65	2	40.0	1	25.0	1	20.0	0	0.0
	>66	40	50.6	28	37.3	16	20.3	11	14.3
MALE	<45	9	22.0	5	12.8	3	7.3	2	4.9
	45-55	1	20.0	1	20.0	1	20.0	0	0.0
	56-65	1	25.0	1	25.0	0	0.0	0	0.0
	>66	27	50.9	18	36.0	13	24.5	6	11.8
FEMALE	<45	8	21.1	6	15.8	1	2.6	3	8.1
	45-55	0	0.0	0	0.0	0	0.0	1	14.3
	56-65	1	100.0	0	0.0	1	100.0	0	0.0
	>66	13	50.0	10	40.0	3	11.5	5	19.2%

The percentages are by the individual refined age grouping within each joint condition.

The differences stated above highlight two discrepancies when making comparisons with modern clinical data: (1) the age groupings used change by study/researcher and (2) the criteria for diagnoses (strict v. broad) and type of diagnoses (symptomatic v. radiographic) changes by study/researcher. The age categories for this study were used based on clinical ages of onset for the highest occurrence of the conditions (65+ for osteoarthritic conditions) (Sharma and Kapoor, 2007), with other studies using their own justifications for age groupings. While the data within this research can be modified to match the age groupings, cross-comparisons with similar clinical research may not be as easily altered to match

When the prevalence of joint condition by age is also broken down by sex, an additional range of patterns emerge (see Table 8.5 and Table 8.6). Under the age of 45, females had the higher prevalence of spinal and knee osteoarthritis, while males had the higher prevalence of general and hip osteoarthritis. In total, knee osteoarthritis was more prevalent under the age of 45 than hip, but over the age of 45 hip became more prevalent. However, the trend does not hold true for both males and females. Males had a higher prevalence of hip osteoarthritis across all ages, while females had a higher prevalence of knee osteoarthritis across all age categories. As the joints have different biomechanical functions and tolerances (Radin and Paul, 1977; Radin *et al.* 1991; Meinzer *et al.,* 2019), this would suggest that the different activity types caused by a division of labour between the sexes could be responsible.

Due to the effects menopause and a deficiency of the hormone estrogen causing a destabilization of the homeostasis within the joint capsule (Roman-Blas *et al.* 2009), females should have the higher prevalence rates of joint condition in the earlier age groups (Nevitt, 2006; Stevens-Lapsley and Kohrt, 2010). The prevalence rates found within this study are higher for general osteoarthritis, spinal osteoarthritis, and knee osteoarthritis, but not hip osteoarthritis (See Table 7.7). Over the age of 50, the rates should sharply increase in females due to menopause (Felson, 1990; Kohrt, 2010; Mahajan and Patni, 2018). While there are anomalies due to low sample sizes between the refined age groups, the prevalence rates more than double for all locations when comparing the <45 and the >65 age groups. This would suggest, that while the prevalence rates differ to clinical models, the change in rates for females as age increases has remained consistent with the clinical literature. However, there is no way to check for potential hormone levels with any certainty for the skeletal remains utilized within for this study to determine if this was the cause.

8.1.4 Historical Trends in Joint condition Prevalence

Comparing the means and ranges of the prevalence rates of osteoarthritis of the post-medieval sites helps to display the historical trends of that time period. However, as all of the sites shown in Table 8.2 are urban sites, with the sole exception of Barton-upon-Humber, the differences between urban and rural populations are not as clear as it could be. The overall urban data from this research displayed prevalence rates higher than the post-medieval means (see Table 8.7). The higher rates of general osteoarthritis can be explained as a comparison

issue, with only three joint locations being used within this study and all available joints being used for the Wellcome Osteological Research Database. For the majority, the mean data from the current study was higher than the mean data from all the post-medieval sites. The exceptions are the total and female mean for spinal osteoarthritis, which were lower. The sites differed in their prevalence, but the mean percentages show that males had a higher prevalence during the post-medieval period of general, spinal, and hip osteoarthritis than the females and the mean percentages of this body of research shared this trend.

Table 8.7 Prevalence rates of the joint conditions with the urban post-Medieval sites include the data from this study as well as the data from Table 8.2.

		POST-MEDIEVAL SITE PREVALENCE RATES								
		TOTAL			MALE			FEMALE		
		MEAN	RANGE		MEAN	RANGE		MEAN	RANGE	
			MIN	MAX		MIN	MAX		MIN	MAX
JOINT LOCATION	GENERAL	22.6	4.1	38.9	25.5	5.2	40.7	24.6	3.4	42.9
	SPINAL	20.6	4.3	31.7	21.9	4.2	34.0	19.8	6.5	32.8
	HIP	6.5	1.1	14.9	7.7	1.7	18.5	5.4	1.4	14.3
	KNEE	5.8	1.1	15.2	6.2	1.2	14.8	8.7	0.7	18.8
		URBAN SITE PREVALENCE RATES FROM THIS STUDY								
		TOTAL			MALE			FEMALE		
		MEAN	RANGE		MEAN	RANGE		MEAN	RANGE	
			MIN	MAX		MIN	MAX		MIN	MAX
JOINT LOCATION	GENERAL	27.9	10.0	38.9	28.5	8.3	42.6	38.6	12.5	42.9
	SPINAL	19.5	11.8	29.4	24.1	10.0	33.3	11.7	14.3	16.7
	HIP	10.3	5.0	14.9	13.8	8.3	19.1	10.0	5.3	14.3
	KNEE	10.8	6.3	15.2	10.6	6.3	14.8	16.2	13.2	18.8

The prevalence rates of the sites listed in Table 8.2 have been merged to create a mean of the percentages as well as displaying the minimum and maximum range limits.

Different diagnosing criteria can explain the higher prevalence rates for the specific site locations. For St Marylebone Church, strict criterion, namely the presence of eburnation, was used by Miles and colleagues to diagnose osteoarthritis (Miles *et al.,* 2008). This is not inherently a problem and is perfectly acceptable when justified, however, when using the strict criterion, the prevalence rate is lowered as it excludes potential arthritic samples that never developed the marker, which can skew the comparison of data. The Waldron and Rogers (1995) criterion was also used for diagnosis within this study; however, the strict criterion was used in conjunction with the diagnosing criteria where in the absence of eburnation, three alternative markers must be present. Eburnation is pathognomic of osteoarthritis, and it creates a

conclusive diagnosis, but eburnation is a late-stage marker that does not always manifest on individuals with osteoarthritis (Waldron, 2009; Burt *et al.,* 2013). Another issue was the manner of recording to be used by other researchers, in that information may not have been as detailed as it could have been (e.g., simply recording the presence of OA but not the markers present). This makes it difficult to check diagnoses and allows for no comparison further than with the final diagnoses. For the purposes of this study, a comparison was able to be made, but the difference in reporting and diagnosing exemplifies the need for a standard to be utilized.

The comparison of osteoarthritis over time shows interesting shifts between prevalence rates (see Table 8.8/ Figure 8.7 - Figure 8.9). Spinal OA prevalence rates have steadily decreased from pre-medieval sites through to modern times, while knee OA has largely increased. Hip OA shows a different trend than the steady increase/decrease, but rather fluctuates over time. The data from this research does give the impression of fitting within the general trends, however the spinal OA is somewhat higher than the other sites of the post-medieval period. There is an observed difference between the general post-medieval period and the research from this study which was post-medieval but specific to a roughly 150-year range within the 18th and 19th centuries CE. However, there is no true consensus as to the appropriate range of the post-medieval time categories, as some authors divide the latter post-medieval period into a separate and distinctive industrial era (1760 – 1840 CE), which ends roughly half a century prior to the end of the post-medieval period. Another difference in terminology lies in the difference between the post-medieval and early modern periods. These periods are considered to start at roughly the same point in time within British history, the dissolution of the monasteries, although the end of the periods differ with the post-medieval ending with the close of the 18th century CE and the early modern ending with the close of the 17th century CE with the union of Great Britain and Ireland being the start of the late modern period (Lang, 2011; Barber *et al.,* 2019). Therefore, one researcher or study may exclude samples that they feel fall into a different historical period, while another may include them as their period definition is different. For the purposes of this study, the author was forced to be selective of the comparative data used to avoid creating a bias in the interpretation and choose selections that fit the range of years as closely as possible.

Table 8.8 Prevalence of osteoarthritis by skeletal location and historical period gathered from English archaeological sites.

	PRE-MEDIEVAL	SAXON/ MEDIEVAL	MEDIEVAL	POST-MEDIEVAL	18-19TH C	MODERN
SPINAL	31.9[3]		31.7[3]	24.0[3]	24.4*	16.9[2]
HIP	12.8[3]	3.7[1]	5.7[3]	3.3[1]/2.9[3]	12.6*	10.9[2]
KNEE	2.1[3]	1.8[1]	5.0[3]	5.5[1] /4.4[3]	9.9*	18.2[2]

The figures represent percentages. The modern data only represents individuals over the age of 45 and is a mix of symptomatic and radiographic diagnoses. [1] - Rogers and Dieppe, 1994; [2] - Versus Arthritis, 2012; [3] - Waldron, 1995; * - this research

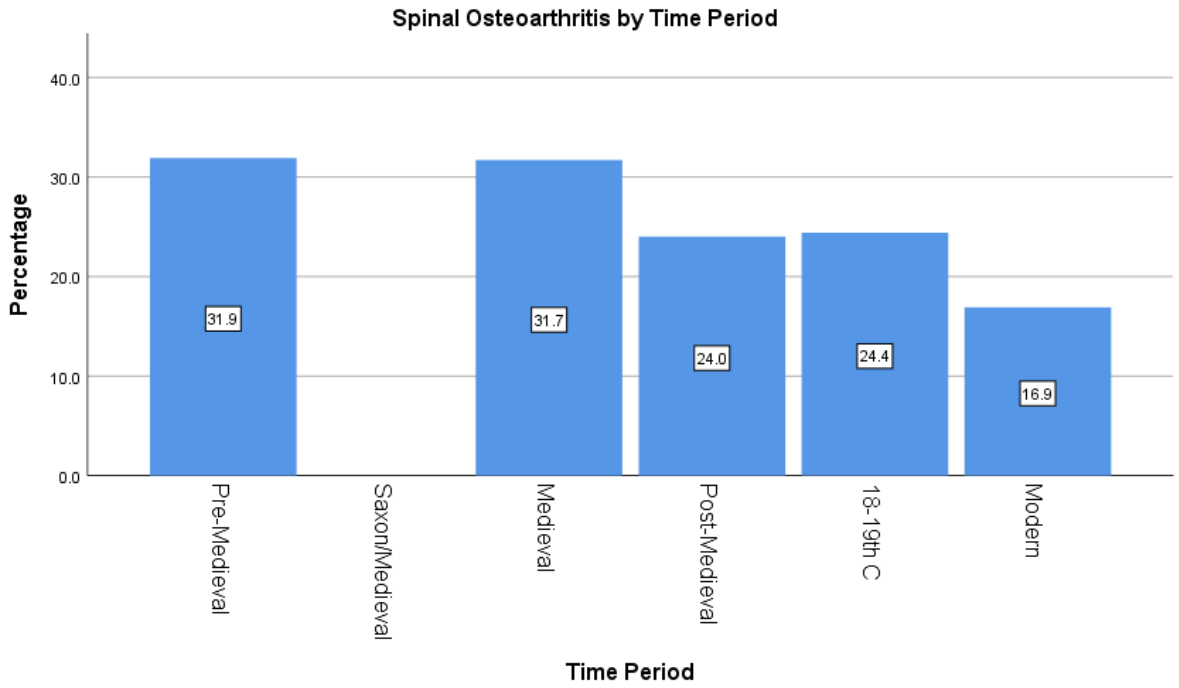

Figure 8.7 Prevalence rates of spinal osteoarthritis from English populations across historical time periods. Note the downward trend. Percentage is in the box.

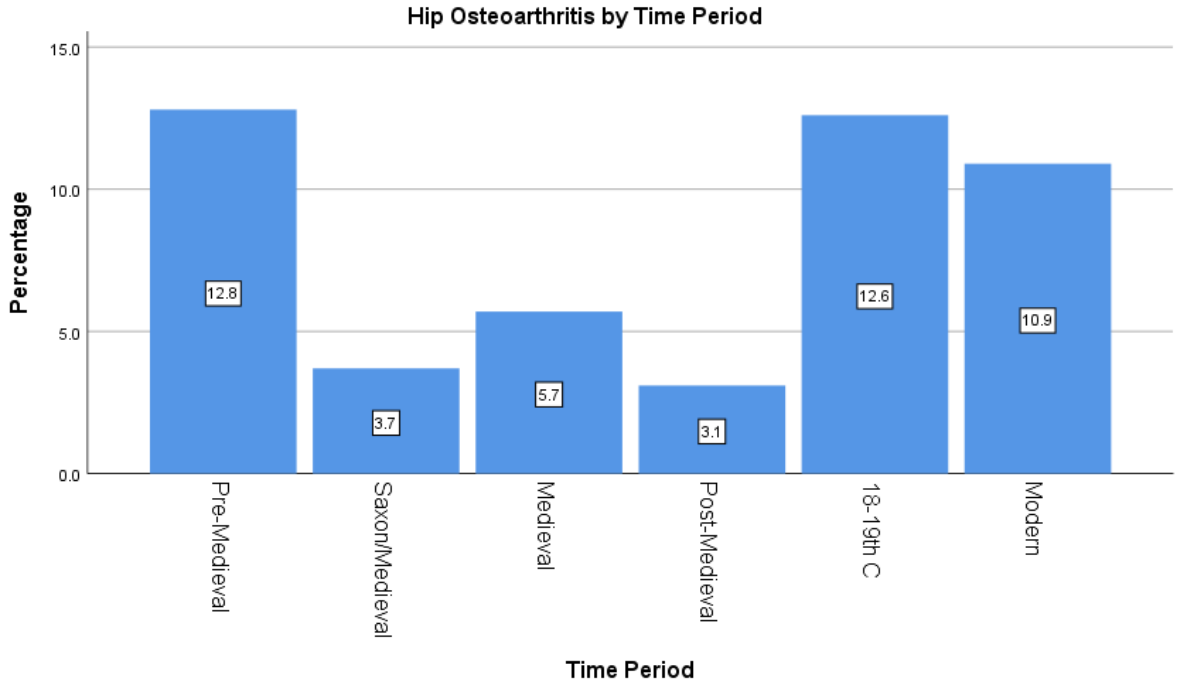

Figure 8.8 Prevalence rates of hip osteoarthritis from English populations across historical time periods. Percentage is in the box.

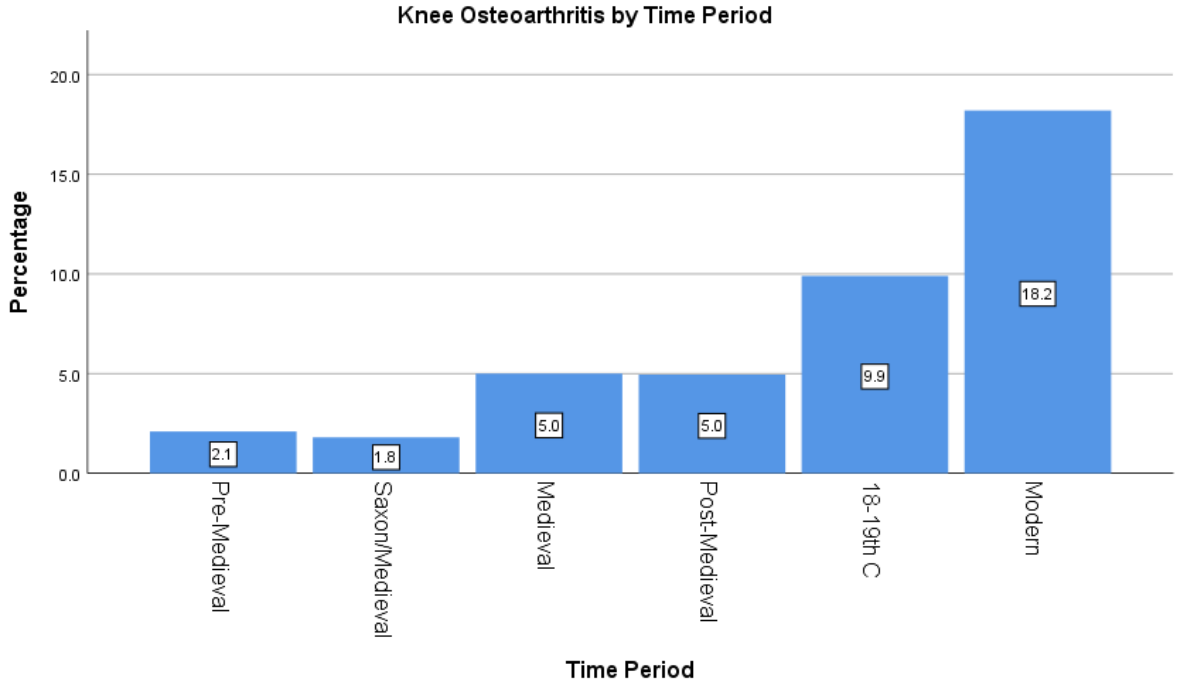

Figure 8.9 Prevalence rates of knee osteoarthritis from English populations across historical time periods. Note the increasing trend leading up to the modern age. This could be explained from the additional tibiofemoral osteoarthritis reported in clinical studies. Percentage is in the box.

There is also a noticeable difference between knee and hip prevalence of the modern population and that of the 18-19th C CE population. Modern clinical literature shows that knee

OA prevalence is almost double that of hip OA (Versus Arthritis, 2012; Williams *et al.*, 2019), however the 18-19th C CE population displayed the hip prevalence being 1.28 times higher than the knee OA rates. The gradual increase of knee osteoarthritis over time could support the theory that before the modern period, the tibiofemoral joint, one of two separate joints of the knee, was not as affected by osteoarthritis as the patellofemoral joint (Rogers and Dieppe, 1994). This would cause an overall rise in prevalence knee osteoarthritis as both joints that comprise the knee joint are being affected, rather than just the one, giving a greater chance that KOA will develop. A second explanation could be the different diagnostic criteria between skeletal and modern medical assessments, however, as the radiographic analysis and skeletal assessments used look at similar objective criteria, the differences should be minimal. Without contemporary records, it is impossible to know whether the individuals of the 18-19th C CE population were outwardly affected by their joint conditions, whereas a clinical assessment takes place after a patient has sought treatment or clinical assistance for the symptoms. Comparisons with modern data, which due to the different diagnosing criteria may displayed differing prevalence rates and trends, may not be the most accurate. Radiographic diagnoses in a clinical setting are comparable to osteological diagnoses, the problem lies in that not all patients will receive that treatment until well after arthritic symptoms have set in. Therefore, a more probable answer could be the shift in lifestyle trends causing increased stress to one joint location, while decreasing the stress to another.

Another explanation for a disparity between the prevalence rates could be a shift in lifestyle pattern, diet and medical treatment brought about during the industrial revolution (Van Saase *et al.*, 1989). Osteoarthritis has been found to shift prevalence rates by joint location dependent on the change of activity patterns of the test group (Williams *et al.,* 2019) and the industrial revolution would have changed the activity patterns and lifestyles of people as new technologies emerged and would explain the difference in prevalence between this specific period population and the general post-medieval population. This would also explain the differences, however small they may have been, between the urban population with a mobile workforce and new industrial jobs, such as at a factory, and the rural population with new innovating farming practices and equipment (Meinzer *et al.,* 2019).

8.1.5 Summary

With the prevalence rates examined in more detail, it was possible to attempt to determine the cause of the trends. The higher prevalence of joint conditions at older ages conforms to the clinical evidence that the conditions are age related and that archaeologically visible cases follow the standard trend pattern seen in clinical research of increased prevalence as the age groups increase (Cross *et al.* 2014). However, the conditions were present in younger individuals and age at death showed significant correlations with only three of the conditions and associations with only two, which would also appear to confirm that underlying issues are influencing the onset of the conditions.

Finally, the prevalence rates were assessed to consider the influence of site category and the demographic profiles on each joint condition. By combining the factors in such a way, it was then possible to assess the entire sample population in a way that would increase specificity of the prevalence rates and would be more representational of the living population. In general, the trends show an increase of joint condition as an individual's age increases for males and females when further divided by each location and the site category, with few exceptions showing a spike in prevalence in either the early or middle adult category.

Prevalence of joint condition by biological sex is not consistent among urban or rural sites or across all the joint conditions. General osteoarthritis prevalence was higher in males than females, but the prevalence among rural males and females shows that the female population had the higher rate, and the males had a spike in the prevalence within early adults. A similar occurrence happens with degenerative disc disease, whereas urban males had a steady prevalence increasing with age, with urban females having the higher prevalence at older ages, but rural females have a spike in prevalence within the early adult age category. As joint condition is regarded as being highly related to age of the individual, these differences would indicate that there are additional factors affecting the development and progression of the joint conditions. The difference between site categories will be explored further within the discussion section concerning risk factors (see Section 8.2), where the different variables for mass and activity will be assessed. The statistical testing showed countertrends to the traditional clinical and palaeopathological literature that bear further examination, such as the lack of signification correlation or association with age at death and biological sex, which would appear to contradict current clinical and palaeopathological research. Biological sex was

not found in the final stages of the regression testing of any joint condition except for HOA, and age at death was found in the final stages for general and hip osteoarthritis. The data from this research has shown interesting and curious findings that bear further testing and interpretation.

The prevalence rates of joint conditions fluctuate not only by site location and biological sex, but by time as seen in current palaeopathological literature. SOA has seen a gradual decline in prevalence, HOA has seen a constant rise and fall, and KOA has seen a steady rise over the last 1500 years. These trends will be explored further in the following section (8.2) to assess with the remaining risk factors used within this thesis.

8.2 Joint Conditions and the Risk Factors

There is a gulf between clinical and palaeopathological approaches to diagnosing and studying joint conditions (see Chapter 3.1). Clinicians can directly observe the symptoms in a living patient and use longitudinal studies to follow individual patients and see how risk factors impact the ecology of the living body and assess how a change to one factor affects the others. Palaeopathologists, on the other hand, examine the individual without that same living variation and will only get to see the effects of conditions as they were at the time of death. To compensate, skeletal approximations, or proxies, of the clinical risk factors that are studied in clinical literature must be used to allow for accurate comparisons of the data. Creating a variable for biological sex or age at death can be relatively easy, as the methods to do so are well documented and are a backbone of palaeopathological assessments, yet there is no consensus on how to measure the risk factors such as activity level or activity type using skeletal material. BMI can easily be assessed by clinicians by taking a standard measurement for height and weight and when assessing activity in a living person, survey questions and an assessment of musculature could help to determine if an individual is over mass due to heavy musculature or obesity. However, palaeopathologists must use multiple methods and variables that rely purely on skeletal material to piece together an estimate of the potential measurements for height or weight, as well as the activity level of an individual. For this study, nine variables were used, each being a single puzzle piece that it was hypothesized would reveal the most meaningful data only when they were assessed together.

8.2.1 Body Mass Index

The skeletal variants of clinical risk factors, body mass index and activity levels, were tested for any level of relationship with the joint conditions assessed during this research. Statistical testing revealed that BMI had no significant correlation or association with any of the studied joint conditions on its own, thus it would suggest that being overweight is not a significant factor for the development of the conditions. Body mass was found in the final predictive steps of regression testing for hip and knee osteoarthritis. However, clinical studies have shown that a positive relationship should exist between increased relative mass and prevalence of osteoarthritis on the weight beating joints, especially the knee (Felson and Chaisson, 1997; Stürmer *et al.*, 2000). Therefore, the lack of significant correlation or association found within the data of this research runs contrary to the clinical trends of the field.

One potential explanation for the lack of statistical correlation or association between the joint conditions and BMI could be the narrow range of BMI scores observed in the archaeological populations. The mean BMI found within this body of research was 24.1 kg/m² (normal mass) with the full range between 19.73 (normal mass) and 29.03 (over-mass) kg/m². Therefore, the range did not reach the obese BMI range (30.00-40.00 kg/m²). A clinical study conducted by Stürmer and colleagues (2000) had a higher mean BMI with knee osteoarthritis patients (29.4 kg/m²) with an OR of 5.92 for overweight and 8.13 for obese individuals and hip osteoarthritis patients (27.3 kg/m²) with an OR of 0.72 for both overweight and obese individuals, illustrating an increased risk for KOA to individuals in a higher BMI category than was found within this research. In a similar study conducted by Anandacoomarasamy and colleagues (2012), the average BMI was 39.6 kg/m², over 10 kg/m² higher than the largest BMI found within this body of research, with a range of ±5.2 kg/m², and at these BMI ranges a prevalence rate of 26% for knee osteoarthritis was evident as opposed to the 9.9% found within this body of research. The study by Anandacoomarasamy and colleagues (2012) also indicated that within the obesity BMI range the thickness of cartilage was decreased and by losing an average of 43kg each, effectively shifting to a lower BMI category, the participants of the study were able to reduce the impact that mass had on cartilage thickness. The lower mean and maximum BMI found within this study suggest that there were individuals who may have been overweight, but their mass did not reach a critical point to impact biomechanical load, leaving the risk of developing an arthritic condition lower than the clinical studies. This is consistent with the theory that it is not simply an increase of mass that caused increased risk, but the

increase of mass passing a critical threshold falling into the obese BMI range (Felson *et al.,* 1988; Lee and Kean, 2012). However, this does not appear to then be in line with the results found within the study of Holmberg and colleagues (2005) in which it was found that males displayed significance with the presence of OA at the lower BMI scores (<25 kg/m²).

According to research conducted by Stürmer and colleagues (2000) the odds ratio should increase or be positively associated for knee osteoarthritis (Adjusted OR = 2.63) as the BMI score increases by 5, but that HOA was not strongly associated with increased mass (Adjusted OR = 0.78). However, the odds found for hip and knee osteoarthritis differed within this body of research (OR = .74/ 1.24 respectively), almost reversing the ratios found by Stürmer and colleagues (2000). This could be due to differing levels of comparison, as the odds ratios found by Stürmer and colleagues (2000) were based on a larger range of BMI values that than was found within this body of research. The decrease of OR for hip osteoarthritis would further indicate that BMI does not affect the joint until a much higher score (30+) and the range found within this study was too small to have created an increase of risk. However, that would not explain the apparent reversal of ratios or explain why knee osteoarthritis would show a positive association for the data of this study using OR but not standard association testing.

A different explanation could be due to the difference of a live patient and a skeletal sample; with a skeletal sample there is no true control group with a manageable independent variable, just a comparison of groups differing by the few known variables. A final explanation could be due to the limitation of the method for calculating skeletal BME. Using a skeletal population, it is impossible, without the proper documentation, to know the mass of individual over the course of their life. An individual may gain or lose weight at will, either through dieting and exercise, or because of an illness and it would be unknown to the researcher if the weight shifted. Therefore, the method, as stated in Section 6.1.2.1, displayed high correlations with living weights of individuals as documented around the age of 18 and lower correlation with individuals of increased age (Ruff *et al.,* 1991) and would not be able to accurately portray the 'living mass' of an individual. This then could have skewed the results towards the lower BMI ranges and future research on this data will need to be reworked with the methods of Ruff and colleagues (2012) that was developed on European Holocene samples and has been further tested and found to be more accurate and reliable (Elliot *et al.,* 2016a; Elliot *et al.,* 2016b; Jeansen *et al.,* 2017). Weiss (2006) noted this on her study of a prehistoric Californian Amerind people, in which mass was correlated with age or sex, but not OA and proposed that it would

be difficult for anthropologists to accurately study this phenomenon without knowing the mass change over time.

8.2.2 Activity

Activity and mass can be inextricably linked when it comes to physical activities. Muscle force is directly linked to mass, as it can take 2kg of force to move a kg of mass (Frost, 1997), creating a new relative mass with greater force while in motion. The binomial logistic regression testing showed that, while not significant on its own, body mass index was a significant variable for the final step of the predictive equations for presence of hip and knee osteoarthritis. In both cases, three additional robusticity variables were included, though not the same ones between the conditions. This would indicate that the increased mass was related with said variables. Though the explanation of such a relationship may be that as mass is increased, the skeletal frame is forced to compensate, with only one of the variables being an actual cause of arthritic conditions. However, when further tested, the association was weak/ negative and after corrections, the associations were no longer significant, indicating that the combination of variables are the key to increasing significance.

8.2.1.1 Entheseal Changes

Entheseal changes displayed significant relationships with general and spinal OA and made a significant contribution to the binomial logistic regression equations of the same condition. However, in isolation, entheseal change can be a poor predictor of activity level or type, as it is strongly related to age (Villotte *et al.,* 2016; Henderson *et al.,* 2017; Palmer and Rist, 2019). It is not accurate to simply say that increased activity is a major factor of joint conditions, as levels of activity are relative and increased activity may only last for a limited period of time; however, a regular, prolonged high activity level may impact the development of joint conditions (Lequesne *et al.,* 1997). Also, the ground surface on which the activity takes place (i.e., soft ground v hard ground), may play a part and cause varying ground force reaction (Benjamin *et al.,* 2006). For example, a casual runner may not be at increased risk, while a competitive runner could be, with a further increase to the risk if the runner is on a hard surface or other surfaces with minimal elasticity. Therefore, it may be possible to conclude that individuals taking part in high-risk activities, and who do not yet show signs of arthritic

conditions, may not have participated in the activity long enough to reach the point of critical wear on the joint (Johnsen *et al.,* 2016). The length of time an individual participated in habitual activities is difficult to determine in skeletal remains and not accurate beyond a short/long term period. However, there are many variables that would need to be considered and the activity itself may not be a cause but another underlying issue (Callahan and Ambrose, 2014; Peeters *et al.,* 2015). It is also important to note that those individuals that do present with symptoms of arthritic conditions and partake in high-risk activities, could be due to the result of an activity induced stress injury.

Historically it has been believed that there should be a marked difference between geographical regions (mountains v. flat land, forest v. cleared land, etc.) (Bjelle, 1981; Carballo-Pérez *et al.,* 2021) and by occupation types (Louyot and Savin, 1966; Craft *et al.,* 1992; Meinzer *et al.,* 2019). The data from this body of research did not strongly conform with this theory, however there was a noted difference between the urban and rural groupings and a weak correlation/association, with the rural population showing increased entheseal changes. While further testing is warranted to extend the sample size and test parameters, the preliminary assessment would lend credence to the more modern and believed theory that these changes would more likely be due to the difference of the regular activities between the sites (Palmer and Waters-Rist, 2019; Refai, 2019; Lafranchi *et al.,* 2020), similar to the differences found by Carballo-Pérez and colleagues (2021). Carballo-Pérez and colleagues (2021) studied an isolated populated on the Canary Islands (200 – 1500 CE) and found differences regarding the development of entheses and CSG properties between the groups of the east and west, most likely caused by the differing daily activities. Each group showed distinctive differences around the muscle groupings that were primarily for heavy load (vastus medialis and adductor muscles) and sedentary lifestyles associated with sustained squatting (gluteus maximus) (Carballo-Pérez *et al.,* 2021). These findings were also noticed by Palmer and colleagues (2016), however, they also observed low correlations between OA and EC in their population sample, similar to what was found within this thesis. This illustrates a potential future avenue for research focusing on specific muscles and the urban and rural groups.

The answer as to the lack of correlations between OA and EC may be found in a recently published article by Alves-Cardosa and Assis (2021), in which they argue that by focusing on the population with the overarching patterns, much the way the research within this thesis does, may instead conceal occupation specific joint use. While this concept may not be entirely

possible on an archaeological sample, the concept and theory would be worth exploring within future research. However, the problem with this is that it requires a known occupation and activity record of the individual, which is simply may not be possible, or reliable, in archaeological populations (Henderson *et al.,* 2013a).

8.2.1.2 Cross-Sectional Indices

Femoral subtrochanteric/midshaft robusticity and area did not show significant relationships with BMI, but according to the study of young women, aged 18 to 40, by Pomeroy and colleagues (2017) body size should show a positive correlation with the cross-sectional properties of the long bones. Figure 8.10 - Figure 8.13 demonstrate the unpredictability found within this body of research when body mass and FSR/A and FMR/A are compared. The R^2 values have been determined and separated by biological sex and are low, demonstrating how extremely little variance is being explained. These discrepancies could be created by an inherent variation when using the calculations to estimate mass and robusticity/area, as opposed to taking measurable figures from living individuals.

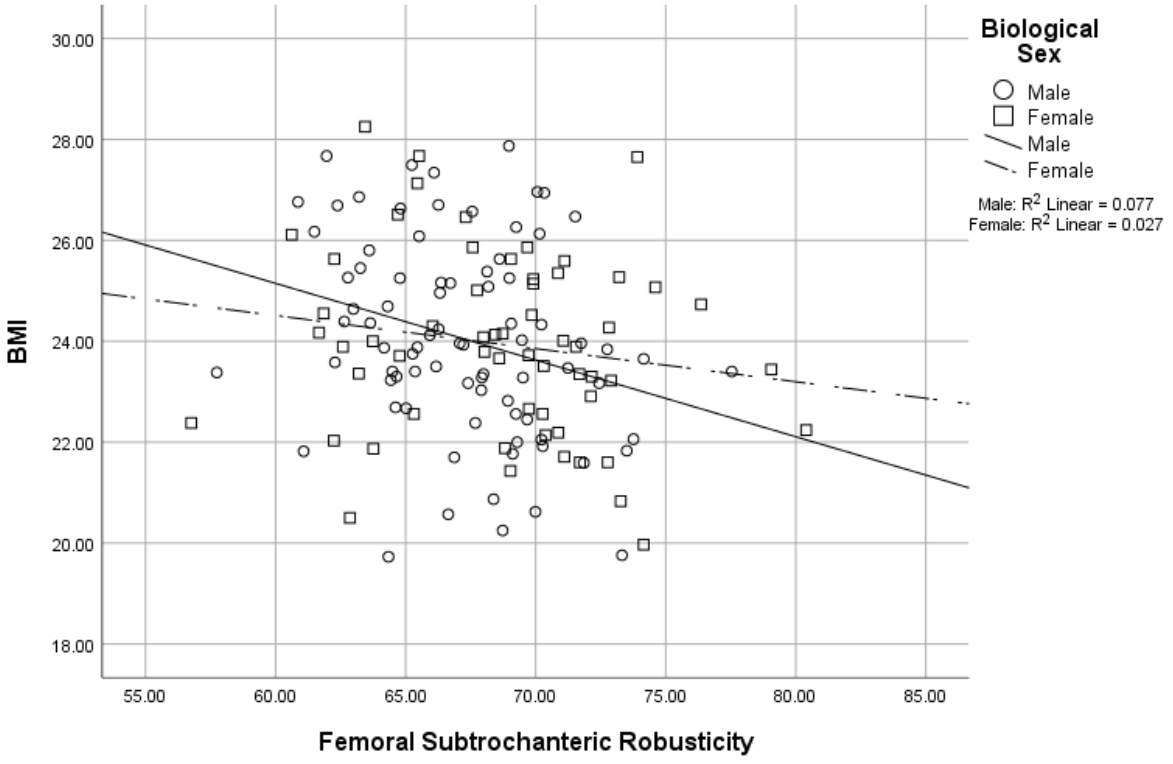

Figure 8.10 Distribution of the sample population by body mass and femoral subtrochanteric robusticity. The samples have further been identified by biological sex with R^2 values.

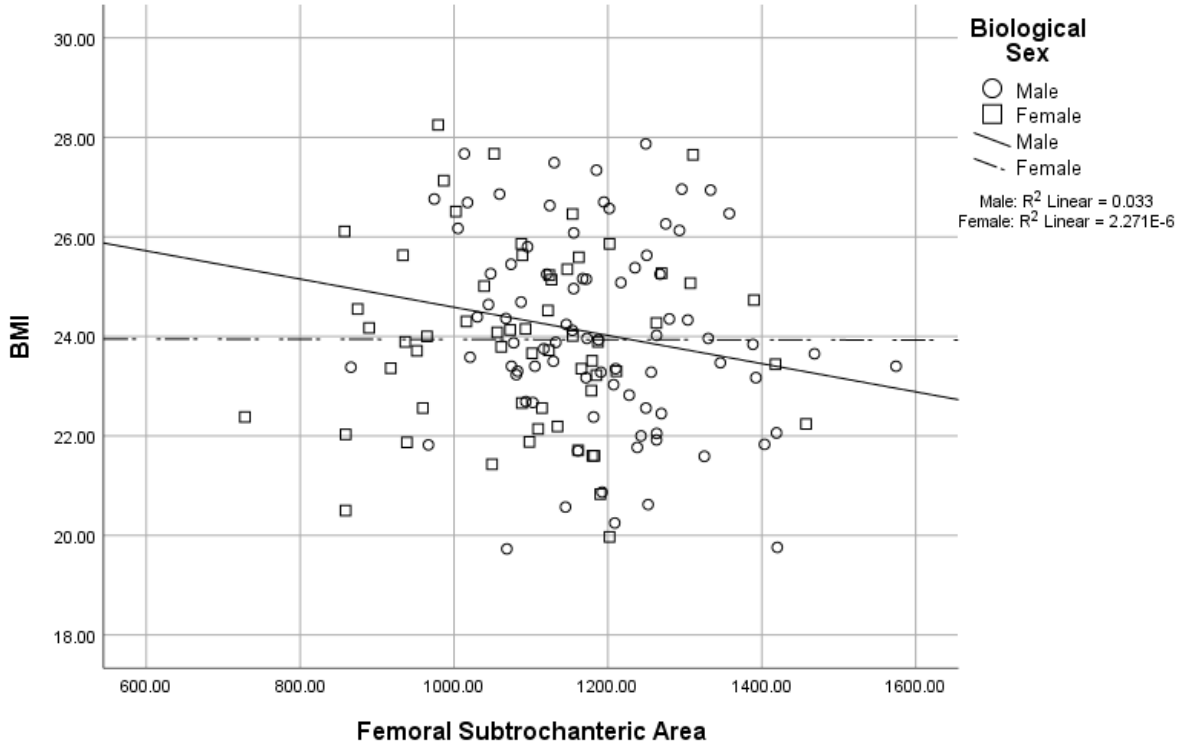

Figure 8.11 Distribution of the sample population by body mass and femoral subtrochanteric area. The samples have further been identified by biological sex with R^2 values.

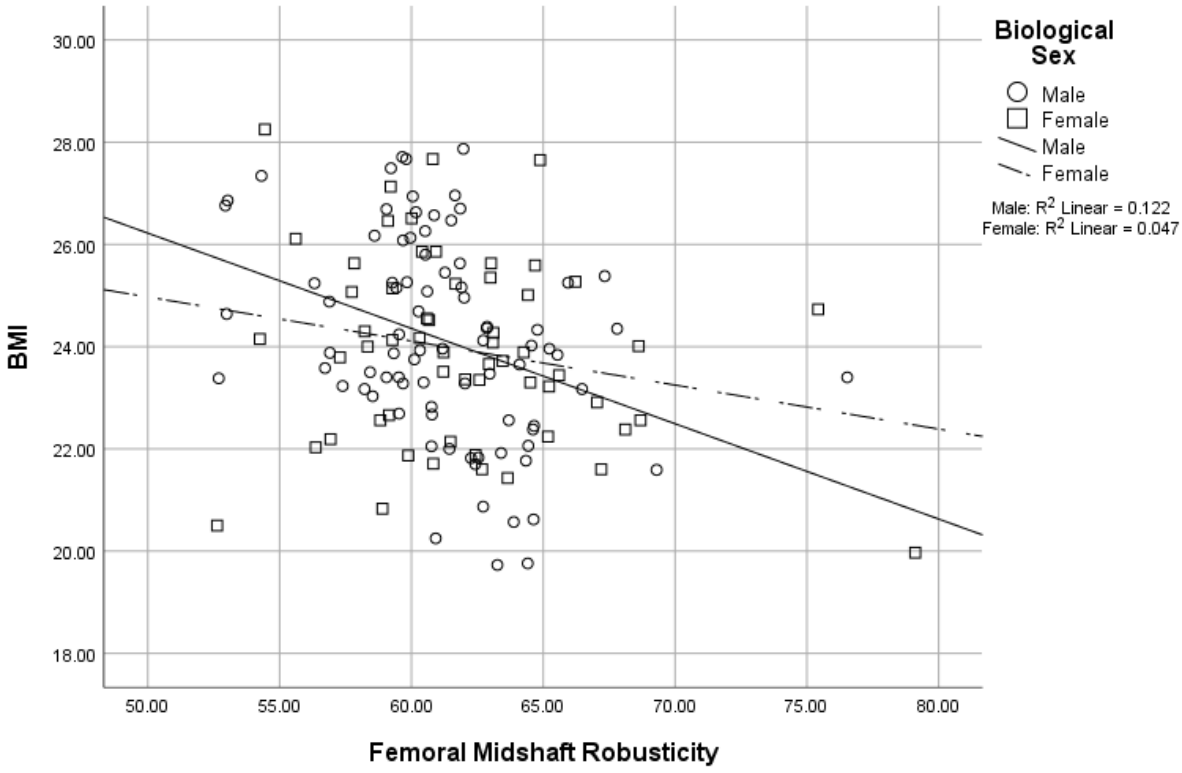

Figure 8.12 Distribution of the sample population by body mass and femoral midshaft robusticity. The samples have further been identified by biological sex with R^2 values.

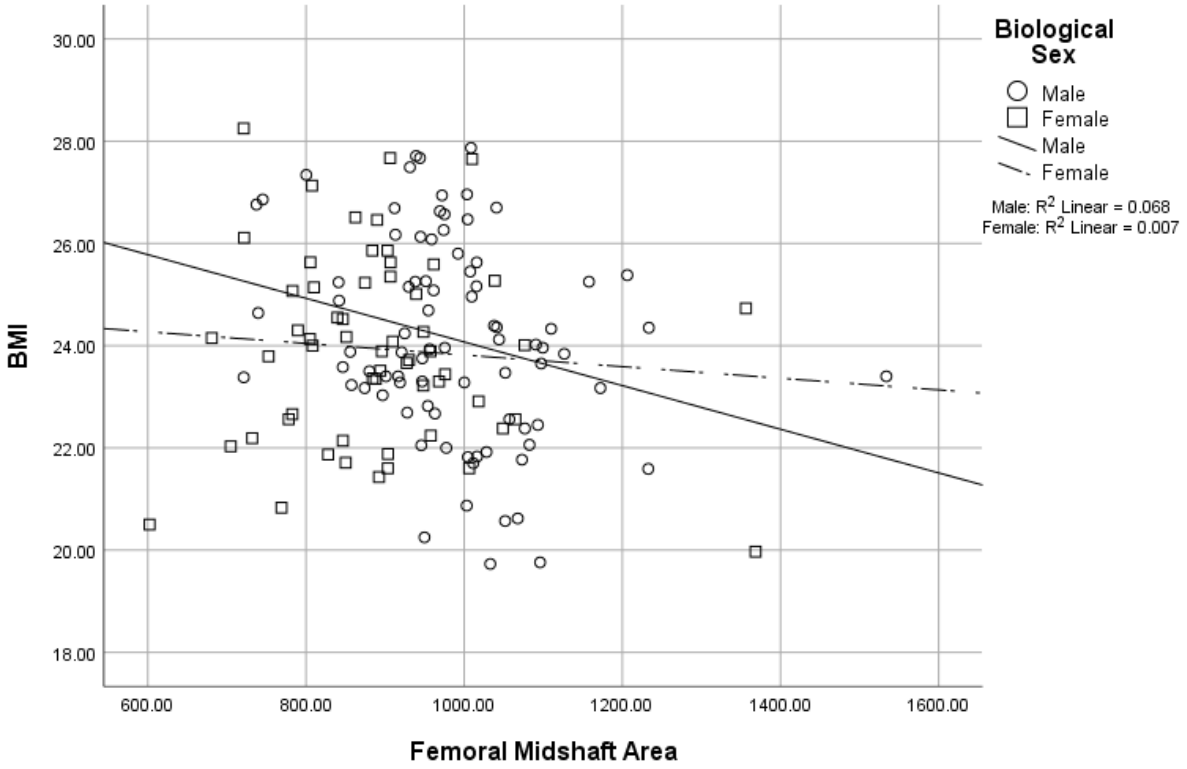

Figure 8.13 Distribution of the sample population by body mass and femoral midshaft area. The samples have further been identified by biological sex with R^2 values.

Attempting to determine activity levels via skeletal markers required the use of nine separate, but interrelated variables, rather than a single variable, making this exploration of this risk factor extraordinarily complex. Compared individually, only a few factors displayed significant correlations or associations with joint condition, suggesting that the individual variables would not support the ability to accurately predict or explain the presence of joint conditions. The activity variables themselves did not appear to correlate or associate with the other variables on a consistently, with all cross-sectional indices displaying strong inter-relationships, except for shape.

The cross-sectional indices do not, in themselves, denote activity. However, the combination of the variables can give an indication as to how the body can react and cope with habitual activity levels. Shape determines the directionality of the torsional forces with >1.0 indicating an antero-posterior direction and <1.0 indicating a medio-lateral direction (Westcott and Zephro, 2016). This would explain why shape displayed no significant relationships with the other variables of the cross-sectional indices, as torsional directionality does not correspond with power or strength. The cross-sectional area and robusticity deal with the relation of overall body size with size of the diaphysis. Robusticity deals with the strength of the bone and its

ability to react to torsional effects. Polar second moment of area is similar to robusticity but deals instead with the level of deflection of the torsional effects. Therefore, the relationships found between the non-shape variables are more understandable, as the different factors can directly affect one another.

Males and females showed significant differences for half of the indices (FSR, FMA, FSJ and FSA), potentially indicating a degree of activity based sexual dimorphism however, this theory would need further testing to validate or refute. As will be displayed in the forthcoming sections, sexual dimorphism concerning historical lifestyles has been studied and with clinical trials has been explored in more depth regarding sport. More common occupational activities that could help to simulate historical activities would be a useful tool for further studies in this area. Moreover, this is not distinctive of the post-medieval period and has been noted in populations across the globe and throughout history (Cameron and Pfeiffer, 2014; Saers *et al.,* 2017; Miller *et al.,* 2018). The lack of significant difference between site categories would indicate that the activities between rural and urban sites causing the same overall levels of stress to the femur.

Comparing cross-sectional values between studies can be difficult if the values are not produced and presented in similar fashions. This limitation was a primary factor in the selection of comparative sites and resulted in a more varied range of comparators than would be ideal. Nevertheless, the use of methodologically comparable data from diverse locations and periods offers the most useful means of contextualizing the data generated here. In the present study, males, overall, had the larger total area (TA) at the midshaft and the greater polar SMA (J) indicating an increased size and ability to deflect torsional forces. This then differs between the site categories, with males displaying higher values in the rural categories and females the urban categories. These differences are also reflected in two other populations in both South Africa and Tibanica, Columbia (see Table 8.9). This demonstrates how the differing lifestyles and occupational types affected the people and the skeletal structures. These populations were chosen due to the availability of data for the TA within the literature, whereas other studies may discuss cortical or medullary area and this issue will be further discussed below. The South African population represents late stone populations of varying subsistence types from differing geographical regions (Fynbos, forested and inland) (Cameron and Pfeiffer, 2014) and the Columbian population represents a native pre-Columbian population dating to around c. 1000-1400 CE (Miller *et al.,* 2018). While these populations may vastly differ in terms of

social, genetic, geographical, and historical profiles, the differing values help to illustrate the variability of these values between populations and by sex.

Table 8.9 Comparison of the cross-sectional geometric values at the femoral midshaft (50%) with sample size, mean value and standard deviation.

		MALES			FEMALES		
		n	MEAN	S.D.	n	MEAN	S.D.
TOTAL*	TA	86	984.24	120.99	60	893.17	134.65
	J	84	178.33	28.37	58	153.92	39.05
URBAN*	TA	47	960.71	128.92	31	1103.79	146.65
	J	45	172.61	30.54	29	232.58	52.56
RURAL*	TA	39	1012.61	105.44	30	898.84	164.02
	J	39	184.92	24.37	29	158.10	46.01
FYNBOS[1]	TA	18	922.90	119.40	9	872.80	95.40
	J	18	176.00	35.80	9	147.90	24.30
FOREST[1]	TA	12	922.90	46.80	8	920.70	117.10
	J	12	181.00	23.50	8	163.80	29.10
INLAND[1]	TA	3	947.70	57.40	12	887.10	81.00
	J	3	167.70	21.00	12	139.50	25.80
TIBANICA, COLUMBIA[2]	TA	31	883.90	94.30	32	825.60	64.20
	J	31	398.10	76.30	32	256.30	56.50

TA = total area (FMA). J = polar second moment of area (FMJ). * - McAfee Thesis. [1] - Cameron and Pfeiffer, 2014. [2] - Miller *et al.,* 2018.

A study conducted by Saers and colleagues (2017) of pre-industrial Dutch populations, not included in Table 8.9 as the data was not presented in a comparable format, found there to be no significant difference in cross-sectional geometric changes in the femur or tibia between various Dutch sites, however, the shape of the tibia differed, indicating a change in habitual mobility patterns. The lack of variation between the femora of the different site categories is similar to what was found within this body of research and was explained as the result of omni-directional loading affecting the femora in a much more complex pattern than previously expected. A different study conducted by Calce and colleagues (2018) studying a modern human population from Lisbon and Sassari (urban populations) found that the forces causing both CSG and OA do not function in the same way, with CSG properties being influenced by factors during childhood and young adulthood with the morphology not changing after, that would be well before the changes to the joint space of OA. These early developments of CSG factors found by Calce and colleagues, could then have formed a sort of insulation or protective barrier, which could then explain the lack of statistical correlation with the joint conditions.

There is a problem with using the calculations to create an approximation for the area of the diaphysis. When using traditional forms of cross-sectional geometry, researchers will be able to differentiate and use three different area variables: total (TA), cortical (CA) and medullary (MA) (Maggiano *et al.,* 2008; Pomeroy *et al.,* 2017). The method of Westcott and Zephro (2016) can only provide a total area, and to measure cortical or medullary area a researcher would either need to have access to MRI or CT technology or conduct an invasive and damaging cross section of the relevant bone (Pomeroy *et al.,* 2017; Saers *et al.,* 2017; Miller *et al.,* 2018). These missing data sets are a limitation of this thesis, particularly for comparing this study to other studies, however, the data do provide a good indication of bone properties.

Femoral subtrochanteric shape had a significant, positive relationship with spinal osteoarthritis, but no other significant relationships were found between the cross-sectional indices and joint conditions singly. However, the cross-sectional indices were found to be significant for the final step of the binomial logistic regression equations, further indicating that the cross-sectional indices should be considered together, rather than separately. With the exception of general osteoarthritis, the joint conditions had at least two of the variables of the cross-sectional indices in the final step of the regression equations. General osteoarthritis included just one of the variables but was paired with entheseal changes. The binomial logistic regression further demonstrated that the cross-sectional indices would have affected the joint conditions individually or in small groupings, rather than the eight variables working in concert. The exception being that the midshaft variables were only significant for hip and knee osteoarthritis. The different variables of the cross-sectional indices have been shown to be related to different levels and types of activity with the different levels and types causing more risk for osteoarthritis to certain joints than others (Hind *et al.*, 2012), however entheseal changes are still complicated and clouded requiring further testing (Henderson *et al.,* 2017; Palmer and Rist, 2019). Further testing with both living patients and skeletal remains of known individuals to create a database for comparison, will help to further the understanding of how these variables affect OA.

8.2.3 Conclusions

Age and the variables found to be related to age, displayed patterns explaining how each related the joint conditions to varying extents. Body mass may indeed play a role in the development of the conditions, but the samples used within this study did not have any individuals falling into the obese range to be able to conclusively make a determination on the matter. Further testing will be needed to be able to test this theory using individuals over 30kg/m², as well as using the latter method by Ruff and colleagues (2012) which may show different results.

Entheseal changes contributed to the prediction of both general and spinal osteoarthritis in the binomial regression. This would suggest that of the osteoarthritic conditions researched, general and spinal osteoarthritis, may be linked with the entheseal changes of the lower limb, although the development of entheseal changes might not be a direct result of the osteoarthritis itself, but a parallel development. Correlation does not equate to causation, and it is an interesting fact that osteoarthritis presenting in the spine showed a higher level of relationship with entheseal changes of the lower limb than osteoarthritis of the hip or knee. A possible explanation is that as both entheseal changes and the joint conditions are considered age related (Mazza, 2019) there is no cause-and-effect relationship, but the development was concurrent and independent, which supports the research of He and Almeida-Prado (2021). Spinal osteoarthritis on the lumbar vertebrae is known to be affected by load on the vertebral column and the increased activity that may have been responsible for the increased wear to the joint, causing the development of osteoarthritis may have also caused an increase in muscle development as the body compensated to the increased habitual workloads. An example of this can be seen with athletes who develop their muscles through an increase to load and habitual activity, which will cause a faster break down of the joint and an increase to the risk of developing osteoarthritis (Lequesne *et al.,* 1997). As a preventative and management treatment for knee osteoarthritis, Griffin and Guilak (2005) have suggested that moderate activity will reduce the effects that the increased mass has on osteoarthritis, which could further explain the lack of relationships the joint conditions and BMI as well as the cross-sectional indices. While this would fit with patterns described in the literature, it does not explain the lack of relationship within this data between age at death and entheseal changes.

There are three potential explanations as to why several of the clinically-identified risk factors for joint condition recorded in skeletal remains did not appear to present the expected relationships to skeletally-observed evidence of joint condition : (1) the skeletal variables used were not accurate or appropriate proxies for the clinical variants of the risk factors, (2) that the small sample size has caused a bias in the data, and/or (3) that, as seen with the examples of BMI in the beginning of this section, the risk factors did not fall far enough above or below the 'normal' range for osteoarthritic change to have occurred. If explanation (1) is true, then further research must be conducted to determine the appropriate variable or combination of variables that would be analogous to the clinical variations, with a larger sample size and potentially using more samples of individuals with known personal histories. If explanation (2) is true, then this research will need to be broadened to include more samples to test. Although seemingly straightforward, archaeological samples are limited in number and restricted by preservation. To increase the sample size substantially, the objectives of a follow-up study would need to be altered to include samples from different historical periods. If explanation (3) is true, then this would further indicate that a critical threshold would need to be met before the risk begins to take effect. In this scenario, clear ranges will need to be understood and set forth for each of the risk factors to include the ideal range with the lower and upper ranges established. Of these three scenarios, (3) seems like the most plausible explanation as the current literature states similar findings, however, it may be that it is a case of all three scenarios being correct. In any eventuality, the lack of correlation/association of the risk factors when assessed individually yet displaying significance when assessed as a group within the regression model, lends credence to the theory that the variables can only help to explain the joint conditions when discussed together, rather than in isolation.

8.3 Ankylosing Spondylitis and Sacroiliitis

Ankylosing spondylitis and sacroiliitis are spondyloarthropathies that can affect the joints of the appendicular skeleton, such as the hip and knee (Weisman, 2011). This link to the appendicular skeleton signifies that knowledge would be increased by combining research on osteoarthritis to the hip and knee with studies of ankylosing spondylitis. Progression of ankylosing spondylitis has been found to reduce quality of life causing a progressive functional impairment overtime affecting physical activities. This causes individuals to be incapable of sustaining the same levels of activity post-onset and may force individuals working in labour

intensive jobs to quit (Khan, 2009). Understanding how the effects that the condition has on the skeletal frame once onset occurs, as well as studying the joint conditions that affect similar joints will create better understanding as to the lifestyles of the individuals of the past.

Analysis of the data found with this research found only an extremely limited number of cases of ankylosing spondylitis and sacroiliitis. It has been noted that it can be difficult to accurately assess and draw further conclusions on small datasets, especially if the testing includes statistical analyses (Waldron, 2011). This makes finding appropriate sample sources difficult, as the researcher needs to consider preservation and completeness of remains, as well as well as the proportion of adult skeletons, when considering a skeletal collection. Without a sufficient sample size, the confidence interval increases, which makes inferential data analysis less reliable or accurate and potentially unable to support any reasonable conclusion or assumption made (Blaikie, 2018). Therefore, the study of seronegative spondyloarthropathies for a population-based study is possible only if the collection has a sufficiently large sample size, or an acceptable sample size can be achieved across multiple collections. This would make assessment of certain collections, where preservation of remains is too low to be able to develop any significant data based on useful numbers, unusable. In terms of the research within this body of work, the sample size was too small to create any useful assumptions or conclusions based on the data gathered.

Sample size is not the only challenge facing researchers of seronegative spondyloarthropathies and sacroiliitis. A second barrier encountered during this body of research was the inability to differentially diagnose ankylosing spondylitis during its early stages of development. This indicates further research is needed concerning the diagnosis of ankylosing spondylitis before the pathognomic or more prominent characteristics appear. At present, a probable diagnosis is the most definitive during latter stages of the condition, owing to the presence of the pathognomonic feature of the bilaterally fused spine. This leads to conflict with other conditions that may look similar and cause phytic activity and fusion (Martin-Dupont *et al.,* 2006). Reactive arthritis is remarkably similar to ankylosing spondylitis, except that the ankylosis is asymmetrical in the sacroiliac joint and intermittent in the vertebrae, which may not be noticeable if the skeletal elements are missing or damaged. However, before fusion occurs in ankylosing spondylitis, it would be safe to assume that there would also be reaching phytic formations during the early stages of the condition. This would mean that

misdiagnosis is not only possible, but probable given the diagnosing criteria and generally variable preservation in archaeological skeletal remains.

The difficulty in diagnosing is not an issue solely for palaeopathologists. Clinically there is no effective blood test for these conditions, hence the seronegative nomenclature, and this makes it difficult for clinicians to diagnose as well. MRI has become a key tool in diagnosing of ankylosing spondylitis (Weisman, 2011) and has been used to view the inflammation of soft tissue in a living individual. Unfortunately for palaeopathologists, the soft tissue may not be available during examination, making this method unusable, however, if the inflammation is severe enough, it may leave traces on the skeletal material in the form of ossified entheses (Ranganathan *et al.,* 2017). Therefore, further study concerning the impact of SnSpA on the entheses could be conducted to test whether or not this would be valid for use in the future.

The challenges with differentially diagnosing SnSpA and SI have also been documented in mummies. Amenhotep II, pharaoh of the 18th dynasty, Ramses II, and his son Merenptah, both pharaohs of the 19th Dynasty, all pharaohs of the New Kingdom (approximately 1550 – 1070 BCE), and are believed to have had ankylosing spondylitis based on palaeopathological radiographic assessment (Feldtkeller *et al.,* 2003). Yet, it is still debated whether Ramses II had ankylosing spondylitis or another of three possible diagnoses, the other two being diffuse idiopathic skeletal hyperostosis and spondylosis deformans. These conditions are decidedly different in aetiology, yet also similar in development and appearance and illustrate how the conditions can be misdiagnosed and confused.

To combat this issue, a joint effort needs to be undertaken between clinicians and palaeopathologists to track the change in the body over time. While diagnosis is difficult, studies have been conducted clinically to allow clinicians to help diagnose at early stages, but this does not necessarily include the study of the changes to the skeletal structures (Song *et al.,* 2007). By trying to ascertain patterns and rates at which the syndesmophytes ossify, it may be possible to better diagnose skeletal ankylosing spondylitis. The progression of the conditions is slow and may be difficult to note via radiographs in a single patient (Wendling *et al.,* 2018), but by tracking the development of the conditions in a longitudinal study using multiple patients, it may be possible to fill gaps in our current knowledge. It would then also be useful to study the conditions that display similar skeletal changes and aetiology to be able to create more accurate differential diagnoses. If this succeeds, it will open up an entire avenue of

research within palaeopathology as the conditions would be more discoverable than ever before. This understanding of the effects on the past individuals will allow their stories to live on through research.

Diagnosing ankylosing spondylitis and sacroiliitis are not simply important for the understanding of the pathological conditions, but also for the study of how people with potentially disabling ailments were treated within the community. Ankylosing spondylitis and SI are conditions that are not exclusive, found in pharaohs and common individuals alike, which illustrates that the knowledge of the condition was available but not that of the level of care (Feldtkeller *et al.,* 2003; Martin-Dupont *et al.,* 2006). Did the ancients know what the conditions actually were, and did they know what symptoms would development in an individual if left unchecked? We have evidence of the upper class and royalty through the pharaohs, but what of the burden that was left on the lower classes or individuals that did not have the power or support of an entire nation/community? The limitation, if any, of work or a drop in lifestyle by individuals with AS and SI is an avenue that should be explored to offer a wider interpretation on individuals of the past.

Due to the lack of cases observed in the present study, there was no consistent way to establish potential relationships between ankylosing spondylitis and sacroiliitis and the risk factors examined. Nevertheless, as ankylosing spondylitis and hip osteoarthritis are said to have a relationship, testing for this one relationship may have proved informative (Khan, 2002; Weisman, 2011). Overall, ankylosing spondylitis and sacroiliitis require research in the future regarding the early stages of their skeletal progression. Due to a lack of knowledge regarding these preliminary stages, the diagnoses are misrepresented in palaeopathological samples. The future of research concerning these conditions will depend on the collaboration between palaeopathologists and clinicians.

Chapter 9 Conclusion

The data produced by this research has delivered results that both contradicted and supported current literature of the clinical and palaeopathological fields. These results have been explained as thoroughly as possible, but the study has also revealed several key areas in which further research will be required to better understand and explain the evidence. To continue forward, the individual branches of palaeopathological and clinical research will need to meld their practices and research data in order to be most effective for all parties.

9.1 Summary of Research Findings

The data within this study has shown that prevalence of joint condition did not appeared to be affected when the factor of site category was introduced; however, surface trends did appear regarding biological sex and age at death. Males had the higher ratios for the prevalence of osteoarthritis at the spine (1.56) and hip (3.4), as well as general osteoarthritis (1.73), and females had the higher prevalence for osteoarthritis at the knee (1.13). however, biological sex was not found to have any significant correlations/associations with osteoarthritis at any joint location. The prevalence rates of joint condition increased as age increased, and there were significant correlations/associations between general and spinal osteoarthritis and age at death. In general, these results fell within the norms for the clinical and palaeopathological research (Waldron, 2009; Allen and Golightly, 2015), although the lack of significant relationships in some cases is most likely explained as an artifact of low sample size.

The analyses of body mass and joint condition did not display the results that were expected as seen in palaeopathological literature, yet the results did bolster current clinical research. The data of this research found that the range of body mass (19.73-29.03 kg/m^2) may have been too small to affect the prevalence of joint condition with the upper values being far lower than found in clinical studies. However, the data may help to validate the theory that simply being over mass (25.0-29.9 kg/m^2) does not cause joint degredation and the onset of arthritic conditions but being critically over mass in the obese range (30.0-40.0 kg/m^2) will need further testing with samples that fall into this range (Stürmer *et al.*, 2000; Anandacoomarasamy *et al.*, 2012).

Looking at the cross-sectional indices, no significant differences existed between the site categories for diagnoses of joint conditions, even when assessed by homogenous biological sex groups. However, a sexual dimorphism which could be related to activity, or could simply be standard biological dimorphism, was found between males and females. While these differences were discovered, the individual variables of the cross-sectional indices appear to have significantly contributed to the diagnoses of osteoarthritis at each joint location differently. This suggests that the muscles used in the activities that help the skeleton to develop the properties illustrated within the cross-sectional indices are related to the development of the joint conditions. As entheseal changes only displayed significant relationships with general and spinal osteoarthritis, this could further indicate that the entheseal changes on these skeletal elements are an effect of the osteoarthritis rather than a cause.

The work on seronegative spondyloarthropathies will need to be considered ongoing, as it was unable to be completed properly due to a very small sample size, however, important issues have been highlighted that must be addressed within the field. With the relationships between ankylosing spondylitis and sacroiliitis and osteoarthritis of the hip and knee, as well as joints of the upper appendicular skeleton, illustrate the need for these conditions to be assessed and studied together, rather than focused on separately (Khan, 2009; Weisman 2011). The importance of such research has been emphasized as well as proposed ideas on how to rectify this issue.

9.2 Proposed Future Research

While the research questions for this research were answered, the research also indicated new avenues of investigation that must be explored. As has been stated throughout this thesis, palaeopathologists and clinicians must marry their knowledge to the benefit of both schools of thought. Further longitudinal clinical studies exploring the onset and progression of the conditions researched as well as taking detailed physical examinations of the musculoskeletal structure and follow up survey information, both fields would greatly benefit by the addition of data that could not be otherwise obtained. A longitudinal study using imaging technology would provide a clinical ‘flip book’ of images showing the gradual progression of the conditions over time and using CT and MRI techniques would allow for metric measurements of the skeletal material to be recorded. Research of this type could create data

that would also help to understand the effects on living patients as well as focus on the individual and track changes in activity, lifestyle, diet, and all other aspects that could affect the progression of joint conditions. Longitudinal studies are not new to this field, but with each new level of understanding, the older studies are simply outdated, and new data is required. This type of research would be an invaluable asset for both communities and allow for many assumptions to be made regarding palaeopathological materials that are not possible at the moment.

This longitudinal approach would be extremely useful also for the spondyloarthropathies, as little is known about the progression of the conditions. Without full fusion or late-stage features, it is difficult to diagnose clinically and even harder to diagnose paleopathologically. Understanding the development of the ossification of the syndesmophytes and associates enthesophytes would be a huge advancement towards providing diagnoses in skeletal material without full ankylosis. As seen within this thesis, specific spondyloarthropathies are being underreported and redesignated simply as undifferentiated spondyloarthropathies when the late-stage skeletal evidence is not present, making this research not only incredibly helpful to the field, but necessary.

At the start of this research, a standardized scoring and recording guide for joint condition was proposed as a continuation of a method devised for a completed MSc dissertation. However, due to a lack of inter-observer testing, this was a facet of the research that was unable to be completed. A standardized system would simplify the process of sharing data between researchers as well as potentially eliminate confusion from matching or understanding different systems. Combined with a database that could be accessed by researchers across the globe, shared knowledge, and the progression of understanding of joint conditions would never be easier. This database could be accessed via professional or academic channels, such as with Digitized Diseases, IsoArchH or the Global History of Health Project, and curated by a group of individuals which would restrict and inhibit the ability for individuals to tamper with databased information at will. After global episodes of pandemic, such as the Covid-19 outbreak of 2020, a database like this would make it possible to continue research while in isolation or in lockdown and from anywhere in the world.

9.3 Final Thoughts

Joint conditions are ubiquitous and due to the relatively high prevalence found amongst humans, it is necessary to understand how they affect humanity, past and present. The high likelihood that many readers of this thesis will be affected by one or more of these conditions is reason enough for the need to study such conditions – unlike many pathological conditions, the experience of living with joint condition is something that is shared by the majority of any human population. Insight into how the conditions have shifted prevalence by joint, between sexes and by age groups through the ages as the trends in lifestyles and technology have changed may help us to understand and prevent the conditions in people living today. Studies and research, such as this one, provide insights into the cause of the conditions which, in theory, can help living patients make changes to help reduce the impact and progression of joint conditions.

It is important to remember that the people of the past are more than just values in a statistic and to acknowledge what each may have undergone while these conditions developed. Awareness of the impact that joint condition had on our ancestors will help researchers in understanding the conditions in living individuals. While it may be easy to forget the humanity of each skeleton, it is the duty of every osteoarcheologist to be able to accurately tell the stories of these people, and in a way, it keeps these individuals alive.

Research into joint conditions continues to advance as new palaeopathological methods are established allowing for avenues of research, otherwise thought to be dead ends, or limited by current methodologies. This field is ever growing and evolving allowing for new research possibilities to be undertaken and objectives to be expanded. The study for this research project may now be concluded, but the research to resolve the many new questions and to continue to learn more about joint conditions will ever remain ongoing.

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Appendix

Cross-Sectional Indices

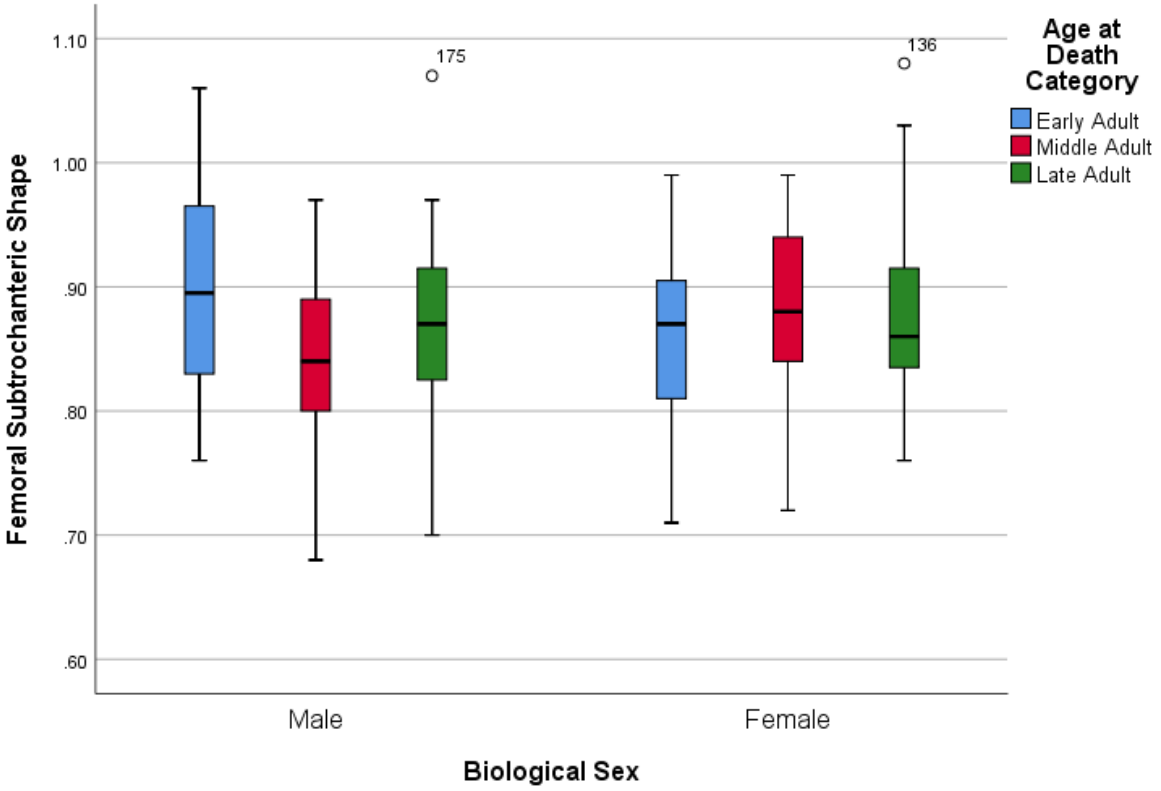

Figure 0.1 Boxplot of femoral subtrochanteric shape by biological sex and age at death, presenting the mean and interquartile ranges.

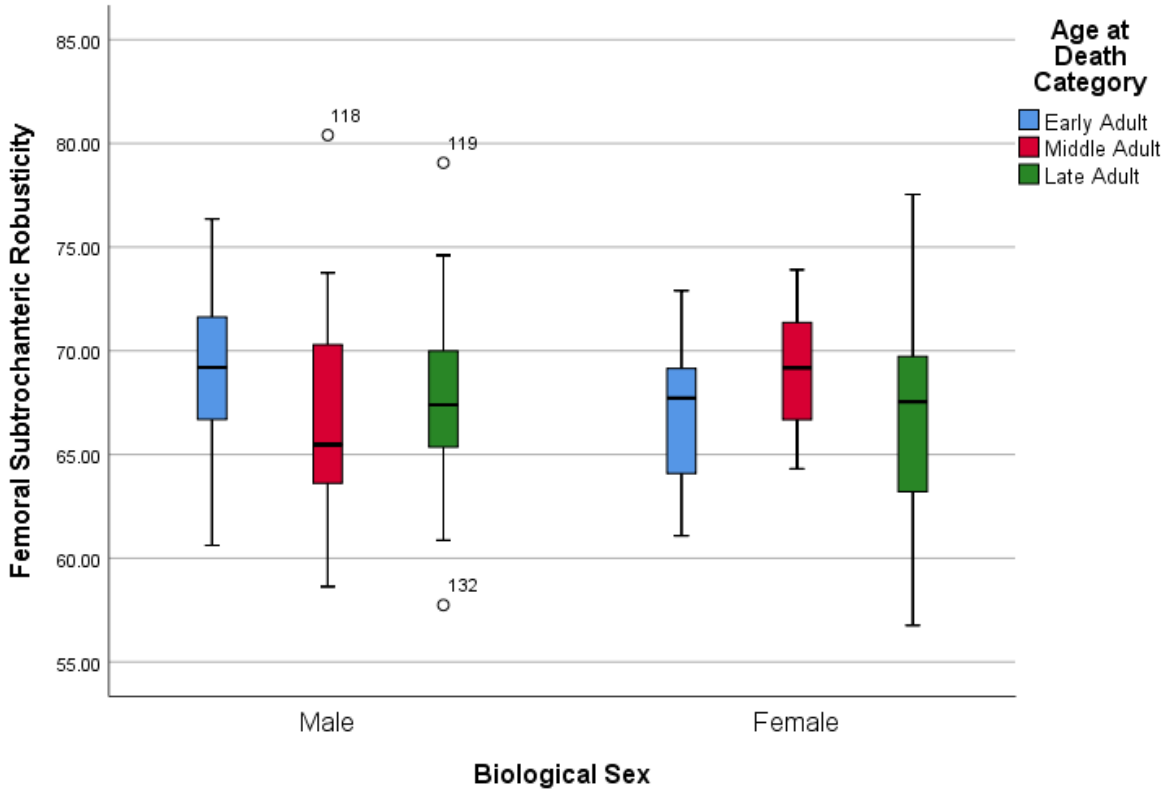

Figure 0.2 Boxplot of femoral subtrochanteric robusticity by biological sex and age at death.

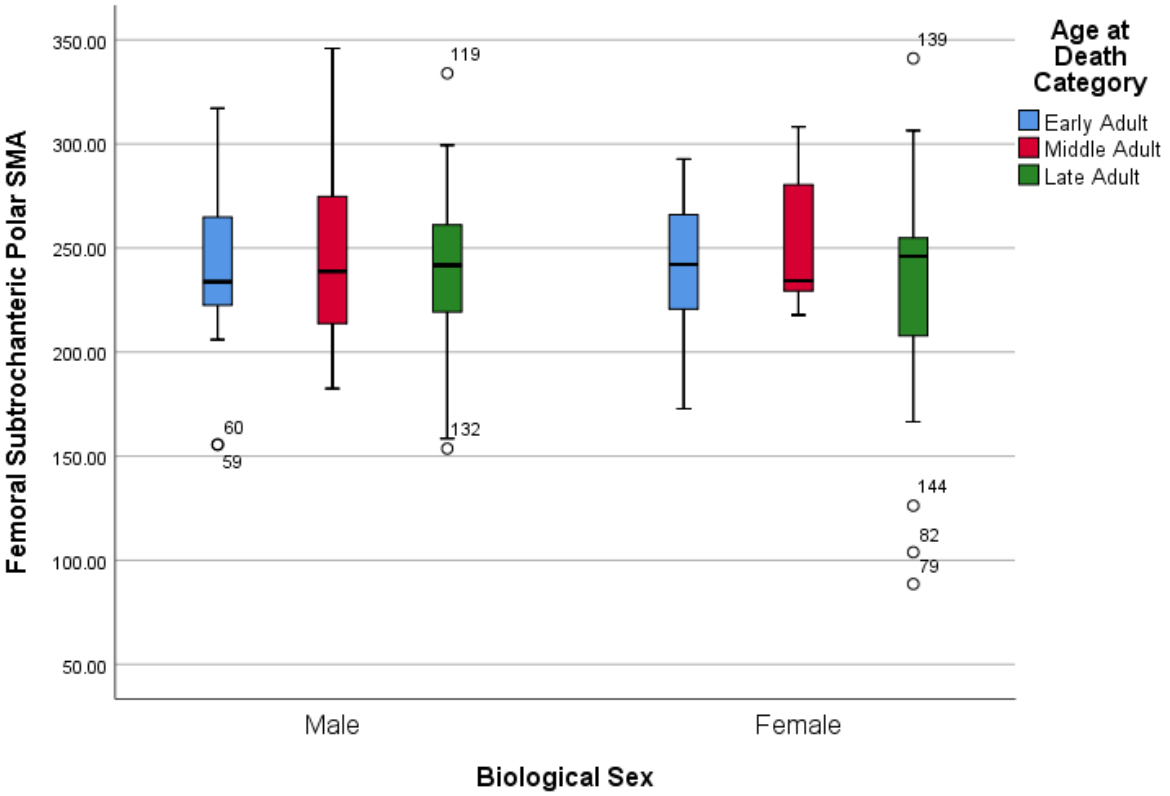

Figure 0.3 Boxplot of femoral subtrochanteric polar SMA by biological sex and age at death.

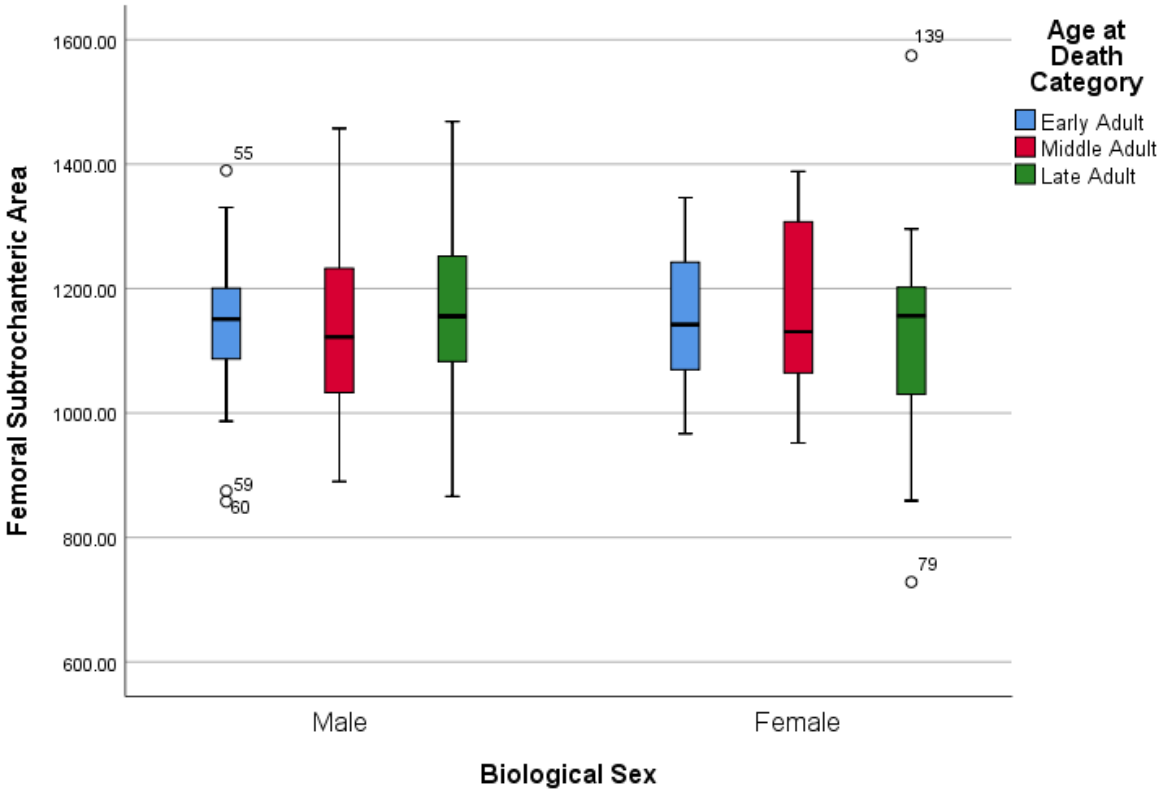

Figure 0.4 Boxplot of femoral subtrochanteric area by biological sex and age at death.

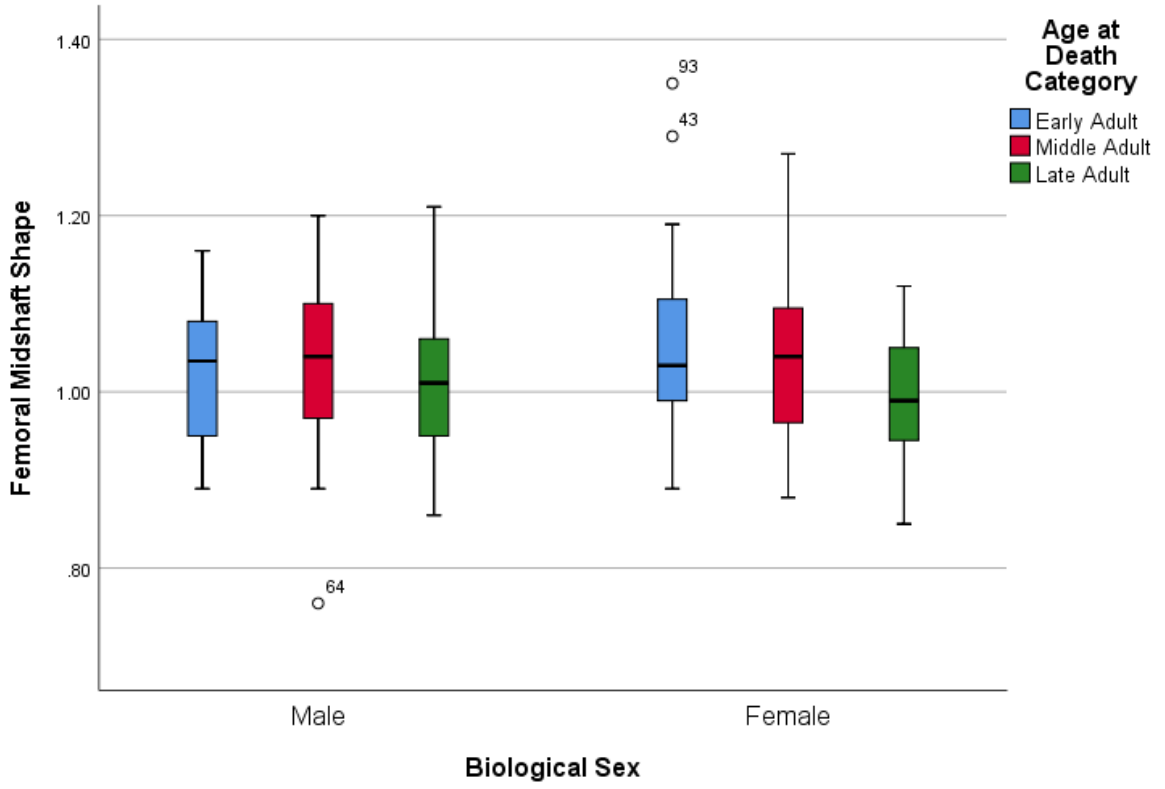

Figure 0.5 Boxplot of femoral midshaft shape by biological sex and age at death.

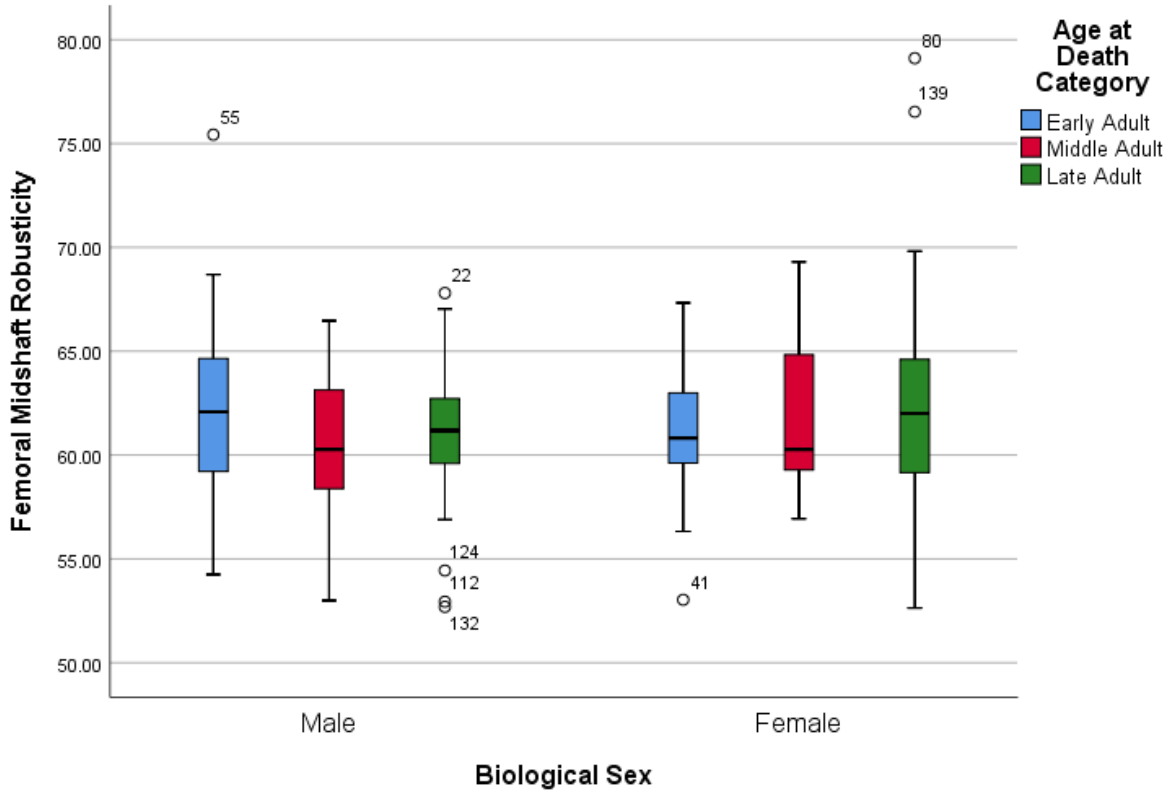

Figure 0.6 Boxplot of femoral midshaft robusticity by biological sex and age at death.

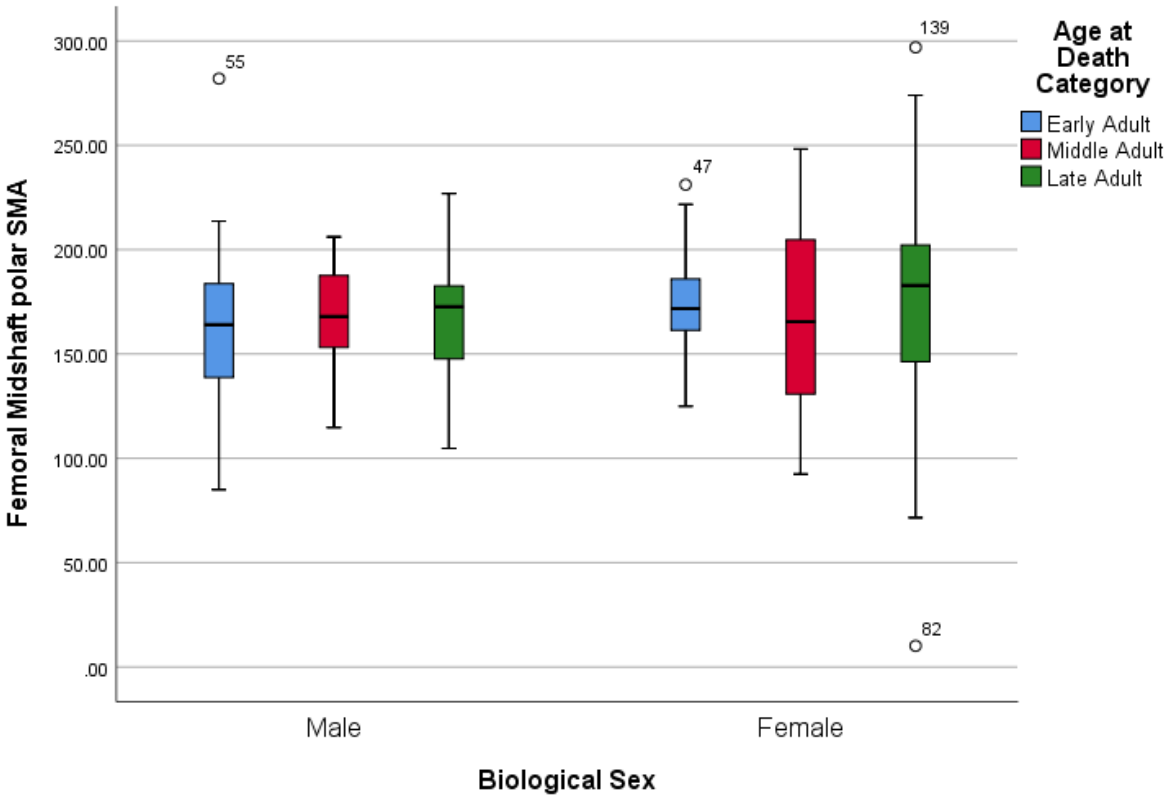

Figure 0.7 Boxplot of femoral midshaft polar SMA by biological sex and age at death.

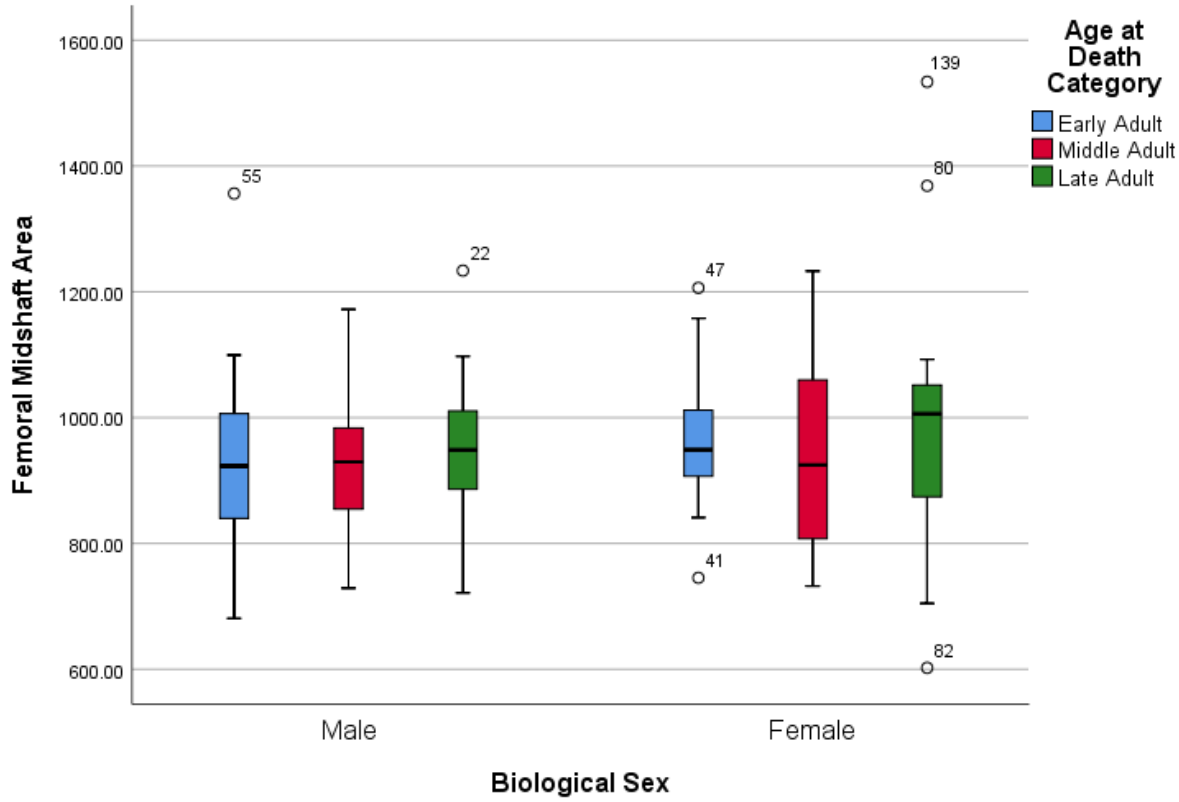

Figure 0.8 Boxplot of femoral midshaft area by biological sex and age at death.

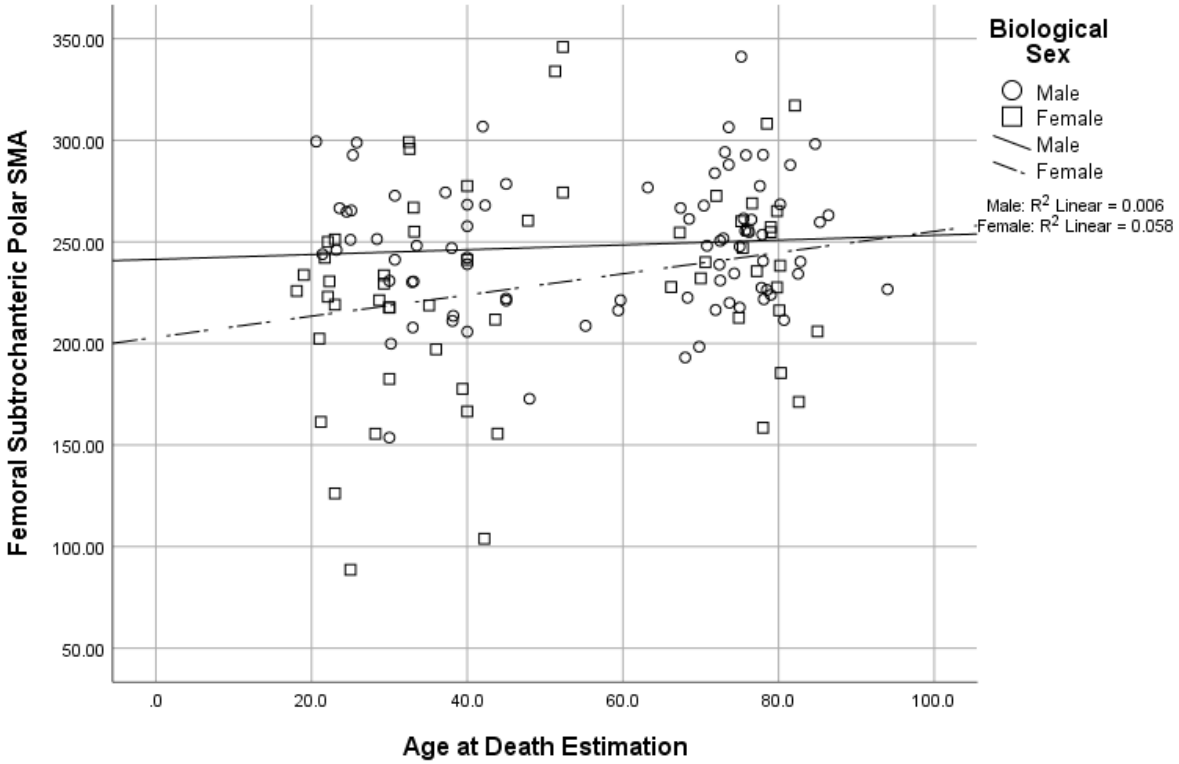

Figure 0.9 Distribution of the sample population by age at death and femoral subtrochanteric polar SMA. The samples have further been identified by biological sex with R^2 values.

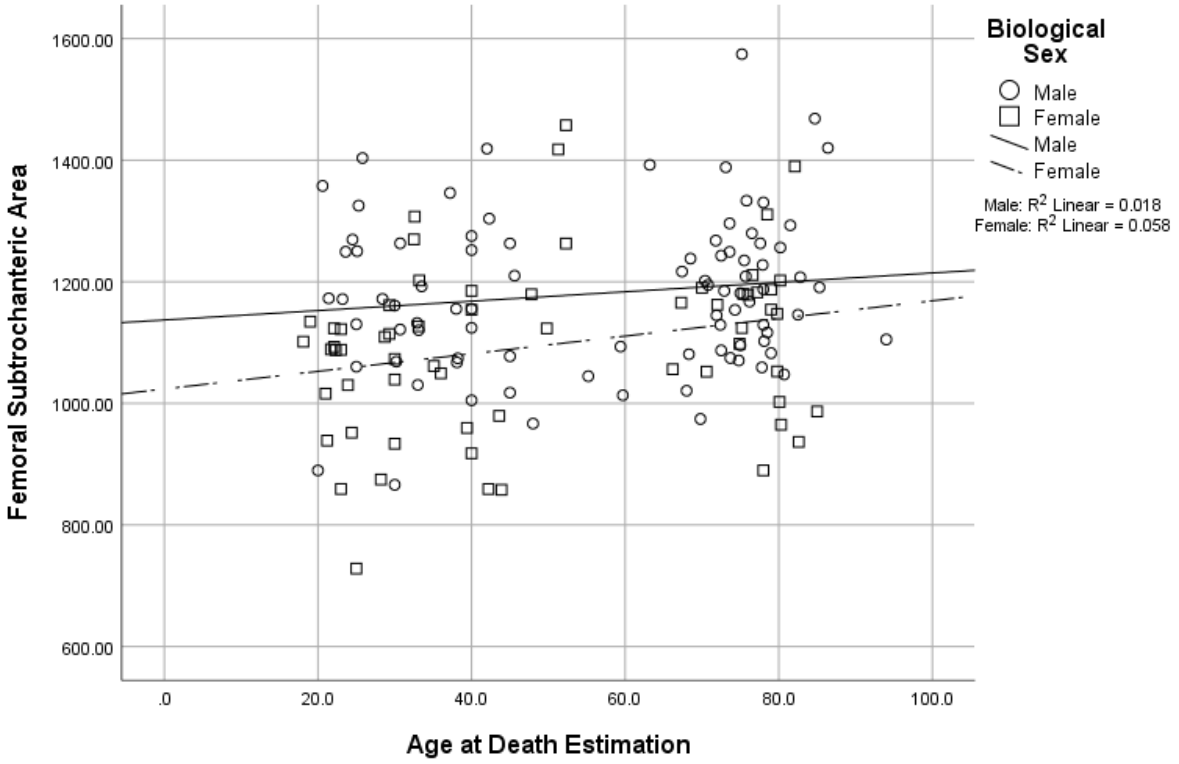

Figure 0.10 Distribution of the sample population by age at death and femoral subtrochanteric area. The samples have further been identified by biological sex with R^2 values.

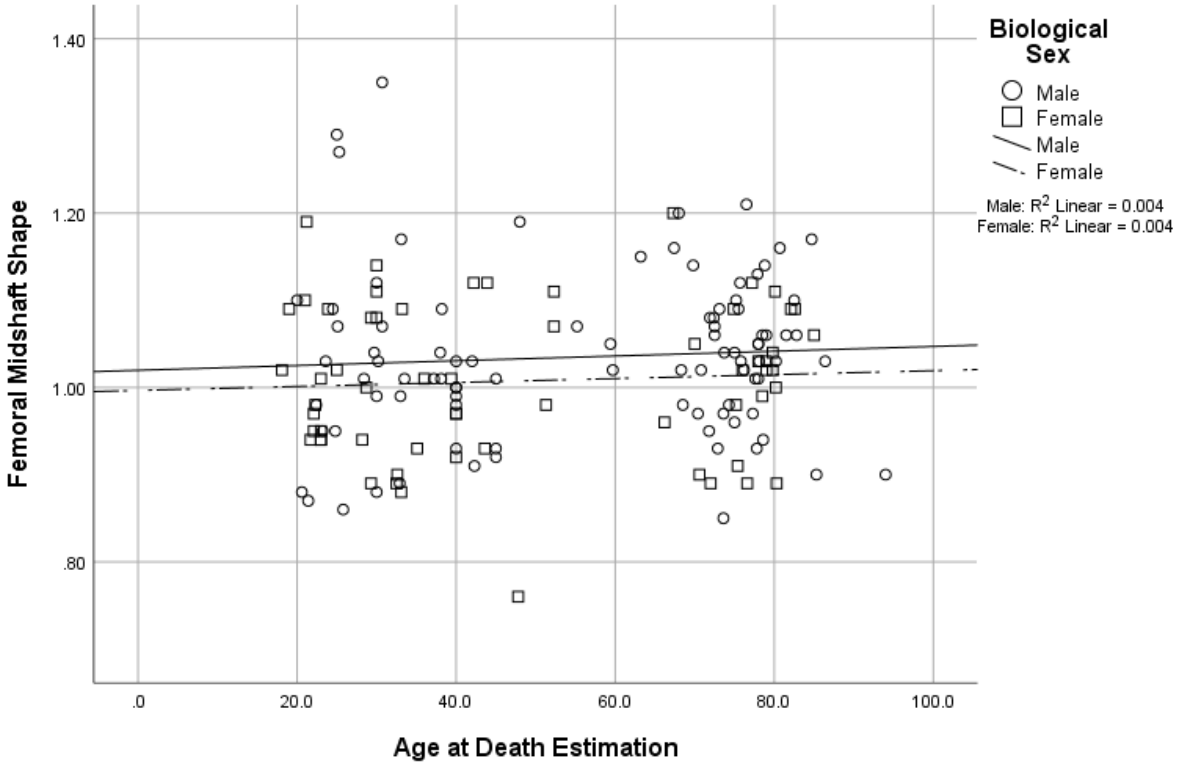

Figure 0.11 Distribution of the sample population by age at death and femoral midshaft shape. The samples have further been identified by biological sex with R^2 values.

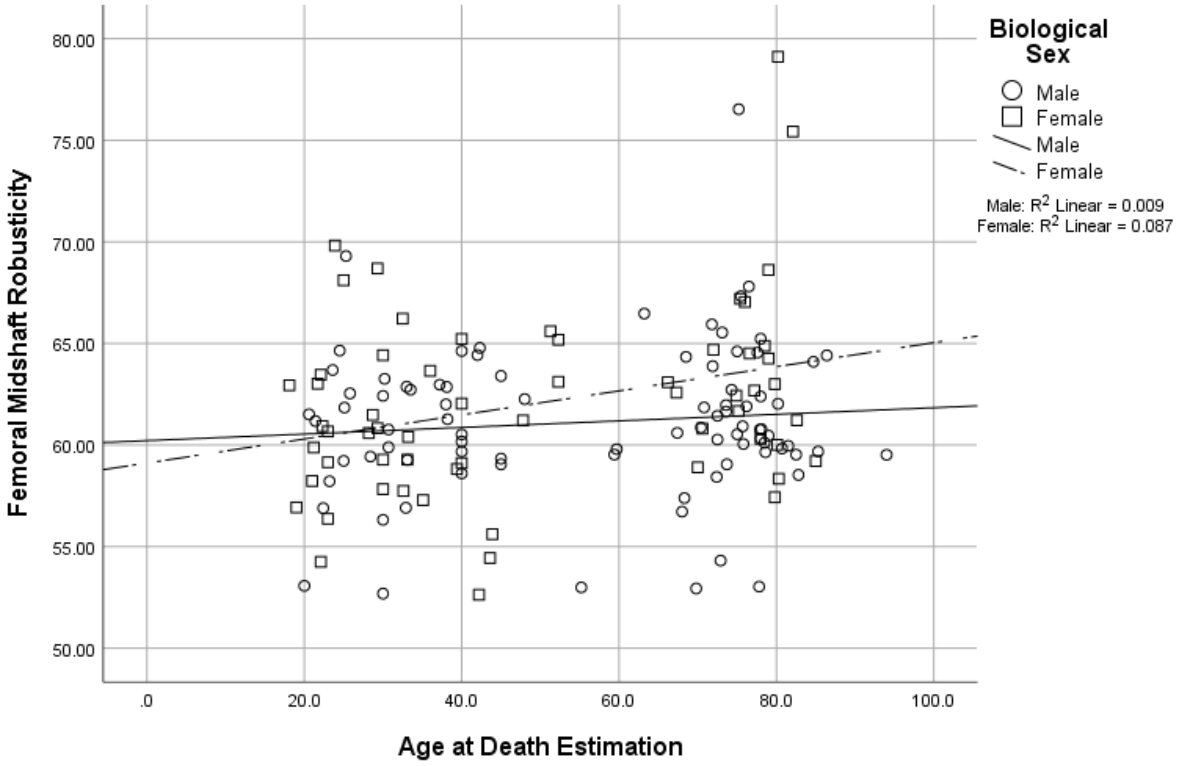

Figure 0.12 Distribution of the sample population by age at death and femoral midshaft robusticity. The samples have further been identified by biological sex with R^2 values.

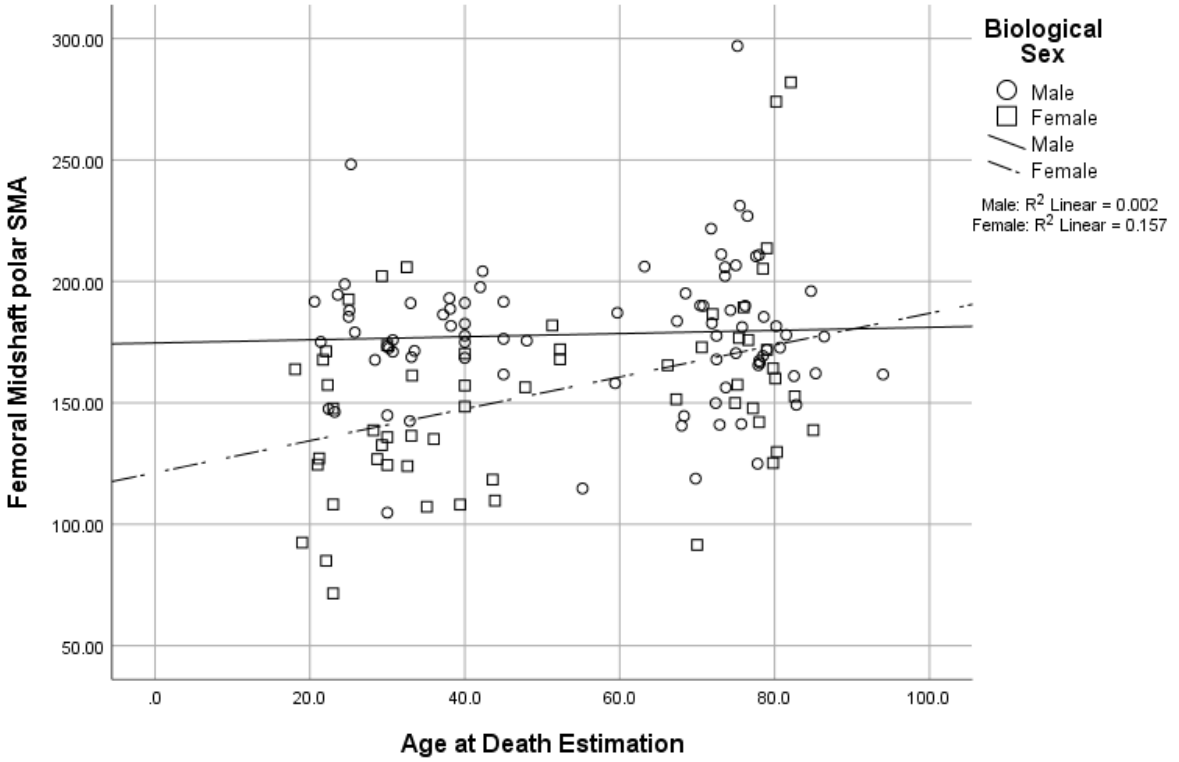

Figure 0.13 Distribution of the sample population by age at death and femoral midshaft polar SMA. The samples have further been identified by biological sex with R^2 values.

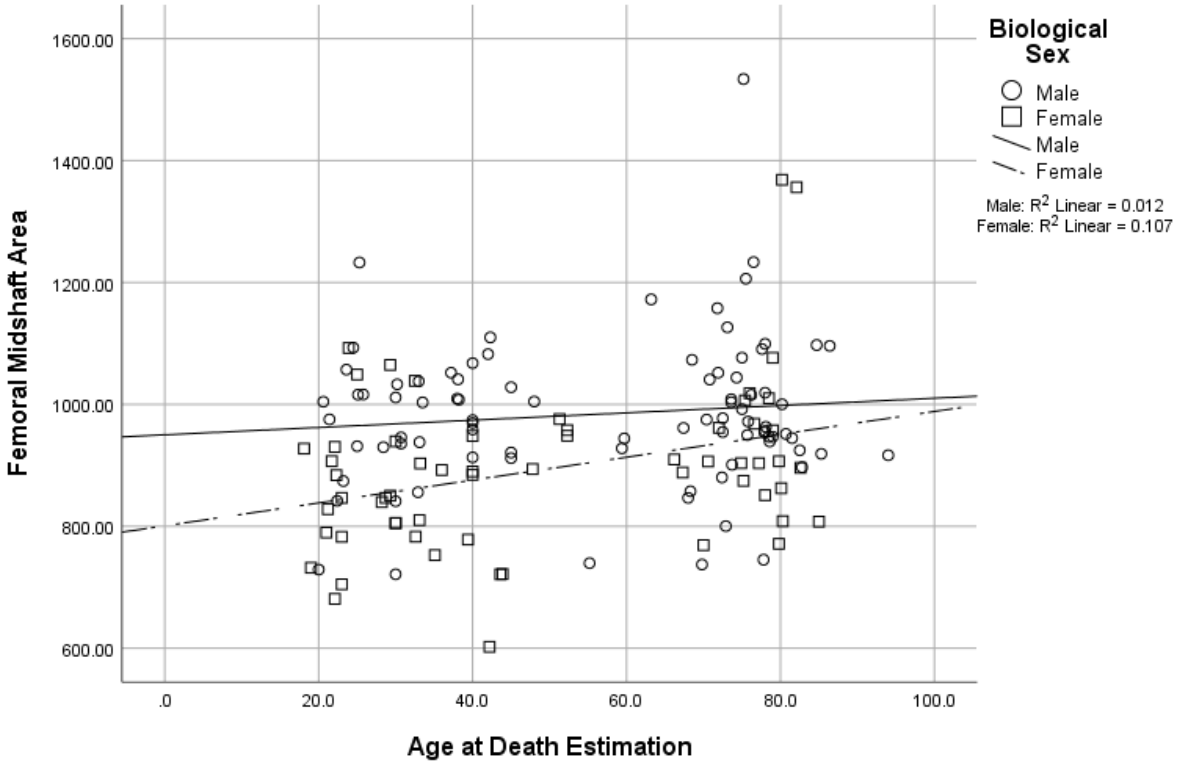

Figure 0.14 Distribution of the sample population by age at death and femoral midshaft area. The samples have further been identified by biological sex with R^2 values.

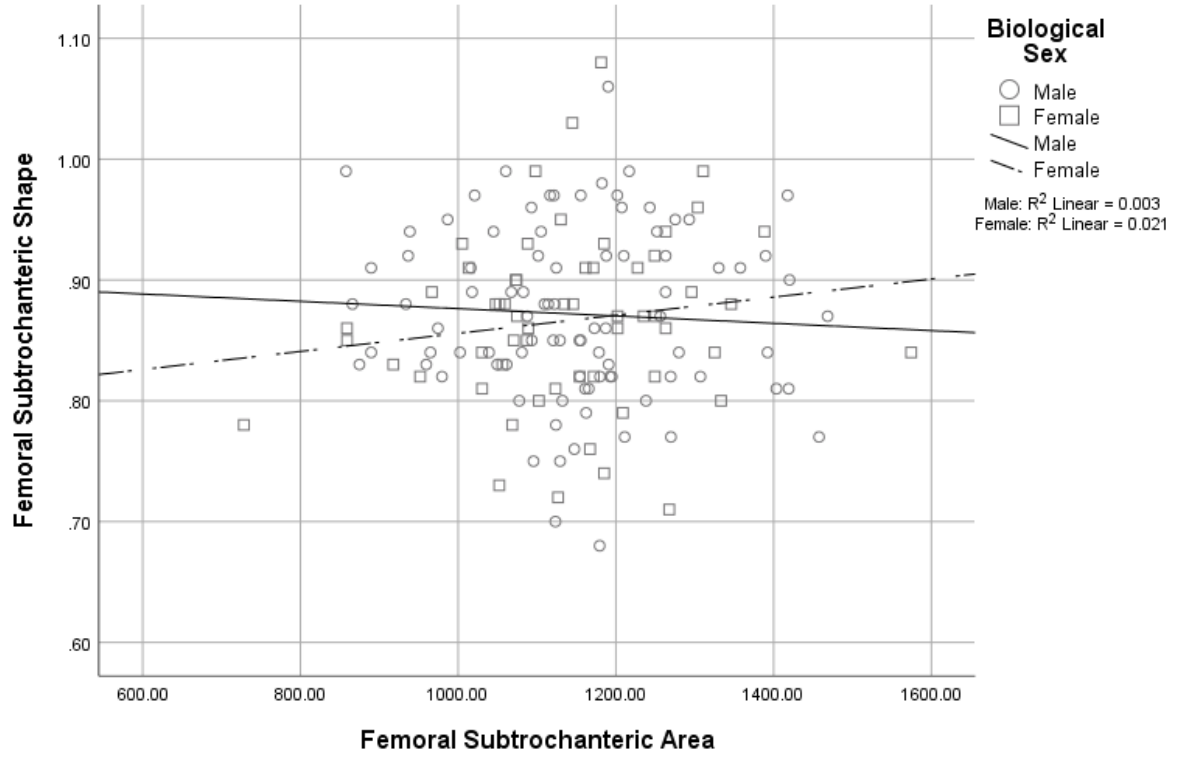

Figure 0.15 Distribution of the sample population by femoral subtrochanteric shape and femoral subtrochanteric area. The samples have further been identified by biological sex with R^2 values.

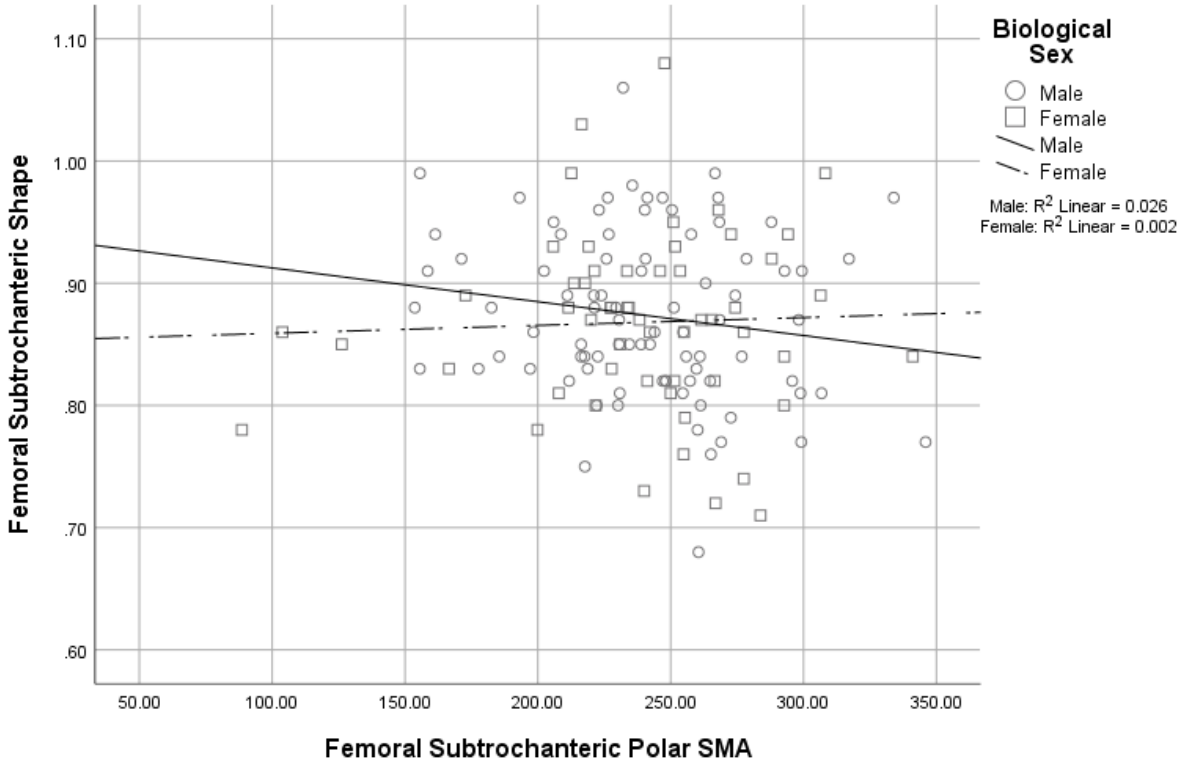

Figure 0.16 Distribution of the sample population by femoral subtrochanteric shape and femoral subtrochanteric polar SMA. The samples have further been identified by biological sex with R^2 values.

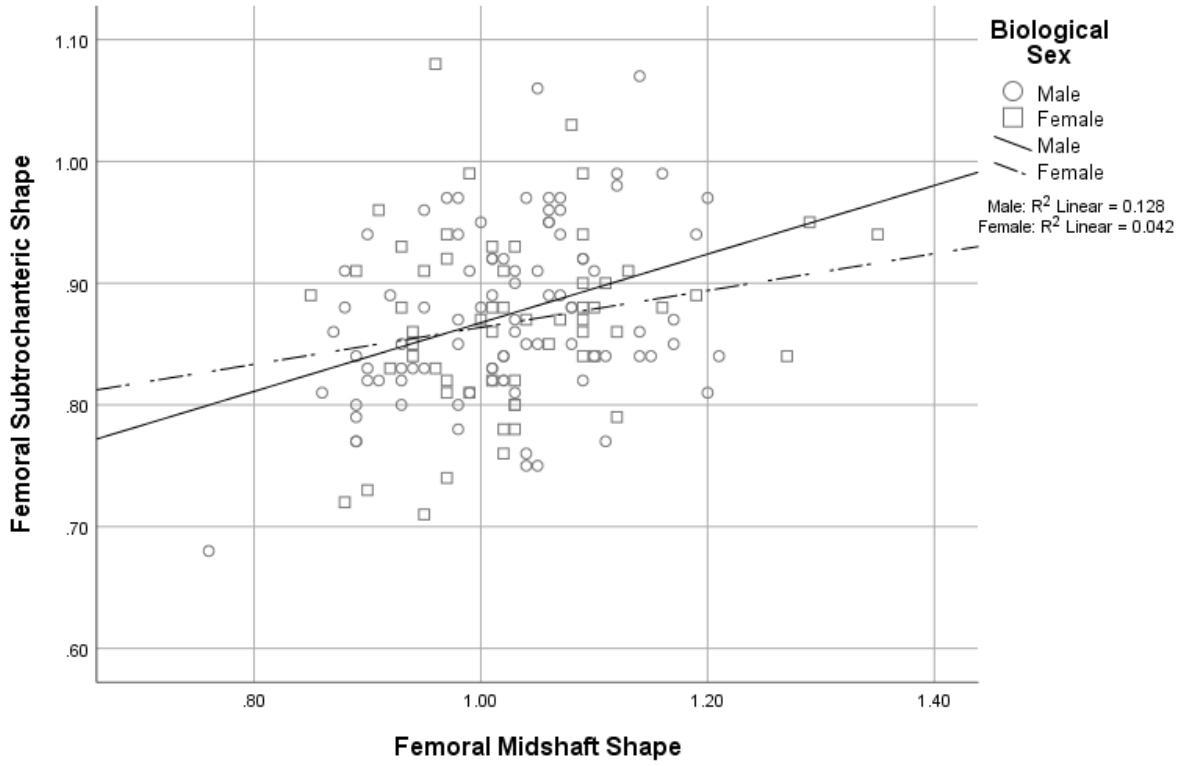

Figure 0.17 Distribution of the sample population by femoral subtrochanteric shape and femoral midshaft shape. The samples have further been identified by biological sex with R^2 values.

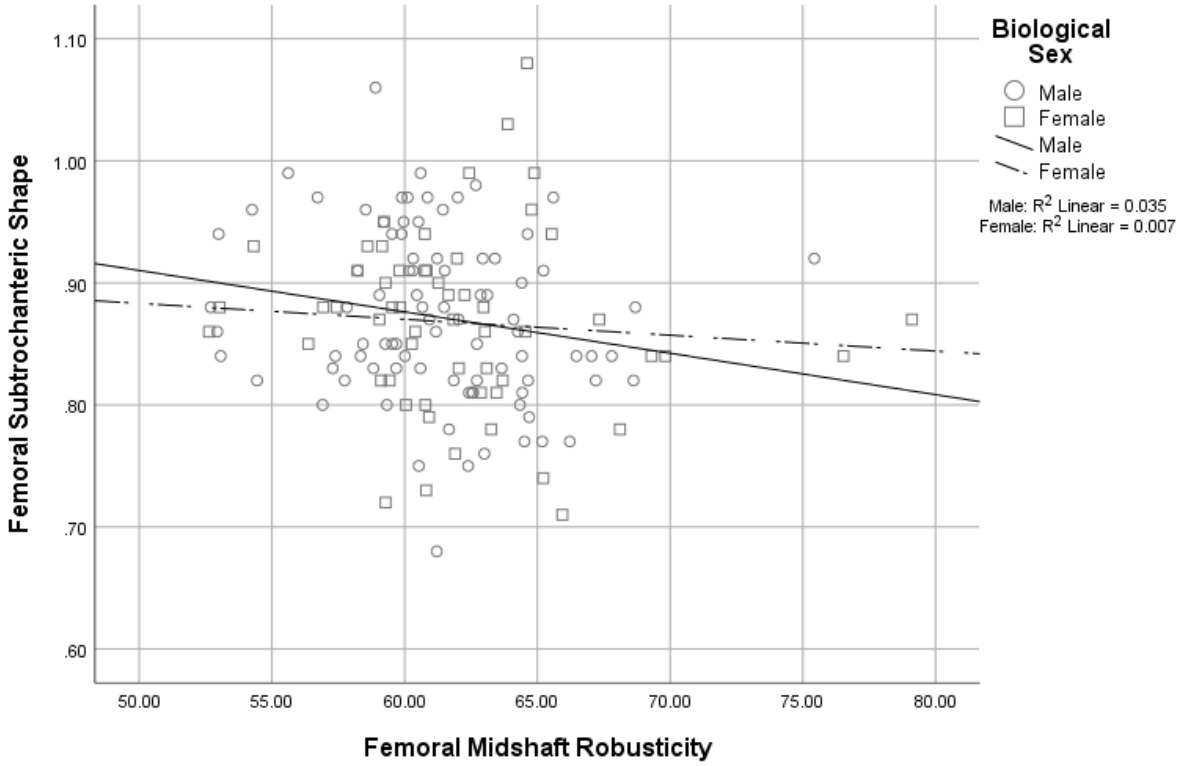

Figure 0.18 Distribution of the sample population by femoral subtrochanteric shape and femoral midshaft robusticity. The samples have further been identified by biological sex with R^2 values.

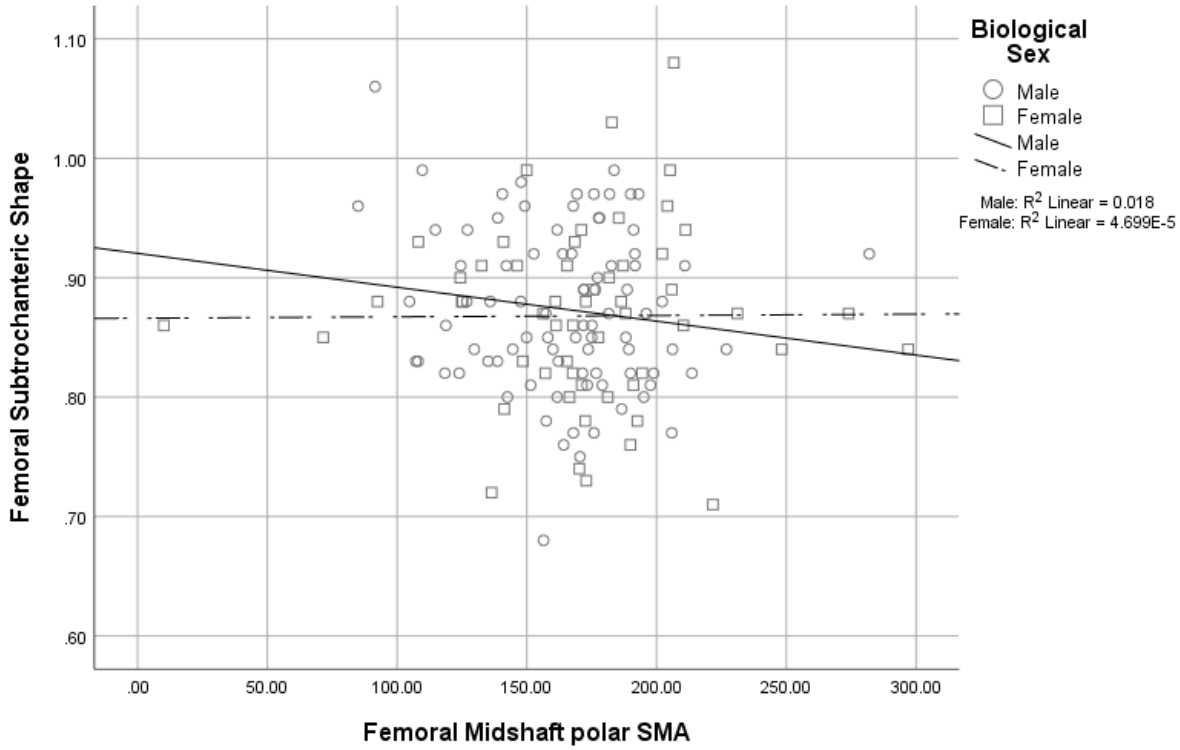

Figure 0.19 Distribution of the sample population by femoral subtrochanteric shape and femoral midshaft polar SMA. The samples have further been identified by biological sex with R^2 values.

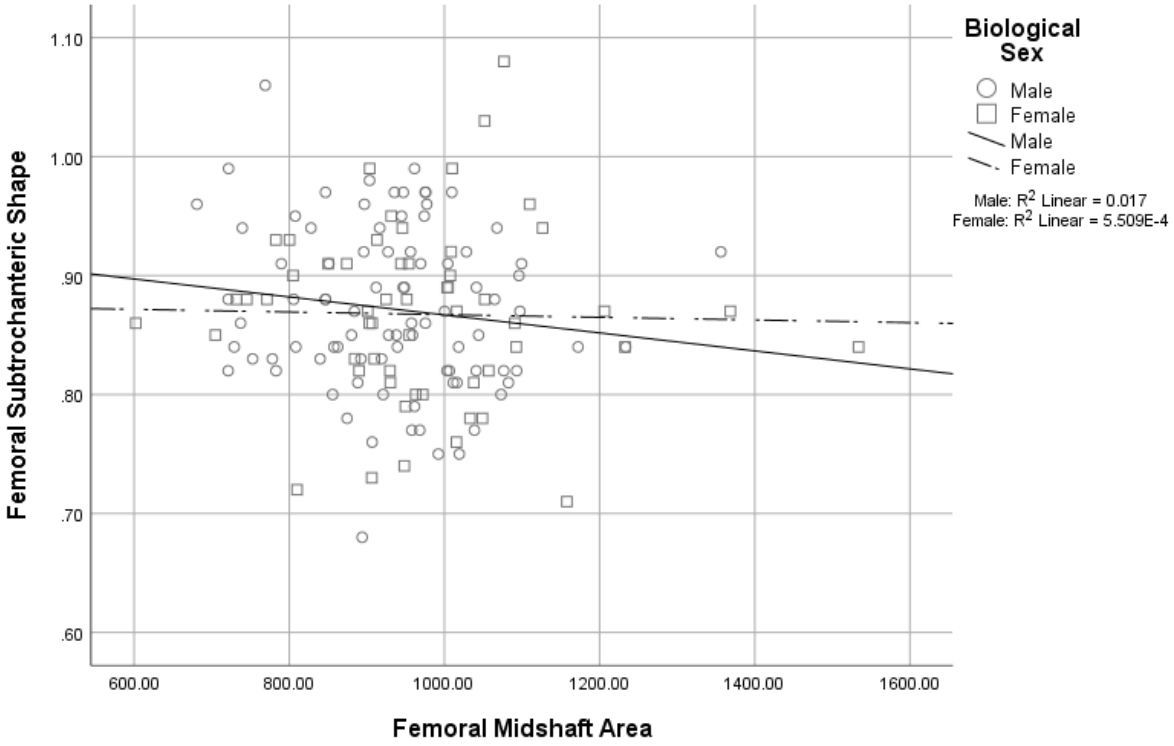

Figure 0.20 Distribution of the sample population by femoral subtrochanteric shape and femoral midshaft area. The samples have further been identified by biological sex with R^2 values.

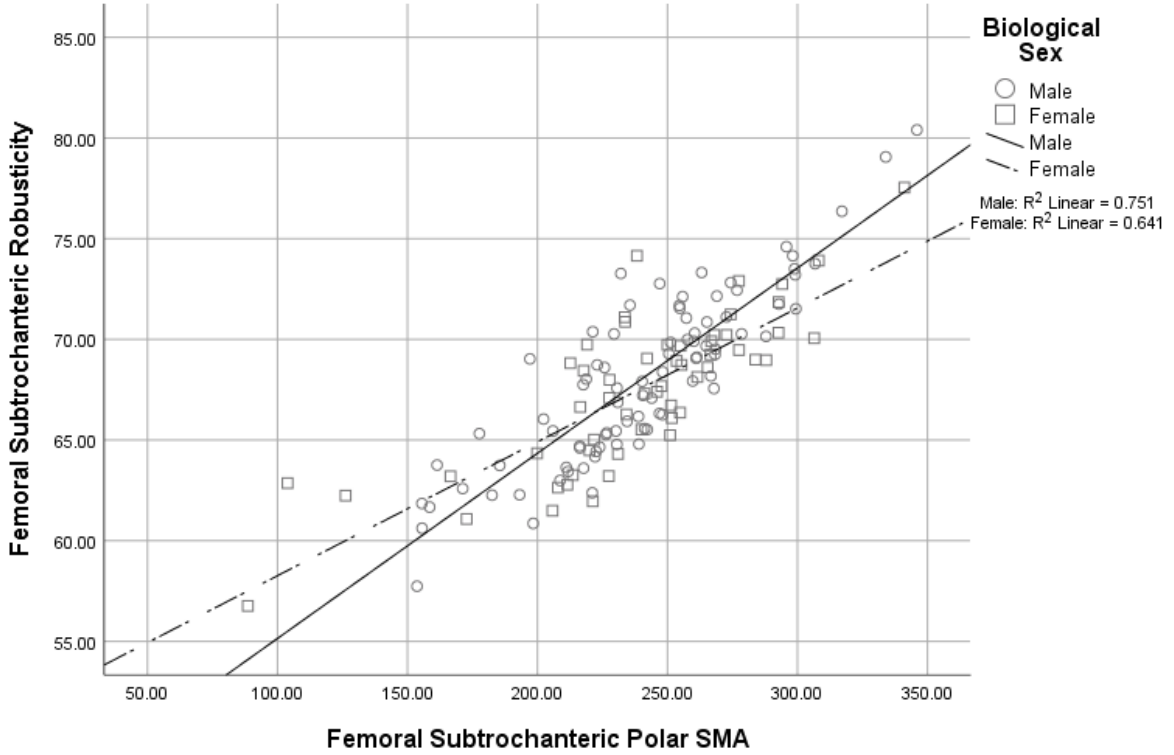

Figure 0.21 Distribution of the sample population by femoral subtrochanteric robusticity and femoral subtrochanteric polar SMA. The samples have further been identified by biological sex with R^2 values.

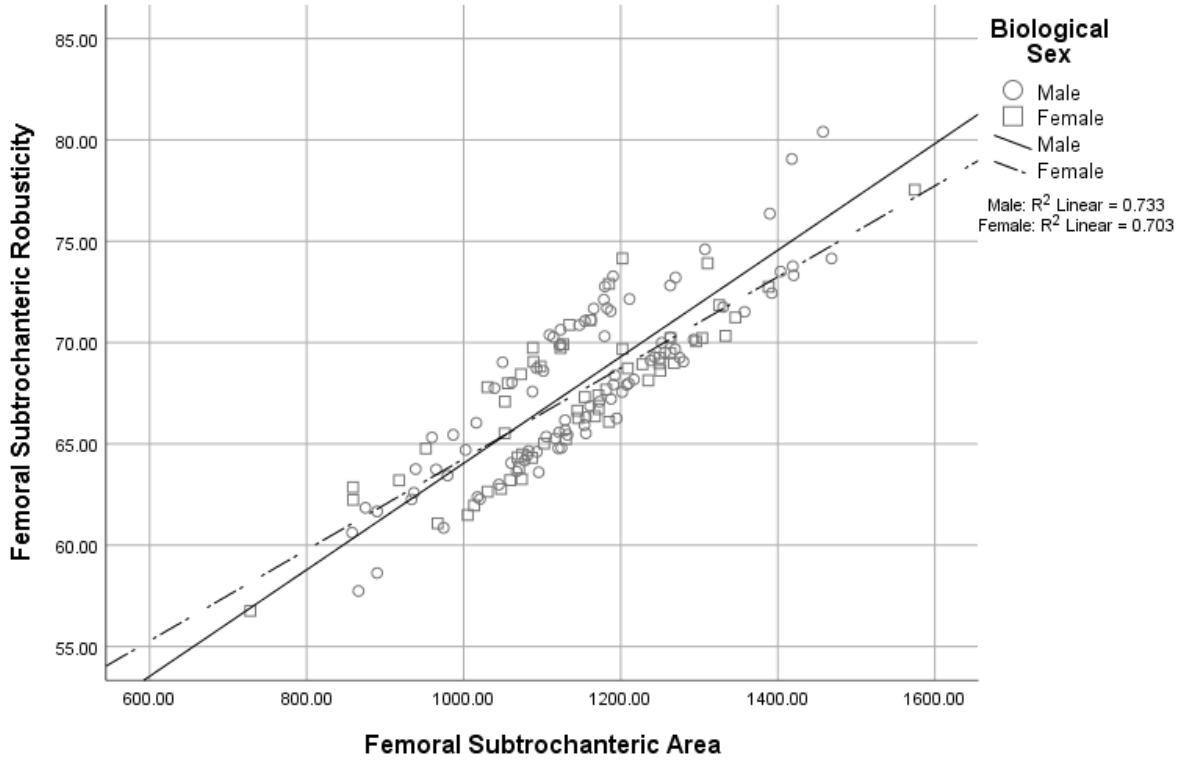

Figure 0.22 Distribution of the sample population by femoral subtrochanteric robusticity and femoral subtrochanteric area. The samples have further been identified by biological sex with R^2 values.

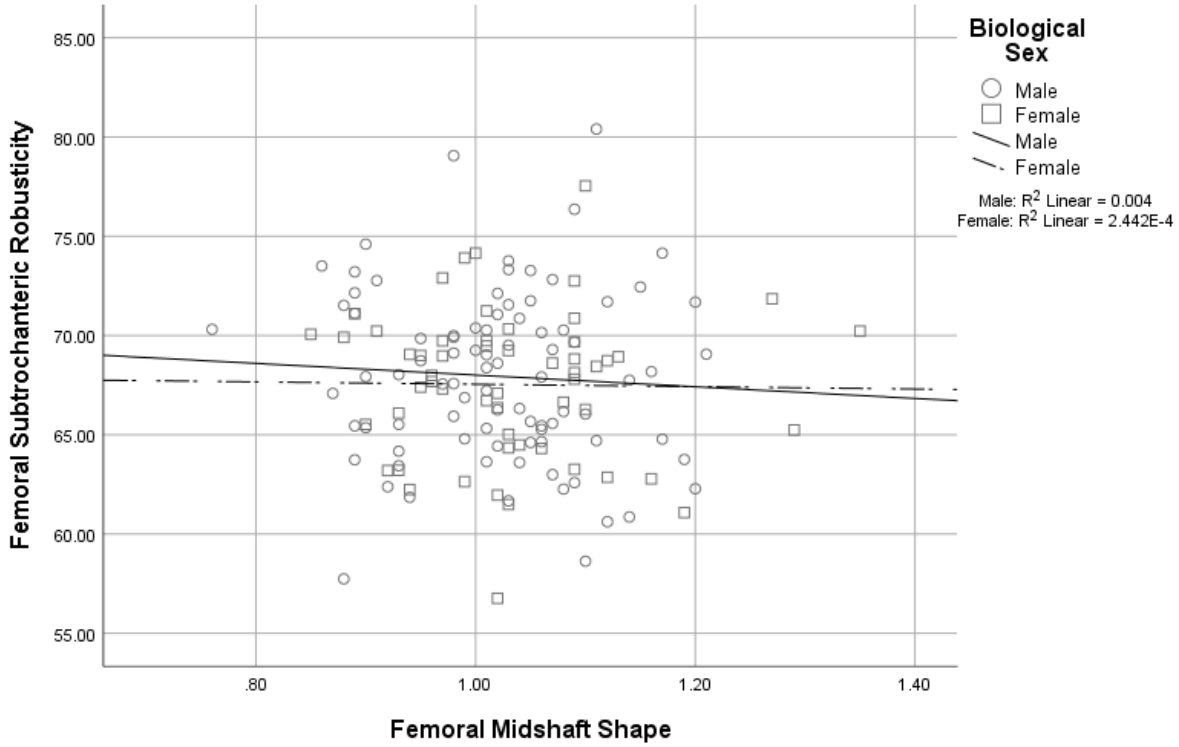

Figure 0.23 Distribution of the sample population by femoral subtrochanteric robusticity and femoral midshaft shape. The samples have further been identified by biological sex with R^2 values.

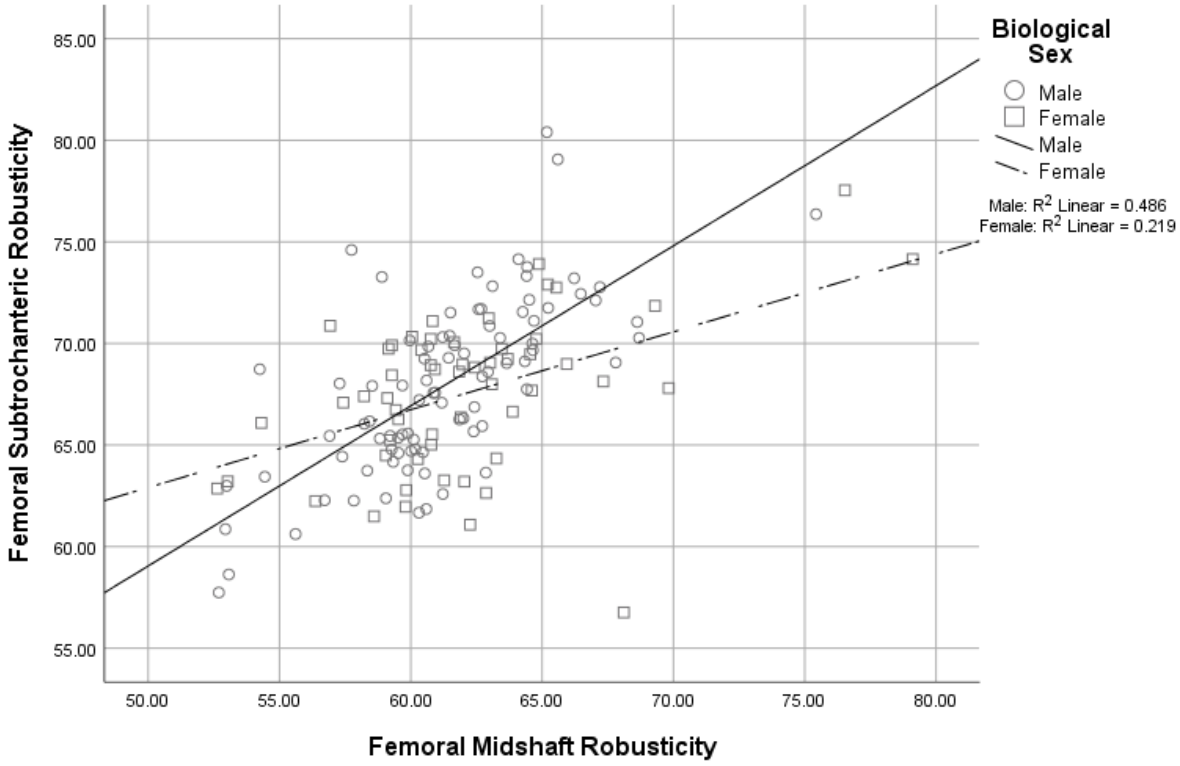

Figure 0.24 Distribution of the sample population by femoral subtrochanteric shape and femoral midshaft robusticity. The samples have further been identified by biological sex with R^2 values.

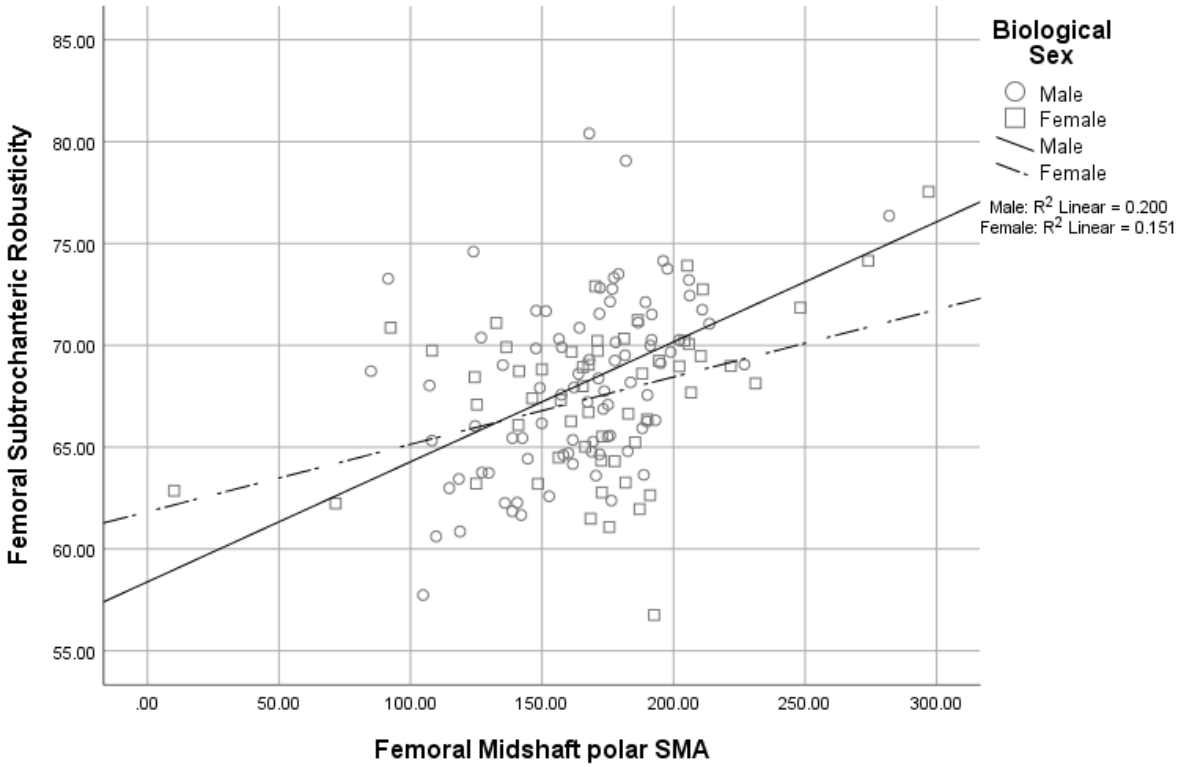

Figure 0.25 Distribution of the sample population by femoral subtrochanteric robusticity and femoral midshaft polar SMA. The samples have further been identified by biological sex with R^2 values.

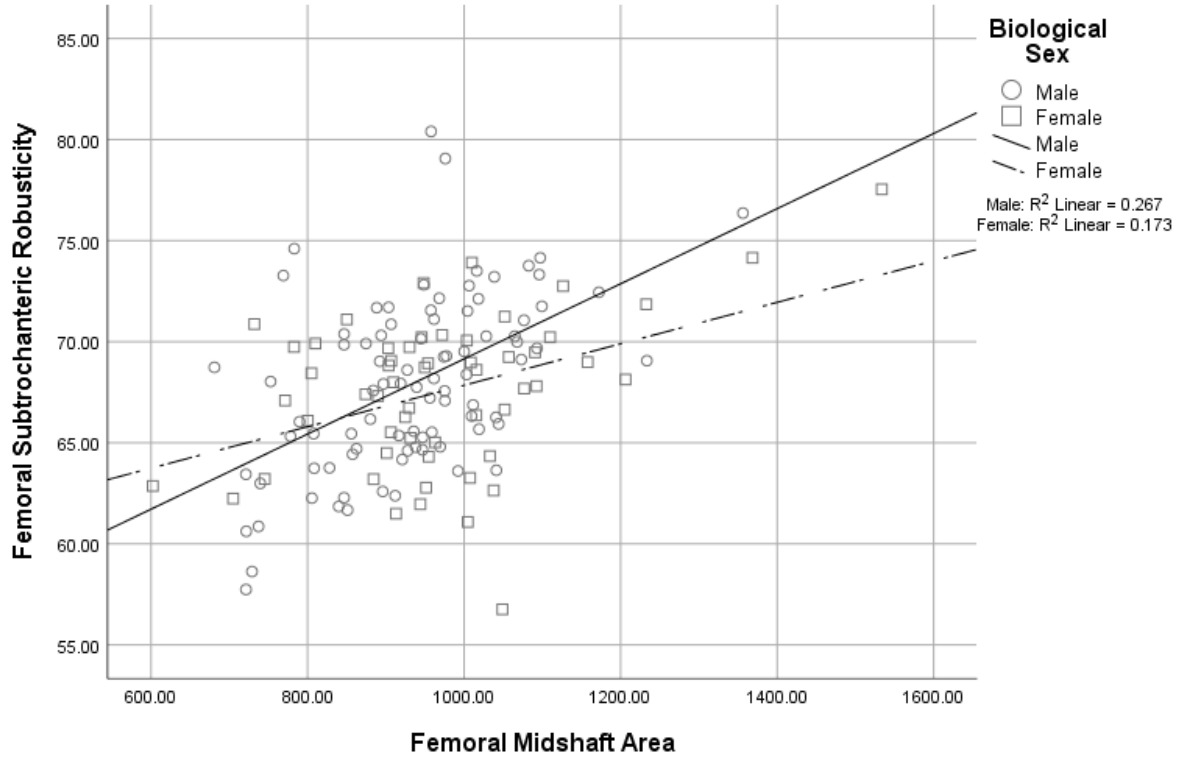

Figure 0.26 Distribution of the sample population by femoral subtrochanteric robusticity and femoral midshaft area. The samples have further been identified by biological sex with R^2 values.

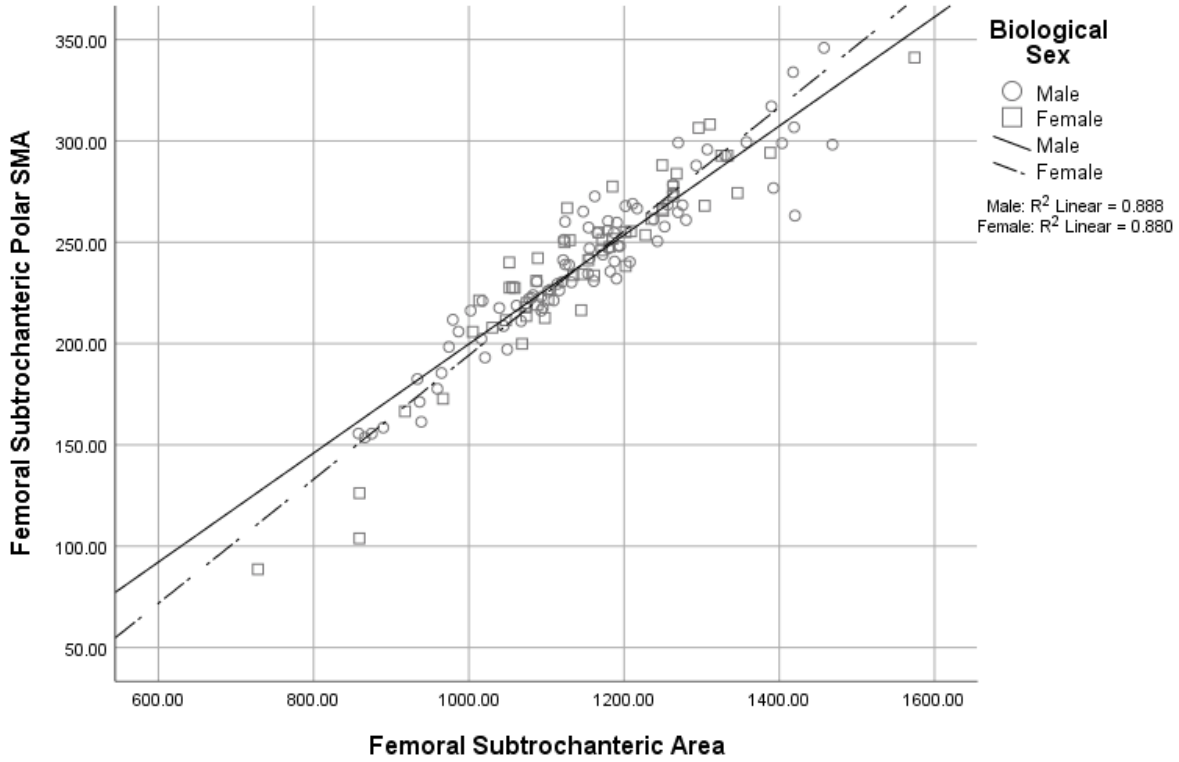

Figure 0.27 Distribution of the sample population by femoral subtrochanteric polar SMA and femoral subtrochanteric area. The samples have further been identified by biological sex with R^2 values.

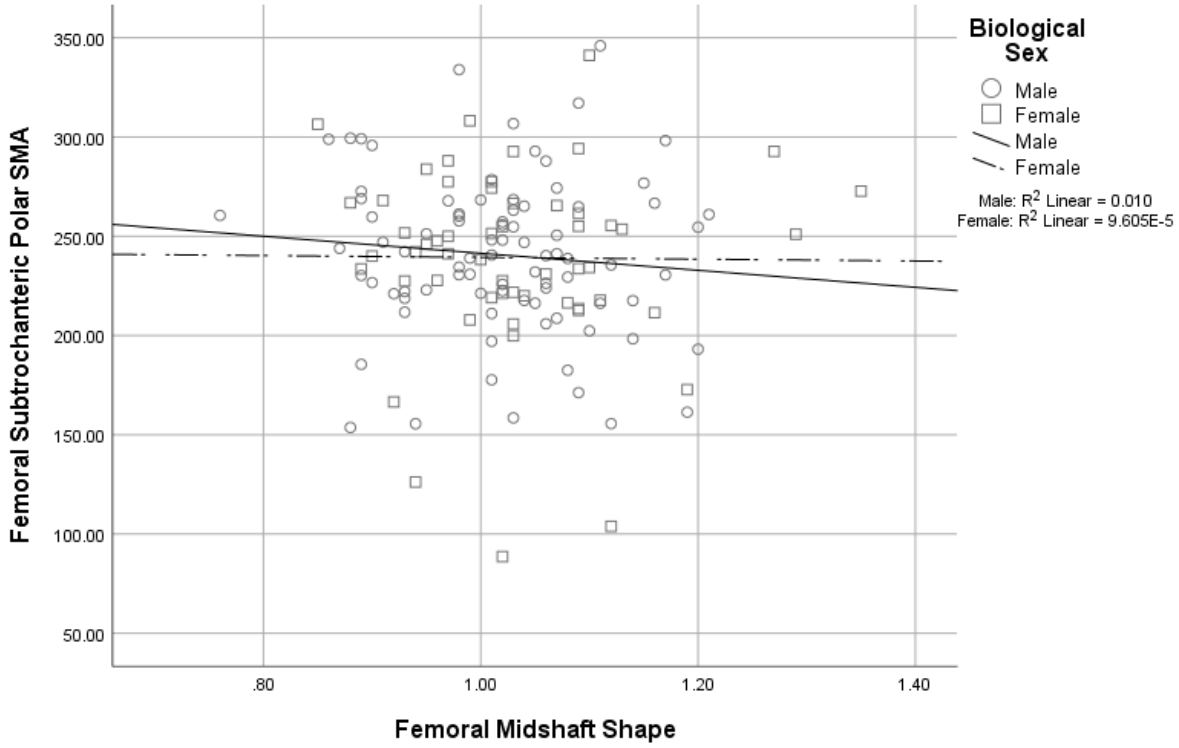

Figure 0.28 Distribution of the sample population by femoral subtrochanteric polar SMA and femoral midshaft shape. The samples have further been identified by biological sex with R^2 values.

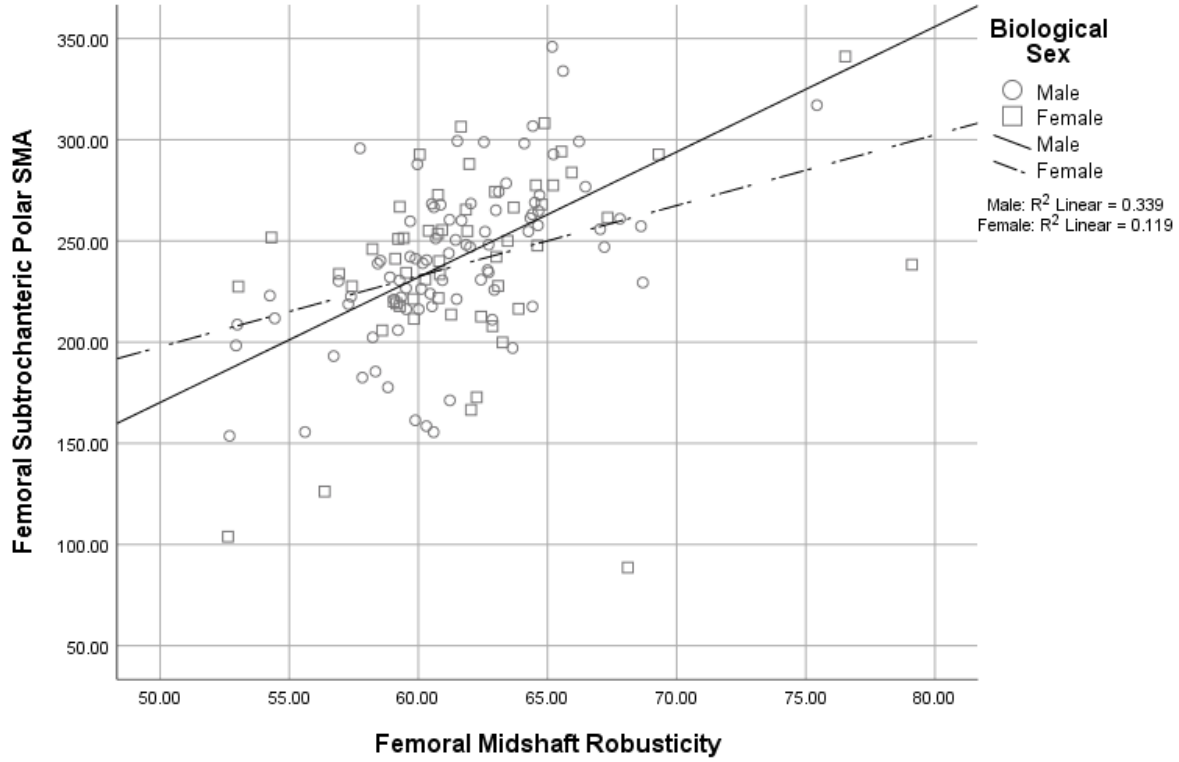

Figure 0.29 Distribution of the sample population by femoral subtrochanteric polar SMA and femoral midshaft robusticity. The samples have further been identified by biological sex with R^2 values.

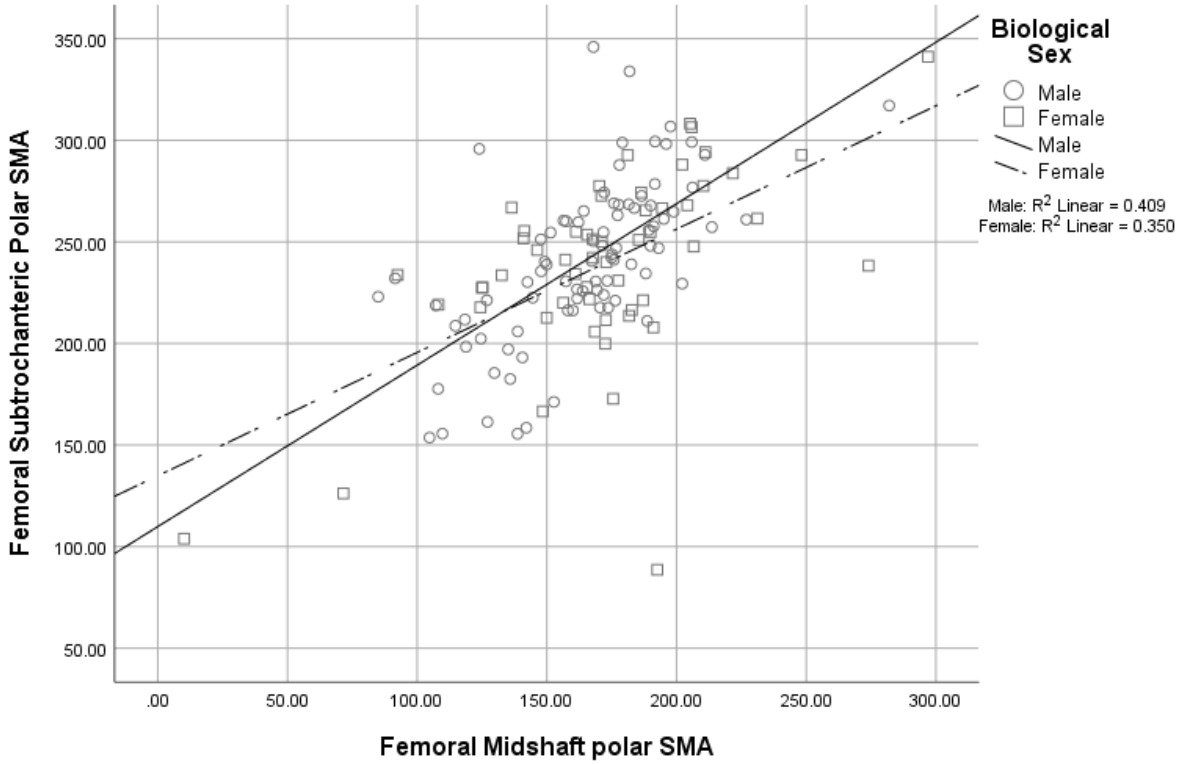

Figure 0.30 Distribution of the sample population by femoral subtrochanteric polar SMA and femoral midshaft polar SMA. The samples have further been identified by biological sex with R^2 values.

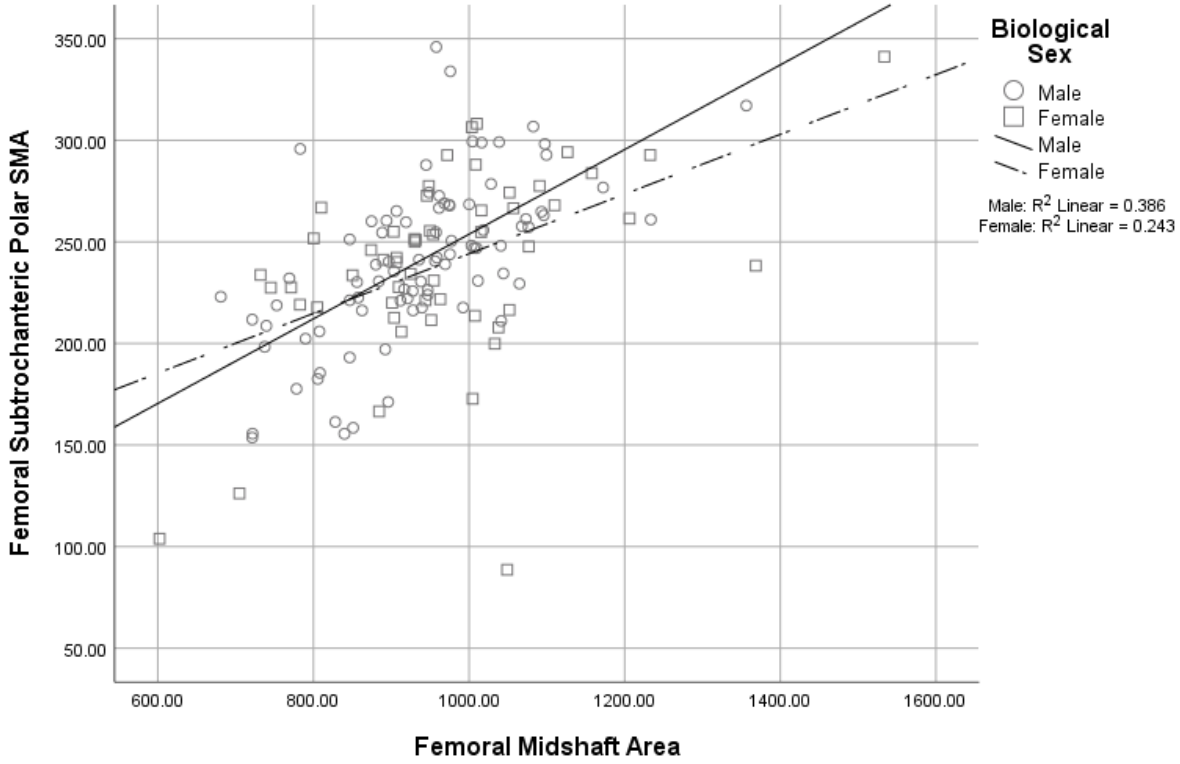

Figure 0.31 Distribution of the sample population by femoral subtrochanteric polar SMA and femoral midshaft area. The samples have further been identified by biological sex with R^2 values.

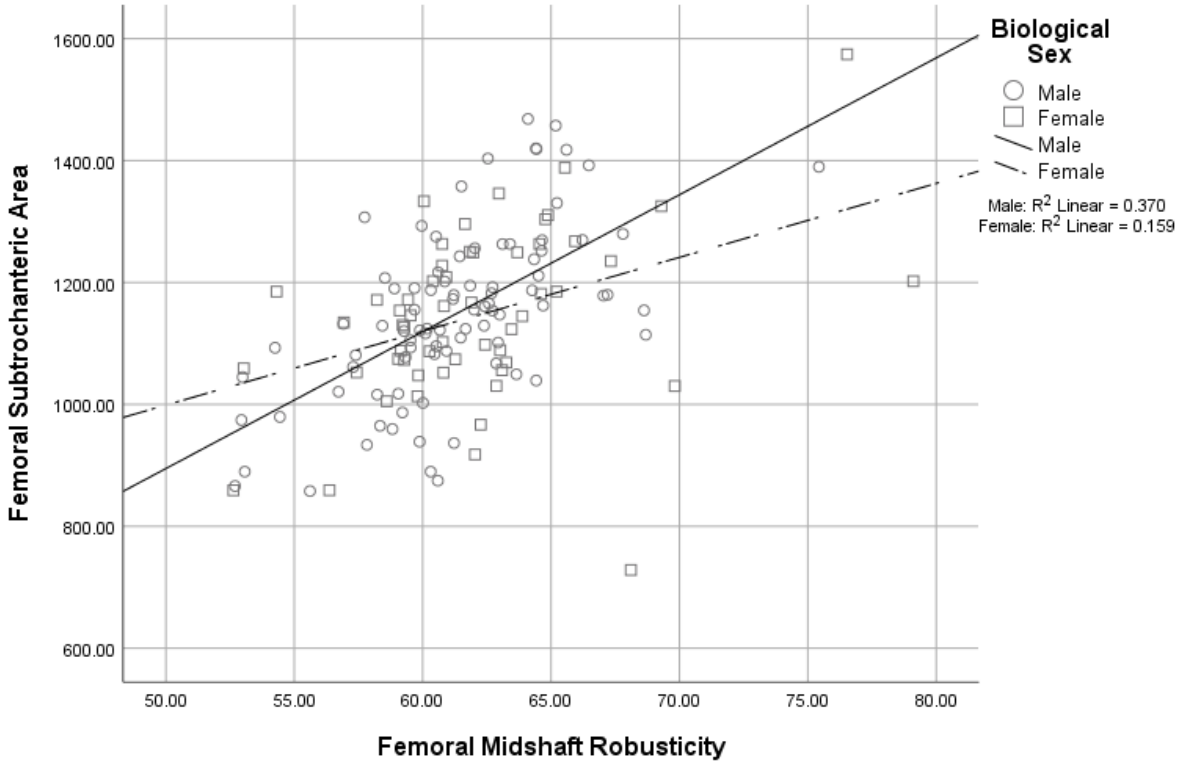

Figure 0.32 Distribution of the sample population by femoral subtrochanteric area and femoral midshaft robusticity. The samples have further been identified by biological sex with R^2 values.

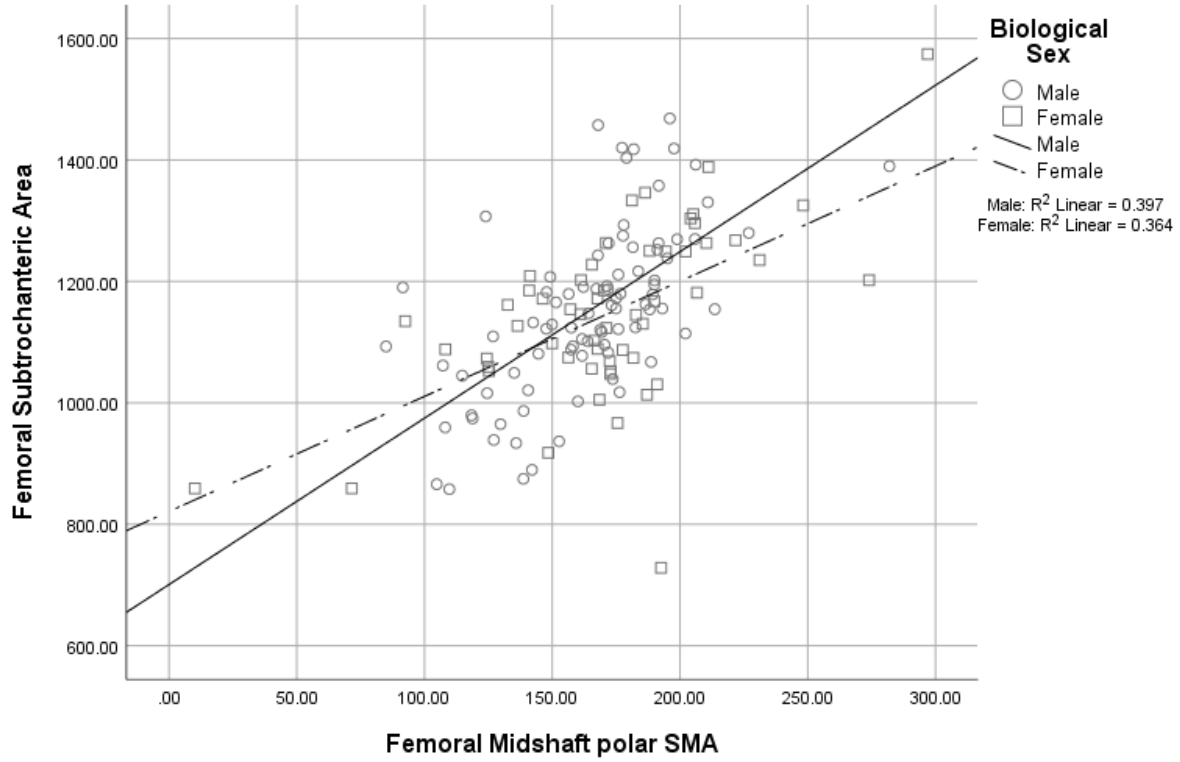

Figure 0.33 Distribution of the sample population by femoral subtrochanteric area and femoral midshaft polar SMA. The samples have further been identified by biological sex with R^2 values.

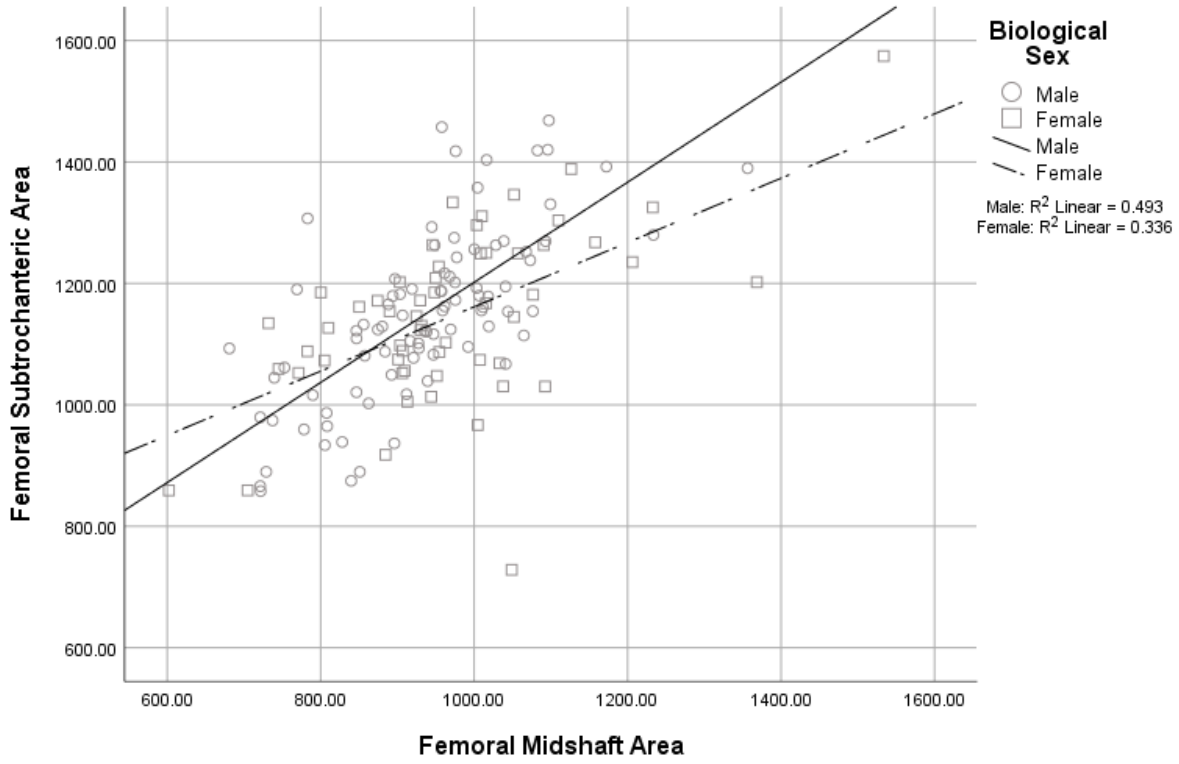

Figure 0.34 Distribution of the sample population by femoral subtrochanteric area and femoral midshaft area. The samples have further been identified by biological sex with R^2 values.

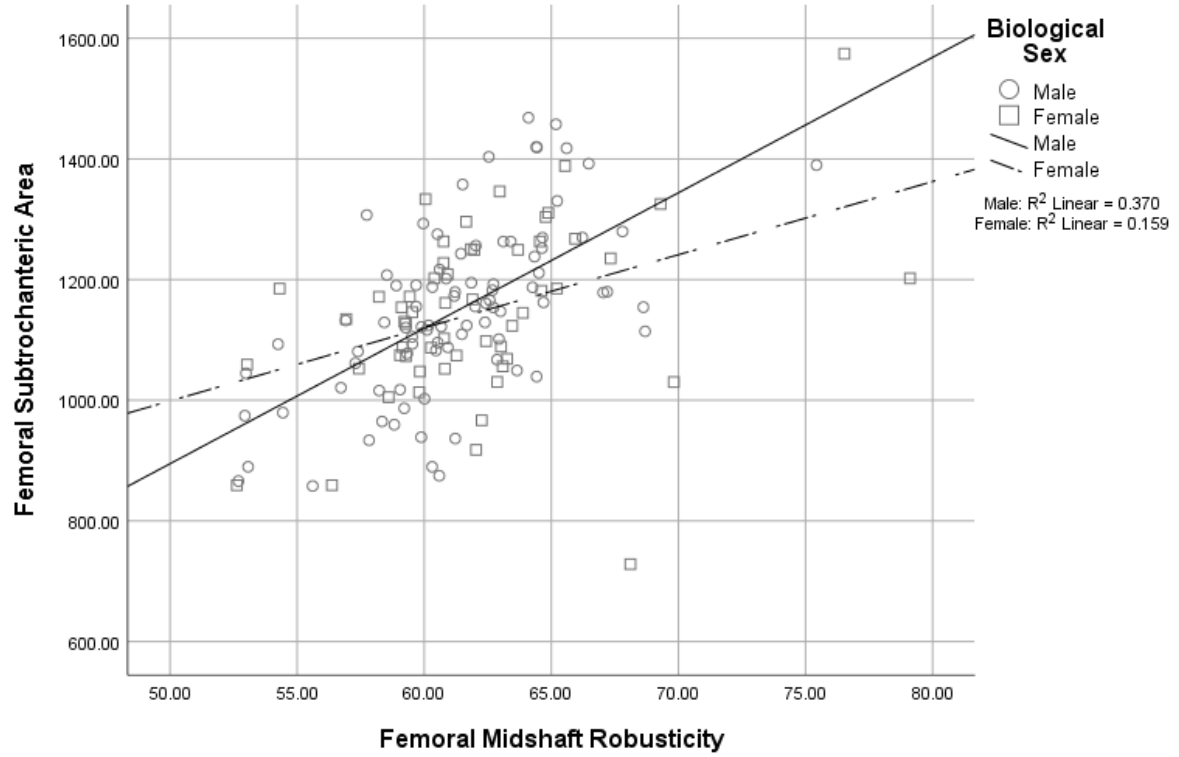

Figure 0.35 Distribution of the sample population by femoral subtrochanteric area and femoral midshaft robusticity. The samples have further been identified by biological sex with R^2 values.

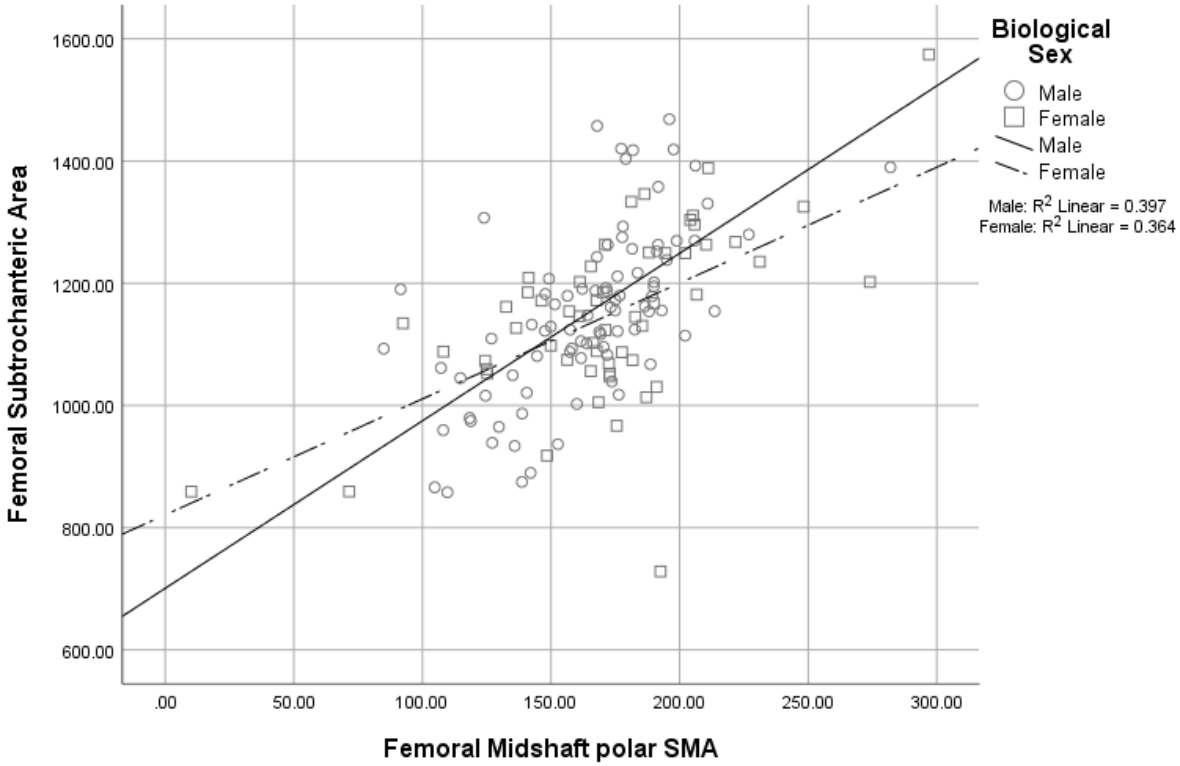

Figure 0.36 Distribution of the sample population by femoral subtrochanteric area and femoral midshaft polar SMA. The samples have further been identified by biological sex with R^2 values.

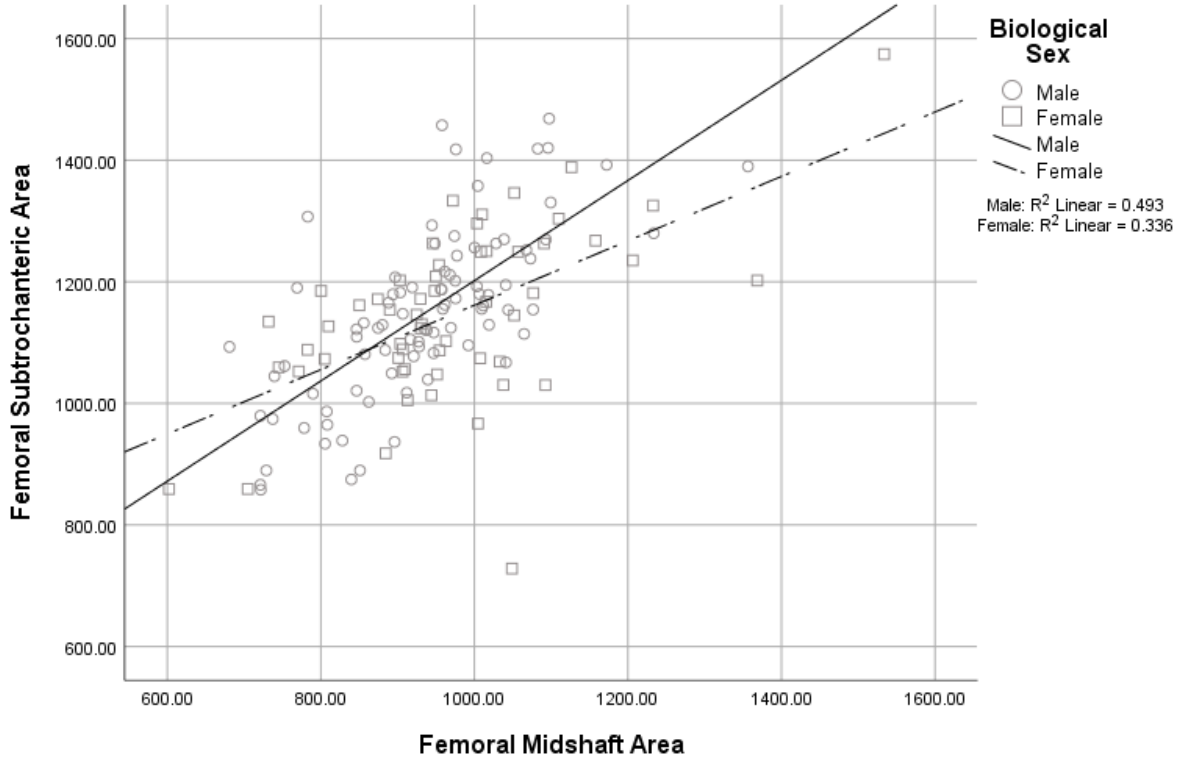

Figure 0.37 Distribution of the sample population by femoral subtrochanteric area and femoral midshaft area. The samples have further been identified by biological sex with R^2 values.

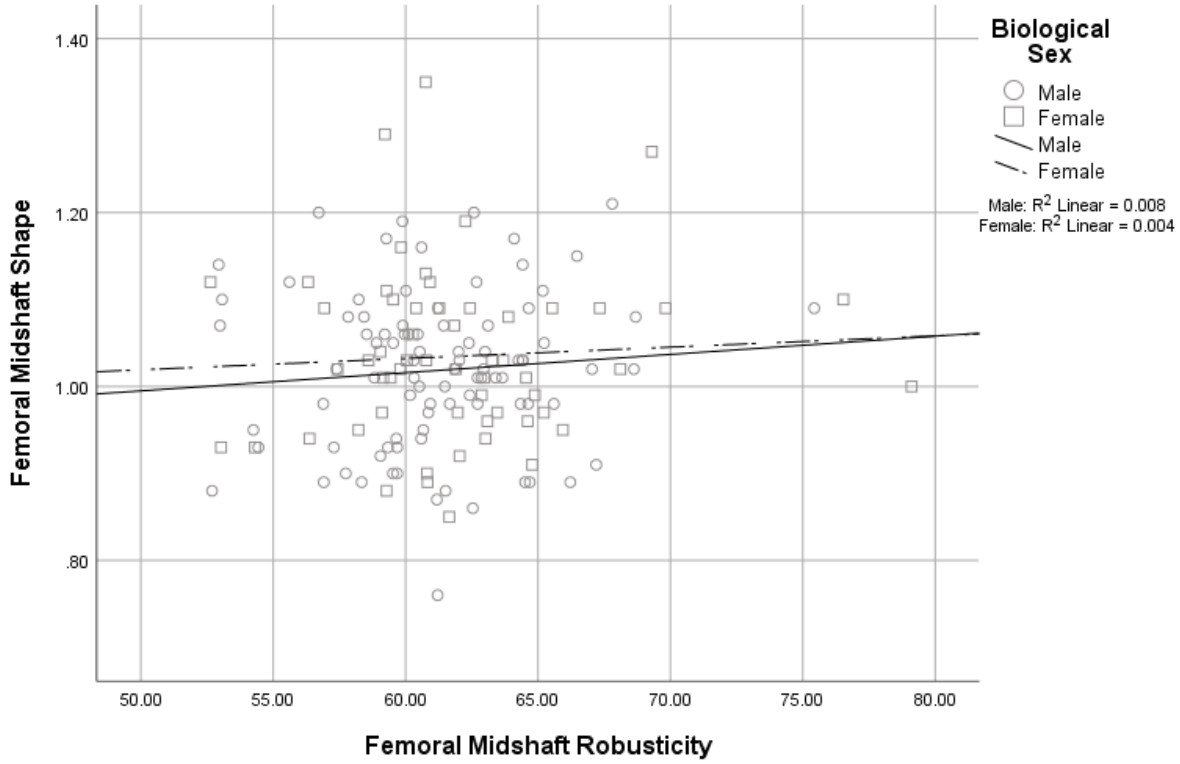

Figure 0.38 Distribution of the sample population by femoral midshaft shape and femoral midshaft robusticity. The samples have further been identified by biological sex with R^2 values.

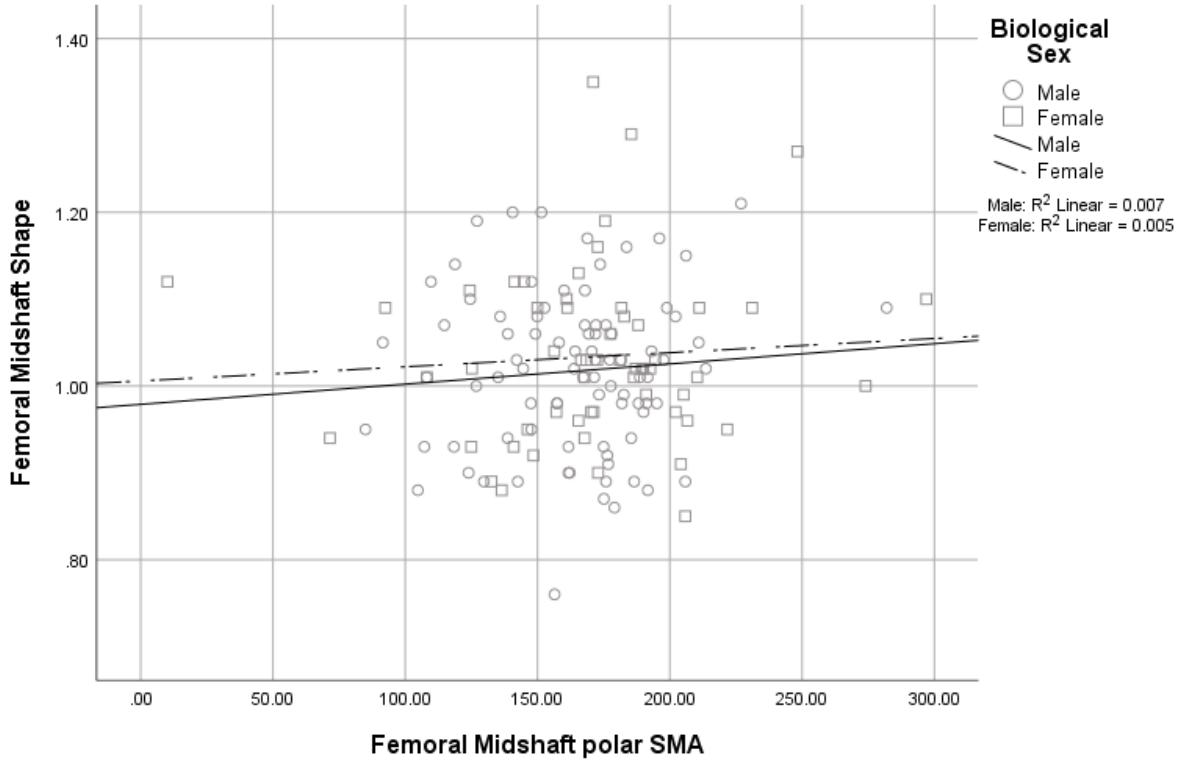

Figure 0.39 Distribution of the sample population by femoral midshaft shape and femoral midshaft polar SMA. The samples have further been identified by biological sex with R^2 values.

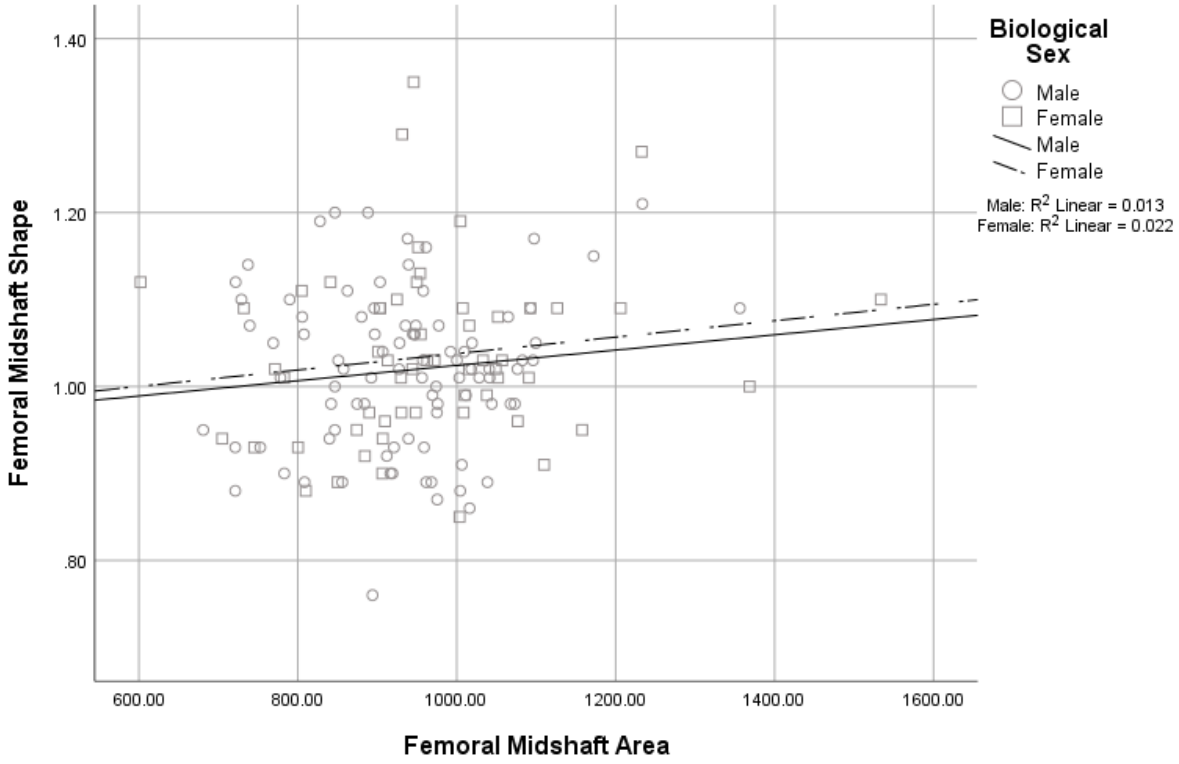

Figure 0.40 Distribution of the sample population by femoral subtrochanteric shape and femoral midshaft area. The samples have further been identified by biological sex with R^2 values.

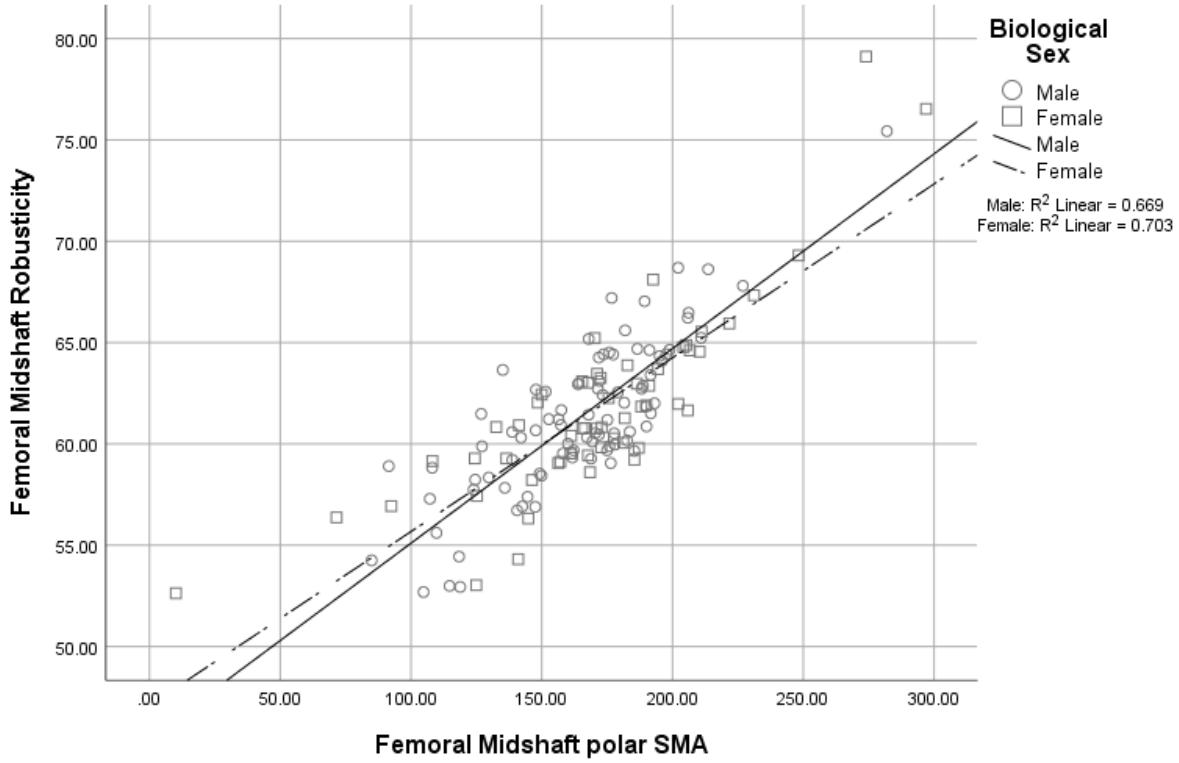

Figure 0.41 Distribution of the sample population by femoral midshaft robusticity and femoral midshaft polar SMA. The samples have further been identified by biological sex with R^2 values.

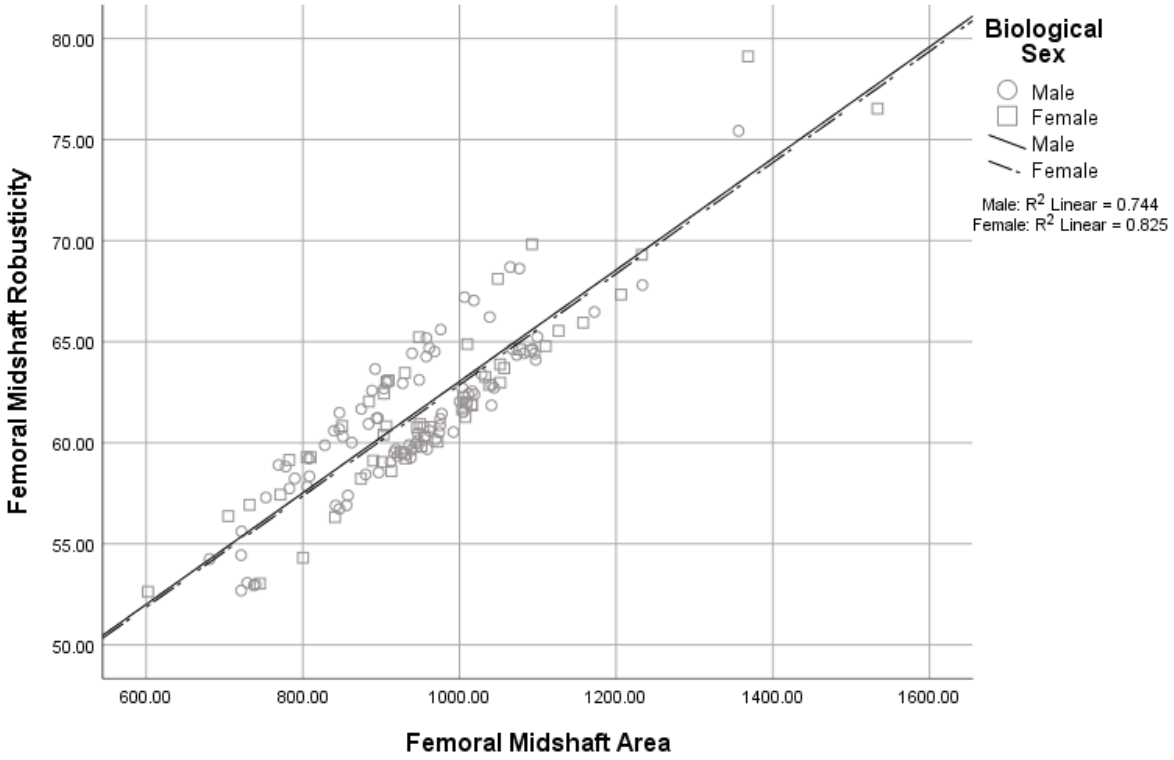

Figure 0.42 Distribution of the sample population by femoral midshaft robusticity and femoral midshaft area. The samples have further been identified by biological sex with R^2 values.

Risk Ratios

Table 0.1 Risk ratio for osteoarthritis with a biological sex control.

		COUNT		PREVALENCE RATE	
OA BIOLOGICAL SEX		A	P	A	P
	MALE	65	38	63.1%	36.9%
	FEMALE	50	22	69.4%	30.6%
		RISK RATIO			
	MALE/FEMALE	1.21			

Table includes the prevalence rates and risk ratios by category.

Table 0.2 Risk ratio for osteoarthritis with an age at death control.

		COUNT		PREVALENCE RATE	
		A	P	A	P
OA AGE AT DEATH CATEGORY	EARLY	45	11	80.4%	19.6%
	MIDDLE	31	9	77.5%	22.5%
	LATE	39	40	49.4%	50.6%
		RISK RATIO			
	MIDDLE/EARLY	1.15			
	LATE/MIDDLE	2.25			
	LATE/EARLY	2.58			

Table includes the prevalence rates and risk ratios by category.

Table 0.3 Risk ratio for osteoarthritis with a body mass index control.

		COUNT		PREVALENCE RATE	
		A	P	A	P
OA BODY MASS INDEX INTERQUARTILE	1: <25%	20	17	54.1%	45.9%
	2: 25-50%	25	13	65.8%	34.2%
	3: 50-75%	26	14	65.0%	35.0%
	4: >75%	24	11	68.6%	31.4%
		RISK RATIO			
	2/1	0.74			
	3/2	1.02			
	4/3	0.90			
	4/1	0.68			

Table includes the prevalence rates and risk ratios by category.

Table 0.4 Risk ratio for osteoarthritis with an entheseal change control.

		COUNT		PREVALENCE RATE	
		A	P	A	P
OA ENTHESEAL CHANGE	SLIGHT	54	16	77.1%	22.9%
	MODERATE	55	34	61.8%	38.2%
	SEVERE	6	10	37.5%	62.5%
		RISK RATIO			
	MODERATE/SLIGHT	1.67			
	SEVERE/MODERATE	1.64			
	SEVERE/SLIGHT	2.73			

Table includes the prevalence rates and risk ratios by category.

Table 0.5 Risk ratio for osteoarthritis with a femoral subtrochanteric shape control.

		COUNT		PREVALENCE RATE	
		A	P	A	P
OA FEMORAL SUBTROCHANTERIC SHAPE	1: <25%	25	13	65.8%	34.2%
	2: 25-50%	26	13	66.7%	33.3%
	3: 50-75%	25	13	65.8%	34.2%
	4: >75%	24	14	63.2%	36.8%
INTERQUARTILE RANGE		RISK RATIO			
	2/1	0.97			
	3/2	1.03			
	4/3	1.08			
	4/1	1.08			

Table includes the prevalence rates and risk ratios by category.

Table 0.6 Risk ratio for osteoarthritis with a femoral subtrochanteric robusticity control.

		COUNT		INCINDENCE RATE	
		A	P	A	P
OA FEMORAL SUBTROCHANTERIC ROBUSITICITY	1: <25%	25	12	67.6%	32.4%
	2: 25-50%	19	18	51.4%	48.6%
	3: 50-75%	33	8	80.5%	19.5%
	4: >75%	20	13	60.6%	39.4%
INTERQUARTILE RANGE		RISK RATIO			
	2/1	1.50			
	3/2	0.40			
	4/3	2.02			
	4/1	1.21			

Table includes the prevalence rates and risk ratios by category.

Table 0.7 Risk ratio for osteoarthritis with a femoral subtrochanteric polar SMA control.

		COUNT		PREVALENCE RATE	
		A	P	A	P
OA FEMORAL SUBTROCHANTERIC POLAR SMA	1: <25%	21	14	60.0%	40.0%
	2: 25-50%	27	9	75.0%	25.0%
	3: 50-75%	23	12	65.7%	34.3%
	4: >75%	23	11	67.6%	32.4%
INTERQUARTILE RANGE		RISK RATIO			
	2/1	0.63			
	3/2	1.37			
	4/3	0.94			
	4/1	0.81			

Table includes the prevalence rates and risk ratios by category.

Table 0.8 Risk ratio for osteoarthritis with a femoral subtrochanteric area control.

		COUNT		PREVALENCE RATE	
		A	P	A	P
OA FEMORAL SUBTROCHANTERIC AREA	1: <25%	22	15	59.5%	40.5%
	2: 25-50%	27	9	75.0%	25.0%
	3: 50-75%	22	17	56.4%	43.6%
	4: >75%	26	10	72.2%	27.8%
INTERQUARTILE RANGE		RISK RATIO			
	2/1	0.62			
	3/2	1.74			
	4/3	0.64			
	4/1	0.69			

Table includes the prevalence rates and risk ratios by category.

Table 0.9 Risk ratio for osteoarthritis with a femoral midshaft shape control.

		COUNT		PREVALENCE RATE	
		A	P	A	P
OA FEMORAL MIDSHAFT SHAPE	1: <25%	24	14	63.2%	36.8%
	2: 25-50%	24	14	63.2%	36.8%
	3: 50-75%	24	14	63.2%	36.8%
	4: >75%	26	12	68.4%	31.6%
INTERQUARTILE RANGE		RISK RATIO			
	2/1	1.00			
	3/2	1.00			
	4/3	0.86			
	4/1	0.86			

Table includes the prevalence rates and risk ratios by category.

Table 0.10 Risk ratio for osteoarthritis with a femoral midshaft robusticity control.

		RISK RATIO		PREVALENCE RATE	
		A	P	A	P
OA FEMORAL MIDSHAFT ROBUSTICITY	1: <25%	20	15	57.1%	42.9%
	2: 25-50%	27	11	71.1%	28.9%
	3: 50-75%	28	11	71.8%	28.2%
	4: >75%	20	14	58.8%	41.2%
INTERQUARTILE RANGE		RISK RATIO			
	2/1	0.68			
	3/2	0.97			
	4/3	1.46			
	4/1	0.96			

Table includes the prevalence rates and risk ratios by category.

Table 0.11 Risk ratio for osteoarthritis with a femoral midshaft polar SMA control.

		COUNT		PREVALENCE RATE	
		A	P	A	P
OA FEMORAL MIDSHAFT POLAR SMA	1: <25%	20	14	58.8%	41.2%
	2: 25-50%	23	12	65.7%	34.3%
	3: 50-75%	20	14	58.8%	41.2%
	4: >75%	32	8	80.0%	20.0%
INTERQUARTILE RANGE		RISK RATIO			
	2/1	0.83			
	3/2	1.20			
	4/3	0.49			
	4/1	0.49			

Table includes the prevalence rates and risk ratios by category.

Table 0.12 Risk ratio for osteoarthritis with a femoral midshaft area control.

		COUNT		INCINDENCE RATE	
		A	P	A	P
OA FEMORAL MIDSHAFT AREA	1: <25%	22	15	59.5%	40.5%
	2: 25-50%	27	10	73.0%	27.0%
	3: 50-75%	18	19	48.6%	51.4%
	4: >75%	28	7	80.0%	20.0%
INTERQUARTILE RANGE		RISK RATIO			
	2/1	0.67			
	3/2	1.90			
	4/3	0.39			
	4/1	0.49			

Table includes the prevalence rates and risk ratios by category.

Table 0.13 Risk ratio for spinal osteoarthritis with a biological sex control.

		COUNT		PREVALENCE RATE	
		A	P	A	P
SOA BIOLOGICAL SEX	MALE	73	25	74.5%	25.5%
	FEMALE	54	16	77.1%	22.9%
		RISK RATIO			
	MALE/FEMALE	1.12			

Table includes the prevalence rates and risk ratios by category.

Table 0.14 Risk ratio for spinal osteoarthritis with an age at death control.

		COUNT		PREVALENCE RATE	
		A	P	A	P
SOA AGE AT DEATH CATEGORY	EARLY	50	6	89.3%	10.7%
	MIDDLE	30	7	81.1%	18.9%
	LATE	47	28	62.7%	37.3%
		RISK RATIO			
	MIDDLE/EARLY	1.77			
	LATE/MIDDLE	1.97			
	LATE/EARLY	3.48			

Table includes the prevalence rates and risk ratios by category.

Table 0.15 Risk ratio for spinal osteoarthritis with a body mass index control.

		COUNT		PREVALENCE RATE	
		A	P	A	P
SOA BODY MASS INDEX INTERQUARTILE	1: <25%	25	12	67.6%	32.4%
	2: 25-50%	25	12	67.6%	32.4%
	3: 50-75%	29	10	74.4%	25.6%
	4: >75%	27	5	84.4%	15.6%
		RISK RATIO			
	2/1	1.00			
	3/2	0.79			
	4/3	0.61			
	4/1	0.48			

Table includes the prevalence rates and risk ratios by category.

Table 0.16 Risk ratio for spinal osteoarthritis with an entheseal change control.

		COUNT		PREVALENCE RATE	
		A	P	A	P
SOA ENTHESEAL CHANGE	SLIGHT	57	10	85.1%	14.9%
	MODERATE	62	24	72.1%	27.9%
	SEVERE	8	7	53.3%	46.7%
		RISK RATIO			
	MODERATE/SLIGHT	1.87			
	SEVERE/MODERATE	1.67			
	SEVERE/SLIGHT	3.13			

Table includes the prevalence rates and risk ratios by category.

Table 0.17 Risk ratio for spinal osteoarthritis with a femoral subtrochanteric shape control.

		COUNT		PREVALENCE RATE	
		A	P	A	P
SOA FEMORAL SUBTROCHANTERIC SHAPE INTERQUARTILE RANGE	1: <25%	28	9	75.7%	24.3%
	2: 25-50%	27	10	73.0%	27.0%
	3: 50-75%	28	9	75.7%	24.3%
	4: >75%	28	8	77.8%	22.2%
		RISK RATIO			
	2/1	1.11			
	3/2	0.90			
	4/3	0.91			
	4/1	0.91			

Table includes the prevalence rates and risk ratios by category.

Table 0.18 Risk ratio for spinal osteoarthritis with a femoral subtrochanteric robusticity control.

		COUNT		PREVALENCE RATE	
		A	P	A	P
SOA FEMORAL SUBTROCHANTERIC ROBUSITICITY	1: <25%	28	7	80.0%	20.0%
	2: 25-50%	22	13	62.9%	37.1%
	3: 50-75%	34	6	85.0%	15.0%
	4: >75%	24	8	75.0%	25.0%
INTERQUARTILE RANGE		RISK RATIO			
	2/1	1.86			
	3/2	0.40			
	4/3	1.67			
	4/1	1.25			

Table includes the prevalence rates and risk ratios by category.

Table 0.19 Risk ratio for spinal osteoarthritis with a femoral subtrochanteric polar SMA control.

		COUNT		PREVALENCE RATE	
		A	P	A	P
SOA FEMORAL SUBTROCHANTERIC POLAR SMA	1: <25%	25	9	73.5%	26.5%
	2: 25-50%	29	5	85.3%	14.7%
	3: 50-75%	23	10	69.7%	30.3%
	4: >75%	27	6	81.8%	18.2%
INTERQUARTILE RANGE		RISK RATIO			
	2/1	0.56			
	3/2	2.06			
	4/3	0.60			
	4/1	0.69			

Table includes the prevalence rates and risk ratios by category.

Table 0.20 Risk ratio for spinal osteoarthritis with a femoral subtrochanteric area control.

		COUNT		PREVALENCE RATE	
		A	P	A	P
SOA FEMORAL SUBTROCHANTERIC AREA	1: <25%	27	9	75.0%	25.0%
	2: 25-50%	27	8	77.1%	22.9%
	3: 50-75%	35	11	76.1%	23.9%
	4: >75%	29	6	82.9%	17.1%
INTERQUARTILE RANGE		RISK RATIO			
	2/1	0.91			
	3/2	1.05			
	4/3	0.72			
	4/1	0.69			

Table includes the prevalence rates and risk ratios by category.

Table 0.21 Risk ratio for spinal osteoarthritis with a femoral subtrochanteric robusticity control.

		COUNT		PREVALENCE RATE	
		A	P	A	P
SOA FEMORAL MIDSHAFT SHAPE	1: <25%	28	9	75.7%	24.3%
	2: 25-50%	28	8	77.8%	22.2%
	3: 50-75%	28	9	75.7%	24.3%
	4: >75%	26	10	72.2%	27.8%
INTERQUARTILE RANGE		RISK RATIO			
	2/1	0.91			
	3/2	1.09			
	4/3	1.14			
	4/1	1.14			

Table includes the prevalence rates and risk ratios by category.

Table 0.22 Risk ratio for spinal osteoarthritis with a femoral subtrochanteric robusticity control.

		COUNT		PREVALENCE RATE	
		A	P	A	P
SOA FEMORAL MIDSHAFT ROBUSTICITY	1: <25%	26	8	76.5%	23.5%
	2: 25-50%	28	8	77.8%	22.2%
	3: 50-75%	30	7	81.1%	18.9%
	4: >75%	23	10	69.7%	30.3%
INTERQUARTILE RANGE		RISK RATIO			
	2/1	0.94			
	3/2	0.85			
	4/3	1.60			
	4/1	1.29			

Table includes the prevalence rates and risk ratios by category.

Table 0.23 Risk ratio for spinal osteoarthritis with a femoral midshaft polar SMA control.

		COUNT		PREVALENCE RATE	
		A	P	A	P
SOA FEMORAL MIDSHAFT POLAR SMA	1: <25%	23	4	85.2%	14.8%
	2: 25-50%	27	5	84.4%	15.6%
	3: 50-75%	23	5	82.1%	17.9%
	4: >75%	33	6	84.6%	15.4%
INTERQUARTILE RANGE		RISK RATIO			
	2/1	1.05			
	3/2	1.14			
	4/3	0.86			
	4/1	1.04			

Table includes the prevalence rates and risk ratios by category.

Table 0.24 Risk ratio for spinal osteoarthritis with a femoral midshaft area control.

		COUNT		PREVALENCE RATE	
		A	P	A	P
SOA FEMORAL MIDSHAFT AREA	1: <25%	27	9	75.0%	25.0%
	2: 25-50%	29	8	78.4%	21.6%
	3: 50-75%	23	10	69.7%	30.3%
	4: >75%	28	6	82.4%	17.6%
INTERQUARTILE RANGE		RISK RATIO			
	2/1	0.86			
	3/2	1.40			
	4/3	0.58			
	4/1	0.71			

Table includes the prevalence rates and risk ratios by category.

Table 0.25 Risk ratio for hip osteoarthritis with a biological sex control.

		COUNT		PREVALENCE RATE	
		A	P	A	P
HOA BIOLOGICAL SEX	MALE	86	17	83.5%	16.5%
	FEMALE	67	5	93.1%	6.9%
		RISK RATIO			
	MALE/FEMALE	2.38			

Table includes the prevalence rates and risk ratios by category.

Table 0.26 Risk ratio for hip osteoarthritis with an age at death control.

		COUNT		PREVALENCE RATE	
		A	P	A	P
HOA AGE AT DEATH CATEGORY	EARLY	52	4	92.9%	7.1%
	MIDDLE	38	2	95.0%	5.0%
	LATE	63	16	79.7%	20.3%
		RISK RATIO			
	MIDDLE/EARLY	0.70			
	LATE/MIDDLE	4.05			
	LATE/EARLY	2.84			

Table includes the prevalence rates and risk ratios by category.

Table 0.27 Risk ratio for hip osteoarthritis with a body mass index control.

		COUNT		INCINDENCE RATE	
		A	P	A	P
HOA BODY MASS INDEX INTERQUARTILE	1: <25%	29	8	78.4%	21.6%
	2: 25-50%	34	4	89.5%	10.5%
	3: 50-75%	36	4	90.0%	10.0%
	4: >75%	32	3	91.4%	8.6%
		RISK RATIO			
	2/1	0.49			
	3/2	0.95			
	4/3	0.86			
	4/1	0.40			

Table includes the prevalence rates and risk ratios by category.

Table 0.28 Risk ratio for hip osteoarthritis with an entheseal change control.

		COUNT		PREVALENCE RATE	
		A	P	A	P
HOA ENTHESEAL CHANGE	SLIGHT	64	6	91.4%	8.6%
	MODERATE	78	11	87.6%	12.4%
	SEVERE	11	5	68.8%	31.3%
		RISK RATIO			
	MODERATE/SLIGHT	1.44			
	SEVERE/MODERATE	2.53			
	SEVERE/SLIGHT	3.65			

Table includes the prevalence rates and risk ratios by category.

Table 0.29 Risk ratio for hip osteoarthritis with a femoral subtrochanteric shape control.

		COUNT		PREVALENCE RATE	
		A	P	A	P
HOA FEMORAL SUBTROCHANTERIC SHAPE	1: <25%	32	6	84.2%	15.8%
	2: 25-50%	35	4	89.7%	10.3%
	3: 50-75%	33	5	86.8%	13.2%
	4: >75%	32	6	84.2%	15.8%
INTERQUARTILE RANGE		RISK RATIO			
	2/1	0.65			
	3/2	1.28			
	4/3	1.20			
	4/1	1.00			

Table includes the prevalence rates and risk ratios by category.

Table 0.30 Risk ratio for hip osteoarthritis with a femoral subtrochanteric robusticity control.

		COUNT		PREVALENCE RATE	
		A	P	A	P
HOA FEMORAL SUBTROCHANTERIC ROBUSITICITY	1: <25%	32	5	86.5%	13.5%
	2: 25-50%	28	9	75.7%	24.3%
	3: 50-75%	40	1	97.6%	2.4%
	4: >75%	27	6	81.8%	18.2%
INTERQUARTILE RANGE		RISK RATIO			
	2/1	1.80			
	3/2	0.10			
	4/3	7.45			
	4/1	1.35			

Table includes the prevalence rates and risk ratios by category.

Table 0.31 Risk ratio for hip osteoarthritis with a femoral subtrochanteric polar SMA control.

		COUNT		PREVALENCE RATE	
		A	P	A	P
HOA FEMORAL SUBTROCHANTERIC POLAR SMA	1: <25%	29	6	82.9%	17.1%
	2: 25-50%	32	4	88.9%	11.1%
	3: 50-75%	30	5	85.7%	14.3%
	4: >75%	30	4	88.2%	11.8%
INTERQUARTILE RANGE		RISK RATIO			
	2/1	0.65			
	3/2	1.29			
	4/3	0.82			
	4/1	0.69			

Table includes the prevalence rates and risk ratios by category.

Table 0.32 Risk ratio for hip osteoarthritis with a femoral subtrochanteric area control.

		COUNT		PREVALENCE RATE	
		A	P	A	P
HOA FEMORAL SUBTROCHANTERIC AREA	1: <25%	31	6	83.8%	16.2%
	2: 25-50%	32	4	88.9%	11.1%
	3: 50-75%	32	7	82.1%	17.9%
	4: >75%	32	4	88.9%	11.1%
INTERQUARTILE RANGE		RISK RATIO			
	2/1	0.69			
	3/2	1.62			
	4/3	0.62			
	4/1	0.69			

Table includes the prevalence rates and risk ratios by category.

Table 0.33 Risk ratio for hip osteoarthritis with a femoral midshaft shape control.

		COUNT		PREVALENCE RATE	
		A	P	A	P
HOA FEMORAL MIDSHAFT SHAPE	1: <25%	31	7	81.6%	18.4%
	2: 25-50%	35	3	92.1%	7.9%
	3: 50-75%	30	8	78.9%	21.1%
	4: >75%	35	3	92.1%	7.9%
INTERQUARTILE RANGE		RISK RATIO			
	2/1	0.43			
	3/2	2.67			
	4/3	0.38			
	4/1	0.43			

Table includes the prevalence rates and risk ratios by category.

Table 0.34 Risk ratio for hip osteoarthritis with a femoral midshaft robusticity control.

		COUNT		PREVALENCE RATE	
		A	P	A	P
HOA FEMORAL MIDSHAFT ROBUSTICITY	1: <25%	30	5	85.7%	14.3%
	2: 25-50%	33	5	86.8%	13.2%
	3: 50-75%	33	6	84.6%	15.4%
	4: >75%	29	5	85.3%	14.7%
INTERQUARTILE RANGE		RISK RATIO			
	2/1	0.92			
	3/2	1.17			
	4/3	0.96			
	4/1	1.03			

Table includes the prevalence rates and risk ratios by category.

Table 0.35 Risk ratio for hip osteoarthritis with a femoral midshaft polar SMA control.

		COUNT		PREVALENCE RATE	
		A	P	A	P
HOA FEMORAL MIDSHAFT POLAR SMA	1: <25%	30	4	88.2%	11.8%
	2: 25-50%	30	5	85.7%	14.3%
	3: 50-75%	25	9	73.5%	26.5%
	4: >75%	38	2	95.0%	5.0%
INTERQUARTILE RANGE		RISK RATIO			
	2/1	1.21			
	3/2	1.85			
	4/3	0.19			
	4/1	0.43			

Table includes the prevalence rates and risk ratios by category.

Table 0.36 Risk ratio for hip osteoarthritis with a femoral midshaft area control.

		COUNT		PREVALENCE RATE	
		A	P	A	P
HOA FEMORAL MIDSHAFT AREA	1: <25%	32	5	86.5%	13.5%
	2: 25-50%	33	4	89.2%	10.8%
	3: 50-75%	28	9	75.7%	24.3%
	4: >75%	32	3	91.4%	8.6%
INTERQUARTILE RANGE		RISK RATIO			
	2/1	0.80			
	3/2	2.25			
	4/3	0.35			
	4/1	0.63			

Table includes the prevalence rates and risk ratios by category.

Table 0.37 Risk ratio for knee osteoarthritis with a biological sex control.

		COUNT		PREVALENCE RATE	
KOA BIOLOGICAL SEX		A	P	A	P
	MALE	93	8	92.1%	7.9%
	FEMALE	62	9	87.3%	12.7%
		RISK RATIO			
	MALE/FEMALE	0.62			

Table includes the prevalence rates and risk ratios by category.

Table 0.38 Risk ratio for knee osteoarthritis with an age at death control.

		COUNT		PREVALENCE RATE	
KOA AGE AT DEATH CATEGORY		A	P	A	P
	EARLY	52	3	94.5%	5.5%
	MIDDLE	37	3	92.5%	7.5%
	LATE	66	11	85.7%	14.3%
		RISK RATIO			
	MIDDLE/EARLY	1.38			
	LATE/MIDDLE	1.90			
	LATE/EARLY	2.62			

Table includes the prevalence rates and risk ratios by category.

Table 0.39 Risk ratio for knee osteoarthritis with a body mass index control.

		COUNT		PREVALENCE RATE	
KOA BODY MASS INDEX INTERQUARTILE		A	P	A	P
	1: <25%	32	5	86.5%	13.5%
	2: 25-50%	35	2	94.6%	5.4%
	3: 50-75%	36	4	90.0%	10.0%
	4: >75%	32	5	86.5%	13.5%
		RISK RATIO			
	2/1	0.40			
	3/2	1.85			
	4/3	1.35			
	4/1	1.00			

Table includes the prevalence rates and risk ratios by category.

Table 0.40 Risk ratio for knee osteoarthritis with an entheseal change control.

		COUNT		PREVALENCE RATE	
KOA ENTHESEAL CHANGE		A	P	A	P
	SLIGHT	62	6	91.2%	8.8%
	MODERATE	79	9	89.8%	10.2%
	SEVERE	14	2	87.5%	12.5%
		RISK RATIO			
	MODERATE/SLIGHT	1.16			
	SEVERE/MODERATE	1.22			
	SEVERE/SLIGHT	1.42			

Table includes the prevalence rates and risk ratios by category.

Table 0.41 Risk ratio for knee osteoarthritis with a femoral subtrochanteric shape control.

		COUNT		PREVALENCE RATE	
		A	P	A	P
KOA FEMORAL SUBTROCHANTERIC SHAPE	1: <25%	33	3	91.7%	8.3%
	2: 25-50%	35	4	89.7%	10.3%
	3: 50-75%	35	3	92.1%	7.9%
	4: >75%	32	5	86.5%	13.5%
INTERQUARTILE RANGE		RISK RATIO			
	2/1	1.23			
	3/2	0.77			
	4/3	1.71			
	4/1	1.62			

Table includes the prevalence rates and risk ratios by category.

Table 0.42 Risk ratio for knee osteoarthritis with a femoral subtrochanteric robusticity control.

		COUNT		PREVALENCE RATE	
		A	P	A	P
KOA FEMORAL SUBTROCHANTERIC ROBUSITICITY	1: <25%	32	5	86.5%	13.5%
	2: 25-50%	32	4	88.9%	11.1%
	3: 50-75%	36	4	90.0%	10.0%
	4: >75%	30	2	93.8%	6.3%
INTERQUARTILE RANGE		RISK RATIO			
	2/1	0.82			
	3/2	0.90			
	4/3	0.63			
	4/1	0.46			

Table includes the prevalence rates and risk ratios by category.

Table 0.43 Risk ratio for knee osteoarthritis with a femoral subtrochanteric polar SMA control.

		COUNT		PREVALENCE RATE	
		A	P	A	P
KOA FEMORAL SUBTROCHANTERIC POLAR SMA	1: <25%	31	4	88.6%	11.4%
	2: 25-50%	33	2	94.3%	5.7%
	3: 50-75%	31	4	88.6%	11.4%
	4: >75%	28	4	87.5%	12.5%
INTERQUARTILE RANGE		RISK RATIO			
	2/1	0.50			
	3/2	2.00			
	4/3	1.09			
	4/1	1.09			

Table includes the prevalence rates and risk ratios by category.

Table 0.44 Risk ratio for knee osteoarthritis with a femoral subtrochanteric area control.

		COUNT		PREVALENCE RATE	
		A	P	A	P
KOA FEMORAL SUBTROCHANTERIC AREA	1: <25%	31	6	83.8%	16.2%
	2: 25-50%	35	0	100.0%	0.0%
	3: 50-75%	32	7	82.1%	17.9%
	4: >75%	32	2	94.1%	5.9%
INTERQUARTILE RANGE		RISK RATIO			
	2/1	0.00			
	3/2	-			
	4/3	0.33			
	4/1	0.36			

Table includes the prevalence rates and risk ratios by category.

Table 0.45 Risk ratio for knee osteoarthritis with a femoral midshaft shape control.

		COUNT		PREVALENCE RATE	
		A	P	A	P
KOA FEMORAL MIDSHAFT SHAPE	1: <25%	29	7	80.6%	19.4%
	2: 25-50%	32	6	84.2%	15.8%
	3: 50-75%	37	1	97.4%	2.6%
	4: >75%	35	2	94.6%	5.4%
INTERQUARTILE RANGE		RISK RATIO			
	2/1	0.81			
	3/2	0.17			
	4/3	2.05			
	4/1	0.28			

Table includes the prevalence rates and risk ratios by category.

Table 0.46 Risk ratio for knee osteoarthritis with a femoral midshaft robusticity control.

		COUNT		PREVALENCE RATE	
		A	P	A	P
KOA FEMORAL MIDSHAFT ROBUSTICITY	1: <25%	27	7	79.4%	20.6%
	2: 25-50%	33	4	89.2%	10.8%
	3: 50-75%	36	2	94.7%	5.3%
	4: >75%	31	3	91.2%	8.8%
INTERQUARTILE RANGE		RISK RATIO			
	2/1	0.53			
	3/2	0.49			
	4/3	1.68			
	4/1	0.43			

Table includes the prevalence rates and risk ratios by category.

Table 0.47 Risk ratio for knee osteoarthritis with a femoral midshaft polar SMA control.

		COUNT		PREVALENCE RATE	
		A	P	A	P
KOA FEMORAL MIDSHAFT POLAR SMA	1: <25%	29	4	87.9%	12.1%
	2: 25-50%	28	6	82.4%	17.6%
	3: 50-75%	30	3	90.9%	9.1%
	4: >75%	38	2	95.0%	5.0%
INTERQUARTILE RANGE		RISK RATIO			
	2/1	1.46			
	3/2	0.52			
	4/3	0.55			
	4/1	0.41			

Table includes the prevalence rates and risk ratios by category.

Table 0.48 Risk ratio for knee osteoarthritis with a femoral midshaft area control.

		COUNT		PREVALENCE RATE	
		A	P	A	P
KOA FEMORAL MIDSHAFT AREA	1: <25%	31	5	86.1%	13.9%
	2: 25-50%	33	3	91.7%	8.3%
	3: 50-75%	30	7	81.1%	18.9%
	4: >75%	33	1	97.1%	2.9%
INTERQUARTILE RANGE		RISK RATIO			
	2/1	0.60			
	3/2	2.27			
	4/3	0.16			
	4/1	0.21			

Table includes the prevalence rates and risk ratios by category.

Table 0.49 Risk ratio for degenerative disc disease with a biological sex control.

		COUNT		PREVALENCE RATE	
		A	P	A	P
DDD BIOLOGICAL SEX	MALE	79	19	80.6%	19.4%
	FEMALE	60	10	85.7%	14.3%
		RISK RATIO			
	MALE/FEMALE	1.36			

Table includes the prevalence rates and risk ratios by category.

Table 0.50 Risk ratio for degenerative disc disease with an age at death control.

		COUNT		PREVALENCE RATE	
		A	P	A	P
DDD AGE AT DEATH CATEGORY	EARLY	49	7	87.5%	12.5%
	MIDDLE	32	5	86.5%	13.5%
	LATE	58	17	77.3%	22.7%
		RISK RATIO			
	MIDDLE/EARLY	1.08			
	LATE/MIDDLE	1.68			
	LATE/EARLY	1.81			

Table includes the prevalence rates and risk ratios by category.

Table 0.51 Risk ratio for degenerative disc disease with a body mass index control.

		COUNT		PREVALENCE RATE	
		A	P	A	P
DDD BODY MASS INDEX INTERQUARTILE	1: <25%	33	4	89.2%	10.8%
	2: 25-50%	29	8	78.4%	21.6%
	3: 50-75%	30	8	78.9%	21.1%
	4: >75%	26	5	83.9%	16.1%
		RISK RATIO			
	2/1	2.00			
	3/2	0.97			
	4/3	0.77			
	4/1	1.49			

Table includes the incidence rates and risk ratios by category.

Table 0.52 Risk ratio for degenerative disc disease with an entheseal change control.

		COUNT		PREVALENCE RATE	
		A	P	A	P
DDD ENTHESEAL CHANGE	SLIGHT	57	10	85.1%	14.9%
	MODERATE	68	18	79.1%	20.9%
	SEVERE	14	1	93.3%	6.7%
		RISK RATIO			
	MODERATE/SLIGHT	1.40			
	SEVERE/MODERATE	0.32			
	SEVERE/SLIGHT	0.45			

Table includes the prevalence rates and risk ratios by category.

Table 0.53 Risk ratio for degenerative disc disease with a femoral subtrochanteric shape control.

		COUNT		PREVALENCE RATE	
		A	P	A	P
DDD FEMORAL SUBTROCHANTERIC SHAPE INTERQUARTILE RANGE	1: <25%	31	6	83.8%	16.2%
	2: 25-50%	31	7	81.6%	18.4%
	3: 50-75%	3	7	30.0%	70.0%
	4: >75%	33	2	94.3%	5.7%
		RISK RATIO			
	2/1	1.14			
	3/2	3.80			
	4/3	0.08			
	4/1	0.35			

Table includes the prevalence rates and risk ratios by category.

Table 0.54 Risk ratio for degenerative disc disease with a femoral subtrochanteric robusticity control.

		COUNT		PREVALENCE RATE	
		A	P	A	P
DDD FEMORAL SUBTROCHANTERIC ROBUSITICITY	1: <25%	30	6	83.3%	16.7%
	2: 25-50%	31	5	86.1%	13.9%
	3: 50-75%	35	4	89.7%	10.3%
	4: >75%	27	5	84.4%	15.6%
INTERQUARTILE RANGE		RISK RATIO			
	2/1	0.83			
	3/2	0.74			
	4/3	1.52			
	4/1	0.94			

Table includes the prevalence rates and risk ratios by category.

Table 0.55 Risk ratio for degenerative disc disease with a femoral subtrochanteric polar SMA control.

		COUNT		PREVALENCE RATE	
		A	P	A	P
DDD FEMORAL SUBTROCHANTERIC POLAR SMA	1: <25%	30	4	88.2%	11.8%
	2: 25-50%	30	5	85.7%	14.3%
	3: 50-75%	28	5	84.8%	15.2%
	4: >75%	28	5	84.8%	15.2%
INTERQUARTILE RANGE		RISK RATIO			
	2/1	1.21			
	3/2	1.06			
	4/3	1.00			
	4/1	1.29			

Table includes the prevalence rates and risk ratios by category.

Table 0.56 Risk ratio for degenerative disc disease with a femoral subtrochanteric area control.

		COUNT		PREVALENCE RATE	
		A	P	A	P
DDD FEMORAL SUBTROCHANTERIC AREA	1: <25%	32	4	88.9%	11.1%
	2: 25-50%	31	4	88.6%	11.4%
	3: 50-75%	30	7	81.1%	18.9%
	4: >75%	30	5	85.7%	14.3%
INTERQUARTILE RANGE		RISK RATIO			
	2/1	1.03			
	3/2	1.66			
	4/3	0.76			
	4/1	1.29			

Table includes the prevalence rates and risk ratios by category.

Table 0.57 Risk ratio for degenerative disc disease with a femoral midshaft area control.

		COUNT		PREVALENCE RATE	
		A	P	A	P
DDD FEMORAL MIDSHAFT SHAPE	1: <25%	30	7	81.1%	18.9%
	2: 25-50%	28	7	80.0%	20.0%
	3: 50-75%	33	5	86.8%	13.2%
	4: >75%	32	4	88.9%	11.1%
INTERQUARTILE RANGE		RISK RATIO			
	2/1	1.06			
	3/2	0.66			
	4/3	0.84			
	4/1	0.59			

Table includes the prevalence rates and risk ratios by category.

Table 0.58 Risk ratio for degenerative disc disease with a femoral midshaft robusticity control.

		COUNT		PREVALENCE RATE	
		A	P	A	P
DDD FEMORAL MIDSHAFT ROBUSTICITY	1: <25%	29	5	85.3%	14.7%
	2: 25-50%	31	5	86.1%	13.9%
	3: 50-75%	32	6	84.2%	15.8%
	4: >75%	29	4	87.9%	12.1%
INTERQUARTILE RANGE		RISK RATIO			
	2/1	0.94			
	3/2	1.14			
	4/3	0.77			
	4/1	0.82			

Table includes the prevalence rates and risk ratios by category.

Table 0.59 Risk ratio for degenerative disc disease with a femoral midshaft polar SMA.

		COUNT		PREVALENCE RATE	
		A	P	A	P
DDD FEMORAL MIDSHAFT POLAR SMA	1: <25%	29	4	87.9%	12.1%
	2: 25-50%	28	5	84.8%	15.2%
	3: 50-75%	28	5	84.8%	15.2%
	4: >75%	33	6	84.6%	15.4%
INTERQUARTILE RANGE		RISK RATIO			
	2/1	1.25			
	3/2	1.00			
	4/3	1.02			
	4/1	1.27			

Table includes the prevalence rates and risk ratios by category.

Table 0.60 Risk ratio for degenerative disc disease with a femoral midshaft area control.

		COUNT		INCINDENCE RATE	
		A	P	A	P
DDD FEMORAL MIDSHAFT AREA	1: <25%	30	5	85.7%	14.3%
	2: 25-50%	32	5	86.5%	13.5%
	3: 50-75%	32	3	91.4%	8.6%
	4: >75%	27	7	79.4%	20.6%
INTERQUARTILE RANGE		RISK RATIO			
	2/1	0.95			
	3/2	0.63			
	4/3	2.40			
	4/1	1.44			

Table includes the prevalence rates and risk ratios by category.

Multivariate Analyses

Table 0.61 Omnibus tests of model coefficients displaying the chi-square values, degrees of freedom and significances of each step as a variable is remvoed from the equation.

			JOINT CONDITIONS													
			OA			SOA			HOA			KOA			DDD		
			CHI²	DF	SIG	CHI²	DF	SIG	CHI²	DF	SIG	CHI²	DF	SIG	CHI²	DF	SIG
BACKWARDS STEPS		STEP	58.271	33	0.004	55.157	33	0.009	42.906	33	0.116	53.478	33	0.014	35.483	33	0.352
	STEP 1	MODEL	58.271	33	0.004	55.157	33	0.009	42.906	33	0.116	53.478	33	0.014	35.483	33	0.352
		STEP	-0.115	1	0.735	-0.001	1	0.977	-0.509	3	0.917	-0.794	3	0.851	-0.176	3	0.981
	STEP 2	MODEL	58.157	32	0.003	55.157	32	0.007	42.398	30	0.066	52.684	30	0.006	35.307	30	0.232
		STEP	-0.178	3	0.620	-0.070	1	0.792	-1.038	3	0.792	-0.694	2	0.707	-1.259	3	0.739
	STEP 3	MODEL	56.381	29	0.002	55.087	31	0.005	41.359	27	0.038	51.990	28	0.004	34.048	27	0.165
		STEP	-2.390	3	0.495	-1.447	3	0.695	-0.194	1	0.659	-0.322	1	0.570	-1.587	3	0.662
	STEP 4	MODEL	53.990	23	0.001	53.641	28	0.002	41.165	26	0.030	51.668	27	0.003	32.461	24	0.116
		STEP	-2.697	3	0.292	-2.224	3	0.527	-0.708	2	0.702	-4.652	3	0.199	-0.518	1	0.472
	STEP 5	MODEL	51.294	23	0.001	51.416	25	0.001	40.457	24	0.019	47.016	24	0.003	31.943	23	0.101
		STEP	-3.730	3	0.292	-2.711	3	0.438	-2.675	3	0.444	-2.224	1	0.136	-2.403	3	0.493
	STEP 6	MODEL	47.563	20	0.000	48.705	22	0.001	37.782	21	0.014	44.793	23	0.004	29.540	20	0.078
		STEP	-1.162	1	0.281	-2.600	3	0.458	-2.633	3	0.452	-3.673	3	0.299	-2.505	3	0.474
	STEP 7	MODEL	46.402	19	0.000	46.106	19	0.000	35.150	18	0.009	41.120	20	0.004	27.036	17	0.058
		STEP	-3.675	3	0.299	-4.188	3	0.242	-4.359	3	0.225	-4.675	3	0.197	-2.326	3	0.508
	STEP 8	MODEL	42.727	16	0.000	41.917	16	0.000	30.791	15	0.009	36.445	17	0.004	24.710	14	0.038
		STEP	-5.659	3	0.129	-4.619	3	0.202				-3.316	3	0.345	-2.479	2	0.290
	STEP 9	MODEL	37.068	13	0.000	37.299	13	0.000				33.129	14	0.003	22.231	12	0.035
		STEP	-5.524	3	0.137	-4.281	3	0.233				-2.481	2	0.289	-4.488	3	0.213
	STEP 10	MODEL	31.544	10	0.000	33.018	10	0.000				30.647	12	0.002	17.743	9	0.038
		STEP	-5.028	3	0.170	-4.364	2	0.113							-1.308	1	0.253
	STEP 11	MODEL	26.515	7	0.000	28.654	8	0.000							16.435	8	0.037
		STEP													-4.005	2	0.135
	STEP 12	MODEL													12.429	6	0.053

Block and model for each step was the same and so were reduced into a single category for this table.

Table 0.62 Model summary with -2 log likelihood, Cox and Schnell R^2 and Nagelkerke R^2.

		JOINT CONDITIONS														
		OSTEOARTHRITIS			SPINAL OSTEOARTHRITIS			HIP OSTEOARTHRITIS			KNEE OSTEOARTHRITIS			DEG. DISC DISEASE		
		-2 LL	C&S R^2	N R^2	-2 LL	C&S R^2	N R^2	-2 LL	C&S R^2	N R^2	-2 LL	C&S R^2	N R^2	-2 LL	C&S R^2	N R^2
STEP	1	123.623	0.346	0.471	101.458	0.342	0.492	59.581	0.269	0.510	40.941	0.325	0.650	85.858	0.239	0.394
	2	123.738	0.346	0.471	101.459	0.342	0.492	60.350	0.266	0.504	41.735	0.321	0.642	86.034	0.238	0.392
	3	125.514	0.337	0.459	101.528	0.341	0.491	61.388	0.261	0.494	42.429	0.318	0.635	87.293	0.230	0.380
	4	127.905	0.326	0.443	103.975	0.334	0.481	61.583	0.260	0.492	42.751	0.316	0.631	88.879	0.221	0.364
	5	130.601	0.312	0.425	105.199	0.323	0.464	62.290	0.256	0.485	47.403	0.292	0.584	89.398	0.218	0.359
	6	134.331	0.293	0.399	107.910	0.309	0.444	64.965	0.241	0.457	49.627	0.281	0.561	91.800	0.203	0.335
	7	135.493	0.287	0.391	110.509	0.295	0.424	67.598	0.226	0.429	53.299	0.261	0.521	94.305	0.188	0.309
	8	139.168	0.268	0.365	114.698	0.272	0.392	71.957	0.201	0.381	57.974	0.235	0.470	96.631	0.173	0.285
	9	144.827	0.237	0.323	119.316	0.246	0.354				61.291	0.216	0.432	99.110	0.157	0.259
	10	150.351	0.206	0.280	123.597	0.221	0.319				63.772	0.202	0.403	103.598	0.128	0.210
	11	155.379	0.176	0.239	127.961	0.195	0.281							104.906	0.119	0.196
	12													108.911	0.091	0.150

Note that while N R^2 decreases -2LL increases.

Table 0.63 Hosmer and Lemeshow Goodness-of-Fit test displayed by step for each joint condition during the regression tests.

		JOINT CONDITIONS														
		OSTEOARTHRITIS			SPINAL OSTEOARTHRITIS			HIP OSTEOARTHRITIS			KNEE OSTEOARTHRITIS			DEG. DISC DISEASE		
		CHI²	DF	SIG	CHI²	DF	SIG	CHI²	DF	SIG	CHI²	DF	SIG	CHI²	DF	SIG
STEP	1	2.918	8	0.939	8.565	8	0.380	6.183	8	0.627	8.141	8	0.420	7.017	8	0.535
	2	7.685	8	0.465	8.589	8	0.378	5.959	8	0.652	9.660	8	0.294	6.470	8	0.595
	3	6.905	8	0.547	3.933	8	0.863	3.072	8	0.930	5.365	8	0.718	6.277	8	0.616
	4	10.680	8	0.220	8.393	8	3.960	12.349	8	0.136	11.576	8	0.171	4.217	8	0.837
	5	9.948	8	0.269	9.185	8	0.327	14.469	8	0.070	5.799	8	0.670	10.458	8	0.234
	6	5.621	8	0.690	7.762	8	0.457	9.754	8	0.283	14.636	8	0.067	7.103	8	0.526
	7	6.707	8	0.569	10.129	8	0.256	3.252	8	0.918	12.261	8	0.140	14.832	8	0.063
	8	11.919	8	0.155	3.523	8	0.897	9.238	8	0.323	5.921	8	0.656	8.518	8	0.385
	9	8.785	8	0.361	5.065	8	0.751				4.705	8	0.789	7.268	8	0.508
	10	5.999	8	0.647	12.635	8	0.125				3.558	7	0.829	3.690	8	0.884
	11	9.697	7	0.206	8.737	8	0.365							3.056	8	0.931
	12													2.622	8	0.956

Table 0.64 Classification table displaying the predictability of the regression equations by step.

			JOINT CONDITIONS				
			OA	SOA	HOA	KOA	DDD
PREDICTABLY BY BACKWARDS STEPS	STEP 1	ABSENT	85.9%	90.5%	99.2%	100.0%	93.5%
		PROBABLE	65.4%	62.2%	47.1%	60.0%	26.1%
		OVERALL	78.1%	82.6%	92.7%	95.6%	81.5%
	STEP 2	ABSENT	85.9%	90.5%	99.2%	100.0%	96.3%
		PROBABLE	65.4%	62.2%	41.2%	60.0%	26.1%
		OVERALL	78.1%	82.6%	92.0%	95.6%	83.8%
	STEP 3	ABSENT	85.9%	91.6%	98.3%	100.0%	94.4%
		PROBABLE	65.4%	62.2%	47.1%	60.0%	26.1%
		OVERALL	78.1%	83.3%	92.0%	95.6%	82.3%
	STEP 4	ABSENT	82.4%	92.6%	98.3%	99.2%	95.3%
		PROBABLE	61.5%	59.5%	47.1%	53.3%	21.7%
		OVERALL	74.5%	83.3%	92.0%	94.1%	82.3%
	STEP 5	ABSENT	82.4%	93.7%	98.3%	99.2%	96.3%
		PROBABLE	61.5%	59.5%	47.1%	73.3%	17.4%
		OVERALL	74.5%	84.1%	92.0%	96.3%	82.3%
	STEP 6	ABSENT	84.7%	91.6%	99.2%	99.2%	98.1%
		PROBABLE	55.8%	59.5%	41.2%	66.7%	32.1%
		OVERALL	73.7%	82.6%	92.0%	95.6%	84.6%
	STEP 7	ABSENT	81.2%	91.6%	99.2%	99.2%	97.2%
		PROBABLE	57.7%	48.6%	35.3%	46.7%	21.7%
		OVERALL	72.3%	79.6%	91.2%	93.4%	83.8%
	STEP 8	ABSENT	85.9%	90.5%	99.2%	98.3%	96.3%
		PROBABLE	55.9%	51.4%	23.5%	26.7%	17.4%
		OVERALL	74.5%	79.5%	89.8%	90.4%	82.3%
	STEP 9	ABSENT	87.1%	90.5%		98.3%	97.2%
		PROBABLE	46.2%	40.5%		26.7%	17.4%
		OVERALL	71.5%	76.5%		90.4%	83.1%
	STEP 10	ABSENT	82.4%	90.5%		98.3%	98.1%
		PROBABLE	50.0%	35.1%		20.0%	13.0%
		OVERALL	70.1%	75.0%		89.7%	83.1%
	STEP 11	ABSENT	88.2%	91.6%			99.1%
		PROBABLE	50.0%	35.1%			13.0%
		OVERALL	73.7%	75.8%			83.8%
	STEP 12	ABSENT					100.0%
		PROBABLE					0.0%
		OVERALL					82.3%

Note that the highest predictability factor for the absence, probable, and overall categories occurs only during Step 1 for hip OA. Green = highest absent prediction. Yellow = highest probable prediction. Blue = highest overall prediction.

Thank you for reading!